



REPUBLIC OF KENYA

MINISTRY OF  
HEALTH

# The National Guidelines for the Prevention and Management of Kidney Conditions

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First Edition 2026



**MINISTRY OF  
HEALTH**

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**1st Edition 2026**

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# Foreword

Chronic kidney disease (CKD) and other kidney disorders represent a growing public health and socio-economic challenge in Kenya, contributing to premature mortality, reduced quality of life, and catastrophic health expenditure for affected individuals and families. Approximately 5% of the deaths in the country are due to CKD. This burden is closely linked to the rising prevalence of non-communicable diseases such as hypertension and diabetes, late diagnosis, and longstanding inequities in access to timely and quality kidney care.

The Government of Kenya has made significant strides in expanding access to kidney services through the decentralisation of dialysis and renal units across counties. These efforts have improved geographical access to life-saving care and reflect our commitment to strengthening specialised services within the public health system. However, it is increasingly evident that a health system response focused predominantly on end-stage kidney disease is not sustainable. A deliberate shift towards prevention, early detection, and integrated long-term management of kidney disease is therefore essential.

Guided by Article 43 of the Constitution of Kenya and anchored in the principles of Universal Health Coverage, the Ministry of Health remains steadfast in its commitment to ensuring equitable access to quality, affordable, and people-centred health services. In alignment with ongoing health sector reforms, including the Primary Health Care Act, 2023; the Social Health Insurance Act, 2023; and the Digital Health Act, 2023—these kidney disease guidelines have been developed to standardise, strengthen, and harmonise kidney care delivery across all levels of the health system.

The guidelines emphasise early identification and continuity of care at the Primary Health Care level, supported by clear referral pathways for specialised services. Additionally, the guidelines call for the integration of kidney care with related programmes such as diabetes, hypertension, cardiovascular disease, HIV, and maternal health. They aim to improve quality of care, promote sustainability of kidney services, and reduce the preventable progression to advanced kidney disease.

I commend the technical teams, the Kenya Renal Association, county governments, and partners whose collective efforts made the development of these guidelines possible. I call upon all stakeholders to support their dissemination and implementation, so that together we may prioritise kidney health, reduce the burden of kidney disease, and advance equitable health outcomes for all Kenyans.



**Dr. Patrick Amoth, CBS**  
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# Preface

These kidney disease guidelines have been developed to provide practical, standardised and evidence-based guidance for the prevention, early detection, diagnosis, management and referral of kidney diseases in Kenya. They respond to the need for consistent, high-quality and standardised kidney care across all levels of the health system in the context of a growing burden of kidney disease.

The guidelines were developed by the Ministry of Health in collaboration with the Kenya Renal Association and a multidisciplinary Technical Working Group drawn from national and county governments, professional associations, academic and training institutions, private institutions, implementing partners and patient representatives. The development process was informed by a review of current scientific evidence, relevant international kidney guidelines, and existing national policies and strategies to ensure technical soundness and contextual relevance.

The guidelines provide clear direction on prevention, screening, diagnosis, management, referral and follow-up of kidney diseases, with emphasis on prevention, early identification, integrated management of co-morbidities, continuity of care, and appropriate use of diagnostics and therapies. They outline the continuum of care from and are intended to support effective service delivery at facility and community levels.

These guidelines are intended for use by health-care workers and health institutions in both the public and private sectors, including clinicians, nurses, pharmacists, laboratory personnel, community health teams, health managers, programme implementers, training institutions and policy makers involved in kidney disease prevention and care.

These guidelines are to be applied in a manner that is appropriate to the level of care and scope of practice within the health system. Users are expected to implement the recommendations in line with their professional competencies, available resources, and the service delivery mandate of their respective levels of care. Finally, the guidelines represent a critical milestone in strengthening the prevention, early detection, and comprehensive management of kidney diseases in Kenya. Their effective implementation will require committed leadership, sustained investment, intersectoral collaboration, and continuous capacity strengthening across all levels of the health system.



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# Acknowledgement

The Ministry of Health acknowledges with appreciation the collective efforts of all individuals and institutions whose expertise, commitment, and collaboration contributed to the development of the National Guidelines for the Prevention and Management of Kidney Conditions.

We acknowledge the support from the office of the Cabinet Secretary, the Principal Secretary State Department of Medical services, Director General for Health and the Director Family Health

We express our sincere gratitude to the Cardiometabolic and Renal Health Technical Working Group, comprising persons with lived experience, nephrologists, renal nurses, clinical officers, nutritionists, pharmacists, public health specialists, and other health professionals. Their dedication in reviewing evidence, contextualising global best practices, and shaping practical recommendations for the Kenyan health system was invaluable.

We particularly acknowledge the technical leadership and invaluable contributions of the Kenya Renal Association and the Kenya Renal Nurses Association for mobilising specialist expertise to support the development of these guidelines. We also extend special recognition to the Renal Patients Society of Kenya for ensuring that the perspectives of individuals with lived experience were meaningfully incorporated into this process

The Ministry further acknowledges the contributions of key technical leads and contributors, including Dr Jonathan Wala, Dr Joyce Bwombengi, Dr Elizabeth Onyango, Dr Stephen Mutiso, Dr Yvette Kisaka, Ms Elaine Muigai, and Mr John Gikonyo whose commitment and stewardship were instrumental in advancing this process.

We gratefully acknowledge the support of partners, including the Kenya Red Cross Society, AstraZeneca, and others, whose technical and financial assistance facilitated stakeholder consultations, workshops, and evidence review processes. We also appreciate the valuable input of County Departments of Health and health facility management teams, whose perspectives ensured that the guidelines are practical, implementable, and responsive to service delivery realities across all levels of care.

Finally, the Ministry of Health extends its appreciation to the administrative and secretariat teams for their logistical and coordination support, and to all stakeholders who reviewed draft versions of the guidelines and provided constructive feedback. Their collective efforts have resulted in a comprehensive and practical resource that will strengthen the quality of kidney care and improve health outcomes for people living with or at risk of kidney disease in Kenya.



**Dr. Gladwell Gathecha**

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# Abbreviations & Acronyms

|          |                                                      |
|----------|------------------------------------------------------|
| ACEi     | Angiotensin Converting Enzyme inhibitor              |
| ACR      | Albumin–Creatinine Ratio                             |
| ADPKD    | Autosomal Dominant Polycystic Kidney Disease         |
| AKI      | Acute Kidney Injury                                  |
| ANA      | Antinuclear Antibody                                 |
| ANC      | Antenatal Care                                       |
| ANCA     | Anti-Neutrophil Cytoplasmic Antibody                 |
| anti-GBM | Anti-Glomerular Basement Membrane antibody           |
| ARB      | Angiotensin Receptor Blocker                         |
| ARNI     | Angiotensin Receptor–Neprilysin Inhibitor            |
| ARPKD    | Autosomal Recessive Polycystic Kidney Disease        |
| ASOT     | Anti-Streptolysin O Titre                            |
| ATN      | Acute Tubular Necrosis                               |
| BMI      | Body Mass Index                                      |
| BP       | Blood Pressure                                       |
| BPE      | Benign Prostatic Enlargement                         |
| BUN      | Blood Urea Nitrogen                                  |
| CAKUT    | Congenital Anomalies of the Kidney and Urinary Tract |
| CKD      | Chronic Kidney Disease                               |
| CKD-EPI  | Chronic Kidney Disease Epidemiology Collaboration    |
| CSF      | Cerebrospinal Fluid                                  |
| CT       | Computed Tomography                                  |
| CT KUB   | Computed Tomography of Kidney, Ureter and Bladder    |
| DJ Stent | Double-J Ureteric Stent                              |
| DMSA     | Dimercaptosuccinic Acid scan                         |
| DTPA     | Diethylenetriamine Pentaacetic Acid scan             |
| eGFR     | Estimated Glomerular Filtration Rate                 |
| ESRD     | End-Stage Renal Disease                              |
| FSGS     | Focal Segmental Glomerulosclerosis                   |
| GFR      | Glomerular Filtration Rate                           |
| HbA1c    | Glycosylated Haemoglobin                             |
| HBV      | Hepatitis B Virus                                    |
| HCV      | Hepatitis C Virus                                    |
| HD       | Haemodialysis                                        |
| HIV      | Human Immunodeficiency Virus                         |
| HUS      | Haemolytic Uraemic Syndrome                          |
| ICU      | Intensive Care Unit                                  |
| IgA      | Immunoglobulin A                                     |
| INR      | International Normalised Ratio                       |
| IV       | Intravenous                                          |

|         |                                                |
|---------|------------------------------------------------|
| KDIGO   | Kidney Disease: Improving Global Outcomes      |
| KUB     | Kidney, Ureter and Bladder                     |
| LMWH    | Low Molecular Weight Heparin                   |
| MCD     | Minimal Change Disease                         |
| MDRD    | Modification of Diet in Renal Disease equation |
| MRI     | Magnetic Resonance Imaging                     |
| MCS     | Microscopy, Culture and Sensitivity            |
| MRSA    | Methicillin-Resistant Staphylococcus Aureus    |
| NaCl    | Sodium Chloride                                |
| NOAC    | Novel Oral Anticoagulant                       |
| NSAIDs  | Non-Steroidal Anti-Inflammatory Drugs          |
| OGTT    | Oral Glucose Tolerance Test                    |
| PCN     | Primary Health Care Network                    |
| PD      | Peritoneal Dialysis                            |
| PHC     | Primary Health Care                            |
| RBC     | Red Blood Cell                                 |
| RPGN    | Rapidly Progressive Glomerulonephritis         |
| RRT     | Renal Replacement Therapy                      |
| RBS     | Random Blood Sugar                             |
| SGLT2i  | Sodium-Glucose Cotransporter-2 inhibitor       |
| SIHG    | Steroid-Induced Hyperglycaemia                 |
| SLE     | Systemic Lupus Erythematosus                   |
| TIN     | Tubulointerstitial Nephritis                   |
| TMP-SMX | Trimethoprim–Sulfamethoxazole                  |
| TTP     | Thrombotic Thrombocytopenic Purpura            |
| UACR    | Urine Albumin-Creatinine Ratio                 |
| UEC     | Urea, Electrolytes and Creatinine              |
| UPCR    | Urine Protein-Creatinine Ratio                 |
| UTI     | Urinary Tract Infection                        |

# Chapter 1: Introduction to Kidney Diseases

## 1.1 Overview of Kidney Diseases

Definition: Kidney diseases are defined as a heterogeneous group of disorders affecting kidney structure or function.

The prevalence of chronic kidney disease is approximately 10% of the population (1), with that of end-stage kidney failure approaching 0.1% of the population. Consequently, kidney disease presents a major burden to the community, causing morbidity, affecting quality of life, impacting the outcome of other diseases, and increasing mortality.

The management of kidney diseases involves correctly identifying them, confirming the diagnosis and applying the proper management.

## 1.2 Classification of Kidney Diseases

Kidney diseases are commonly classified based on time course (duration and progression). This time-based classification helps clinicians determine the underlying cause, urgency, and appropriate management approach.

The major categories are:

- Acute Kidney Injury / Acute Kidney Disease (AKI/AKD)
- Chronic Kidney Disease
- Rapidly Progressive Glomerulonephritis (RPGN): Although not a separate classification of kidney disease, RPGN is a clinical syndrome resulting from glomerular diseases among other causes and can cause AKI.

Table 1: Classification of Kidney Diseases

| Classification                            | Description                                                                                                                                                                                                                                                                                                                                   |
|-------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Acute Kidney Injury (AKI)                 | <ul style="list-style-type: none"><li>• Sudden decline in kidney function occurring within hours to days</li><li>• Marked rise in creatinine or drop in urine output</li><li>• Common causes: dehydration, sepsis, toxins, obstruction</li></ul>                                                                                              |
| Chronic Kidney Disease (CKD)              | <ul style="list-style-type: none"><li>• Abnormal kidney structure or function lasting ≥3 months, with implications for health</li><li>• Causes include diabetes, hypertension, long-standing glomerular disease</li><li>• Progressive, long-term decline</li><li>• Managed through slowing progression and preventing complications</li></ul> |
| Rapidly Progressive Kidney Disease (RPGN) | <ul style="list-style-type: none"><li>• Kidney function deteriorates rapidly over weeks to months, faster than chronic disease</li><li>• Often due to severe glomerulonephritis (vasculitis, anti-GBM disease)</li><li>• Medical emergency</li><li>• Leads to early kidney failure without prompt treatment</li></ul>                         |

## 1.3 Presentation of Kidney Diseases

- Kidney diseases present in many ways. Often, the early course is silent, with individuals remaining asymptomatic until the disease is advanced.
- In other instances, kidney disease may present more abruptly, with severe illness requiring urgent care.
- While some conditions originate within the kidneys themselves, many are linked to, or occur as a consequence of, other systemic diseases such as hypertension, diabetes, infections, or autoimmune disorders.
- Because early stages often go unnoticed, a high index of suspicion is essential, particularly among populations at increased risk.
- Routine screening and timely use of basic laboratory or imaging tests at all levels of care are critical for early detection. This approach provides an important window to initiate appropriate management, slow disease progression, avert complications, and ultimately reduce the risk of kidney failure and premature mortality.

Table 2: Presentation of Kidney Diseases

|                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>1. Abnormal Kidney Function (GFR)</b> <ul style="list-style-type: none"><li>Reflects impaired filtration leading to accumulation of nitrogenous waste.</li><li>Detected through routine laboratory tests</li></ul> Key indicators: <ul style="list-style-type: none"><li>Elevated serum creatinine or BUN (UEC panel)</li><li>Classified by rate of decline:<ul style="list-style-type: none"><li>Hours –days: Acute Kidney Injury (AKI)</li><li>Weeks –months: Rapidly Progressive Glomerulonephritis (RPGN)</li><li>Months–years: Chronic Kidney Disease (CKD)</li></ul></li></ul> |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
| <b>2. Abnormal Urine Findings</b> <ul style="list-style-type: none"><li>Indicates impaired glomerular filtration, tubular dysfunction, or abnormalities along the urinary tract.</li><li>Urinalysis is a key, low - cost screening tool</li></ul> Key indicators:                                                                                                                                                                                                                                                                                                                       |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
| Urine Dipstick <ul style="list-style-type: none"><li>Proteinuria (1<sup>+</sup>,2<sup>+</sup>,3<sup>+</sup> 4<sup>+</sup>)</li><li>Haematuria (1<sup>+</sup>,2<sup>+</sup>,3<sup>+</sup> 4<sup>+</sup>)</li><li>Leucocytes, nitrites, ketones</li></ul>                                                                                                                                                                                                                                                                                                                                 | Microscopy <ul style="list-style-type: none"><li>Cells:<ul style="list-style-type: none"><li>White blood cells (=leucocytes = pus cells)</li></ul></li><li>Red blood cells<ul style="list-style-type: none"><li>Intact: indicating bleeding into the urinary system</li><li>Dysmorphic: indicating glomerular injury (pathognomonic of glomerulonephritis)</li></ul></li><li>Casts (hyaline, granular, RBC, WBC)</li><li>Crystals (risk of stones intrarenal, calyceal, ureteric, bladder or urethral) or tubular crystallopathy)</li><li>Organisms - Bacteria (confirm with culture), ova (e.g. Schistosoma haematobium)</li></ul> | Protein Measurement <ul style="list-style-type: none"><li>24-hour urine protein (gold standard)</li><li>Estimation<ul style="list-style-type: none"><li>Urine protein on dipstick</li><li>Urine albumin to-creatinine ratio (UACR)</li><li>Urine protein -to-creatinine ratio (UPCR)</li></ul></li></ul>                                                                                                                                                                                                                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                      |
| <b>3. Electrolyte Abnormalities</b> <ul style="list-style-type: none"><li>Disturbances in electrolyte balance commonly occur in kidney disease and may indicate impaired regulation</li><li>Key indicators<ul style="list-style-type: none"><li>Sodium: hyponatremia, hypernatremia</li><li>Potassium: hypokalemia, hyperkalemia</li><li>Calcium: hypocalcemia, hypercalcemia</li><li>Magnesium: hypomagnesemia, hypermagnesemia</li><li>Phosphate abnormalities: hyperphosphatemia, hyperphosphatemia</li></ul></li></ul>                                                              |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
| <b>4. Acid-Base Disorders</b> <p>Reflect reduced renal capacity to maintain normal acid-base balance</p> Examples <ul style="list-style-type: none"><li>Metabolic acidosis</li><li>Metabolic alkalosis</li></ul>                                                                                                                                                                                                                                                                                                                                                                        |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
| <b>5. Abnormal Kidney Imaging</b> <p>Structural or anatomical abnormalities identified through ultrasound or other imaging modalities.</p> <p>Essential for evaluating chronic disease, obstruction or congenital anomalies.</p> Examples:                                                                                                                                                                                                                                                                                                                                              |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                                                                                                                                      |
| i. Number:<br>single kidney,<br>supernumerary                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           | iii. Structural Changes                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  | iv. Obstructed Urinary Flow:                                                                                                                                                                                                                                                                                                                         |
| ii. K idneys Shape:<br>horseshoe kidney,<br>atrophic kidney,<br>enlarged kidney                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         | <ul style="list-style-type: none"><li>Scarring (hyperechoic kidneys, poor cortico - medullary differentiation)</li><li>Cystic kidneys:<ul style="list-style-type: none"><li>Simple cysts:<ul style="list-style-type: none"><li>Isolated:</li><li>Developmental</li><li>Acquired (in dialysis patients)</li><li>Bilateral:</li></ul></li><li>Enlarged kidneys:</li></ul></li></ul>                                                                                                                                                                                                                                                   | <p>Polycystic kidney disease:<br/>Autosomal dominant (ADPKD) or<br/>autosomal recessive (ARPKD)</p> <ul style="list-style-type: none"><li>Normal/small kidneys: -<ul style="list-style-type: none"><li>Medullary cystic kidney disease (autosomal dominant tubulointerstitial kidney disease)</li><li>Medullary sponge kidney</li><li>Nephronophthisis</li></ul></li><li>Complex cysts:<ul style="list-style-type: none"><li>Benign:<ul style="list-style-type: none"><li>Angiomyolipoma</li></ul></li><li>Malignant:<ul style="list-style-type: none"><li>Renal cell carcinoma - Tumour syndromes</li></ul></li></ul></li></ul> | <ul style="list-style-type: none"><li>Obstructive uropathy: leads to obstructive nephropathy</li><li>Sites: kidney, ureter, bladder, urethra</li><li>Causes: -<ul style="list-style-type: none"><li>Intraluminal (stones/clots)</li><li>Mural (strictures/tumours)</li><li>Extrinsic (cervical cancer, retroperitoneal fibrosis)</li></ul></li></ul> |

## 1.4 Laboratory and Imaging Evaluation for Kidney Disease

| Evaluation / Test Category                     | Description                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |
|------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| UEC                                            | <b>GFR determination:</b> <ul style="list-style-type: none"> <li>From serum creatinine and blood urea</li> <li>Estimated GFR calculated using formula: <ul style="list-style-type: none"> <li>CKD-EPI formula – 2021</li> <li>MDRD formula</li> <li>Cockcroft-Gault formula</li> </ul> </li> </ul> <b>Electrolytes:</b> <ul style="list-style-type: none"> <li>Assess sodium, potassium, chloride</li> </ul>                                                                                            |
| Urine evaluation                               | <b>Spot urine tests:</b> <ul style="list-style-type: none"> <li>Urine dipstick</li> <li>Urine microscopy</li> <li>Urine culture</li> <li>Urine protein measurement</li> </ul> <b>24h urine measurements:</b> <ul style="list-style-type: none"> <li>Volume (urine output rate)</li> <li>Protein</li> <li>Electrolytes: sodium, potassium, calcium, phosphorous, magnesium</li> <li>Hormones: catecholamines</li> <li>For urine stone analysis: calcium, phosphorous, urate, oxalate, cystine</li> </ul> |
| Special urine tests                            | <ul style="list-style-type: none"> <li>Urine electrolyte concentration (often compared to urine creatinine in order to adjust for changes in urine concentration)</li> <li>Urine osmolality: measured and calculated</li> </ul>                                                                                                                                                                                                                                                                         |
| Other tests                                    | <ul style="list-style-type: none"> <li>Blood gas analysis</li> <li>Diabetes assessment: blood sugar, HbA1C</li> </ul> <b>Serological tests:</b> <ul style="list-style-type: none"> <li>Viral serologies: HIV, HBV, HCV</li> <li>Other infections: ASOT, VDRL</li> <li>Connective tissue disease: ANA, ANCA, anti-GBM</li> </ul> <b>Complement activation:</b> <ul style="list-style-type: none"> <li>Serum C3 level</li> <li>Serum C4 level</li> <li>Serum total complement level</li> </ul>            |
| Ultrasound                                     | <ul style="list-style-type: none"> <li>KUB (kidney, ureter and bladder)</li> <li>Bladder (including post-void bladder volume)</li> <li>Prostate</li> <li>Doppler ultrasound of renal vessels</li> </ul>                                                                                                                                                                                                                                                                                                 |
| CT KUB                                         | <ul style="list-style-type: none"> <li>Without contrast</li> <li>With contrast: CT urogram</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                   |
| Other imaging                                  | <ul style="list-style-type: none"> <li>MRI of the kidneys</li> <li>Intravenous urogram</li> </ul> Radionuclide scans: <ul style="list-style-type: none"> <li>DTPA: for kidney perfusion</li> <li>DMSA: for kidney structure</li> </ul>                                                                                                                                                                                                                                                                  |
| Kidney biopsy for histology (Light microscopy) | <ul style="list-style-type: none"> <li>Light microscopy: <ul style="list-style-type: none"> <li>With usual stains</li> <li>With special stains</li> </ul> </li> </ul>                                                                                                                                                                                                                                                                                                                                   |
| Kidney biopsy (Immunohistology)                | <ul style="list-style-type: none"> <li>Immunofluorescence</li> <li>Immunohistochemistry</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                      |
| Kidney biopsy (Electron microscopy)            | <ul style="list-style-type: none"> <li>Electron microscopy</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                   |
| Genetic testing                                | <ul style="list-style-type: none"> <li>Genetic testing</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                       |

# Chapter 2: Integrating Kidney Disease Prevention and Care within Primary Healthcare and Referral Networks

## 2.1 Introduction

Primary Health Care (PHC) is the foundation of Kenya's health system, providing essential, evidence-based, and socially acceptable services that are universally accessible at a cost the community and country can sustain. In line with the Astana Declaration (2018), Kenya developed the Primary Health Care Strategic Framework 2019–2024 to strengthen the delivery of PHC services across Levels 1, 2, and 3.

### The Primary Health Care Network

- A Primary Health Care Network (PCN) is “an administrative health region established to increase access to primary health care services for patients, as well as coordinate with local health facilities in order to improve the overall operational efficiency of the network”
- The PCN begins at the community level where it is provided by community health care workers (Level 1 Health Care) and is linked to Level 2 and Level 3 health care facilities
- A PCN comprises Level 2 and Level 3 facilities as spokes, linked to one Level 4 facility that serves as the hub.

## 2.2 Roles in Kidney Disease across Levels of Care

Kidney disease prevention and management require a coordinated, tiered approach across all levels of the health system. Each level of care—from the community to national referral hospitals—has clearly defined roles in health promotion, early detection, risk factor control, complication management, and referral. Strengthening linkages between these levels ensures continuity of care, timely intervention, and improved patient outcomes.

*Table 3: Roles of Service Delivery Levels In Kidney Disease Prevention, Early Detection, Management, and Referral*

| PHC Level                                                            | Key Roles                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    |
|----------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Level 1 – Community (CHPs, Households)                               | <ul style="list-style-type: none"> <li>Promote kidney-healthy lifestyles: balanced diet, regular physical activity, rational and cautious use of herbal remedies, smoking and alcohol cessation, and safe OTC use.</li> <li>Educate communities on CKD risk factors, including hypertension, diabetes, and obesity.</li> <li>Encourage routine screening: blood pressure, blood glucose, and BMI/waist circumference monitoring.</li> <li>Identify early danger signs: facial or limb swelling, haematuria, and reduced urine output.</li> <li>Support self-management: appointment reminders, medication adherence, adequate hydration, and dietary compliance.</li> <li>Refer suspected cases to Level 2/3 facilities for further evaluation.</li> </ul>                                                                                   |
| Level 2 & 3 – Primary Care Facilities (Dispensaries, Health Centres) | <ul style="list-style-type: none"> <li>Formal risk assessment and screening for CKD: BP, glucose, urinalysis (protein, glucose, blood), eGFR</li> <li>Initial management of risk factors (hypertension, diabetes, obesity) using standard PHC protocols</li> <li>Health promotion and patient education (diet, physical activity, lifestyle modification)</li> <li>Early management of high-risk groups (ANC, GMP, NCD clinics)</li> <li>Management of uncomplicated CKD and monitoring disease progression</li> <li>Identification of complications: uncontrolled BP/glucose, recurrent UTIs, oedema, haematuria, reduced urine output</li> <li>Referral to Level 4 for further evaluation, imaging, specialist review or complications</li> </ul>                                                                                          |
| Level 4 – County Referral Hospital (The Hub)                         | <ul style="list-style-type: none"> <li>Diagnosis and management of kidney diseases including staging, evaluation, and follow-up</li> <li>Laboratory and imaging services: renal function tests, urinalysis, ultrasound</li> <li>Management of complications: acute kidney injury, severe hypertension, electrolyte abnormalities, recurrent UTIs</li> <li>Initiation of renal-protective therapies (ACEi/ARBs), anaemia management, and comorbidity control</li> <li>Specialist consultation (physician/nephrologist where available)</li> <li>Coordination of PCN: HRH oversight, financing, administration, quality improvement, M&amp;E</li> <li>Supportive supervision and mentoring of lower levels of care</li> <li>Referral to Level 5/6 for advanced care (dialysis initiation, nephrologist review, transplant services)</li> </ul> |
| Level 5/6 – National & Teaching Referral Hospitals                   | <ul style="list-style-type: none"> <li>Advanced kidney disease management, including dialysis (haemodialysis/peritoneal dialysis)</li> <li>Management of complex complications of CKD and AKI</li> <li>Kidney transplant services and post-transplant follow-up (where available)</li> <li>Sub-specialist care: nephrology, urology, cardiology for comorbid NCDs</li> <li>Training, research, guideline development and national capacity-building</li> <li>Back-referral to Level 4 and PCN for ongoing follow-up and continuity of care</li> </ul>                                                                                                                                                                                                                                                                                        |

## 2.3 Prevention of Kidney Diseases

- Prevention of kidney diseases requires a comprehensive, life-course approach embedded within a strong primary health care framework.
- Kidney disease evolves along a continuous health–disease spectrum, and its prevention is best conceptualised through the natural history of the condition.
- Accordingly, effective strategies integrate all four levels of prevention—primordial, primary, secondary, and tertiary—to reduce risk exposure, enable early detection, slow disease progression, and prevent complications across the lifespan.

Table 4: Levels of Prevention in Kidney Disease

| Level of Prevention   | Focus / Description                                                                                                                                 | Key Actions / Components                                                                                                                                               |
|-----------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Primordial Prevention | <ul style="list-style-type: none"><li>• Focuses on preventing the emergence of risk factors</li></ul>                                               | <ul style="list-style-type: none"><li>• Includes promoting overall wellness and addressing environmental and societal factors contributing to kidney disease</li></ul> |
| Primary Prevention    | <ul style="list-style-type: none"><li>• Aims to prevent the onset of CKD by managing known risk factors such as hypertension and diabetes</li></ul> | <ul style="list-style-type: none"><li>• Public health measures, lifestyle modifications, and early health education are key components</li></ul>                       |
| Secondary Prevention  | <ul style="list-style-type: none"><li>• Involves early detection and treatment to prevent disease progression</li></ul>                             | <ul style="list-style-type: none"><li>• Regular screenings, monitoring of individuals with risk factors, and addressing early symptoms are essential</li></ul>         |
| Tertiary Prevention   | <ul style="list-style-type: none"><li>• Focuses on managing established CKD to prevent complications and improve quality of life</li></ul>          | <ul style="list-style-type: none"><li>• Includes advanced medical interventions, ongoing care, and rehabilitation</li></ul>                                            |

## 2.4 Strategies for Intervention in Kidney Disease Prevention

- The prevention and management of kidney disease require coordinated strategies that address both population-wide risk reduction and targeted care for high-risk individuals.
- The table below summarises the two complementary intervention approaches and the key implementation framework necessary to ensure effective, context-responsive delivery within the health system.

*Table 5: Summary of Strategic Approaches and Implementation Framework for Kidney Disease Prevention and Management*

| Strategy / Component          | Purpose & Focus                                                                                                                  | Key Elements / Considerations                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
|-------------------------------|----------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Population Strategy           | <ul style="list-style-type: none"> <li>Reduce average kidney disease risk across the entire population.</li> </ul>               | <ul style="list-style-type: none"> <li>Broad preventive measures</li> <li>Health promotion and lifestyle education</li> <li>Policy and environmental interventions</li> <li>Does not require identification of high-risk groups</li> <li>High population impact, though individual benefit may be modest</li> </ul>                                                                                                                                                                                        |
| High-Risk Individual Strategy | <ul style="list-style-type: none"> <li>Prevent onset or progression among individuals with identifiable risk factors.</li> </ul> | <ul style="list-style-type: none"> <li>Focuses on identifying and targeting individuals who are at higher risk due to specific exposures or risk factors</li> <li>Involves tailored interventions, targeted screenings, and personalized management plans</li> <li>Particularly effective for preventing disease progression in high-risk individuals but requires accurate identification and can be resource-intensive</li> </ul>                                                                        |
| Implementation Framework      | <ul style="list-style-type: none"> <li>Ensure coordinated and effective delivery of prevention strategies.</li> </ul>            | <ul style="list-style-type: none"> <li>Effective health promotion involves a multisectoral collaboration including health services, media, civil societies, policy makers among others</li> <li>A comprehensive approach that addresses biological, psychological, and social factors is necessary, with active involvement from individuals, families, and communities</li> <li>Developing community-specific programs tailored to local kidney disease needs and risks enhances effectiveness</li> </ul> |

# Chapter 3: Glomerular Diseases: Nephrotic and Nephritic Syndrome

## 3.1 Introduction

- The unit of the kidney is called the nephron.
- The glomerulus is part of the unit of the kidney which is involved in filtration of blood, allowing plasma filtrate to pass through into the tubules.
- Plasma proteins and blood cells are prevented from crossing the glomerular filtration barrier by size (size barrier) and by the negative charge (charge barrier).
- Glomerular abnormalities present with either abnormalities in the urine or with impaired glomerular filtration rate (GFR)
- Glomerular diseases can present either as nephrotic syndrome or nephritic syndrome

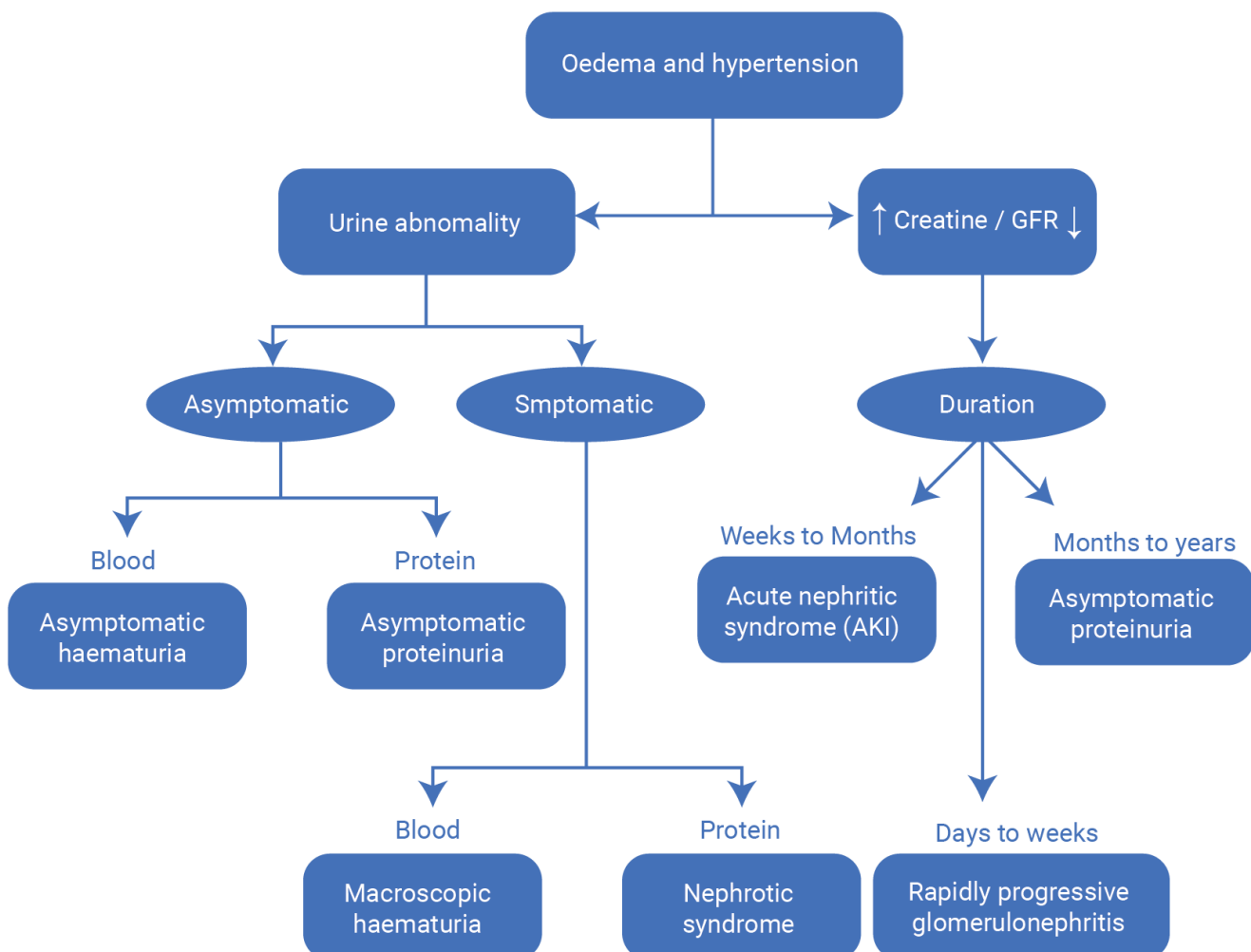


Figure 1: Presentation of Glomerular Diseases

↑: increased; ↓: decreased; AKI: acute kidney injury; GFR: glomerular filtration rate

## 3.2 Nephrotic Syndrome

### 3.2.1 Definition

Nephrotic syndrome is a serious and debilitating kidney condition caused by a range of different diseases that damage the glomeruli allowing proteins (such as albumin) to pass through into the nephron tubules and be lost in urine.

### 3.2.2 Presentation

The pentad of nephrotic syndrome comprises five defining features: heavy proteinuria (>3.5 g/24 h), hypoalbuminaemia (<30 g/L), generalised oedema, hyperlipidaemia, and lipiduria. Protein loss through the glomerulus lowers oncotic pressure, causing oedema and stimulating hepatic lipid production. Together, these findings reflect glomerular filtration barrier dysfunction and form the basis of diagnosis and management.

| Typical Pentad                                                                                                                                                                                                                                                                                      |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>1. Proteinuria:</b>                                                                                                                                                                                                                                                                              | <ul style="list-style-type: none"><li>Initial test:<ul style="list-style-type: none"><li>Urinalysis: Dipstick urine protein 3+ or 4+</li></ul></li><li>Quantify proteinuria:<ul style="list-style-type: none"><li>24h urine: &gt;3.5g of protein/24h</li><li>Spot urine:<ul style="list-style-type: none"><li>Urine protein: creatinine ratio (UPCR) &gt;500mg/g (&gt;50mg/mmol)</li><li>Urine albumin: creatinine ratio (UACR) &gt;300mg/g (&gt;30mg/mmol)</li></ul></li></ul></li></ul> |
| <b>2. Hypoalbuminemia</b>                                                                                                                                                                                                                                                                           | <ul style="list-style-type: none"><li>Serum albumin &lt;30g/L</li></ul>                                                                                                                                                                                                                                                                                                                                                                                                                   |
| <b>3. Peripheral oedema</b>                                                                                                                                                                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
| <b>4. Hyperlipidaemia</b>                                                                                                                                                                                                                                                                           | <ul style="list-style-type: none"><li>Increased serum total cholesterol</li><li>Increased serum triglycerides</li></ul>                                                                                                                                                                                                                                                                                                                                                                   |
| <b>5. Lipiduria</b>                                                                                                                                                                                                                                                                                 | <ul style="list-style-type: none"><li>Lipid casts in urine</li></ul>                                                                                                                                                                                                                                                                                                                                                                                                                      |
| Other features                                                                                                                                                                                                                                                                                      |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
| <ul style="list-style-type: none"><li>Relatively well patient apart from gross oedema</li><li>Usually normal BP</li><li>At risk for (especially if serum albumin &lt;20g/L):<ul style="list-style-type: none"><li>Deep venous thrombosis</li><li>Malnutrition</li><li>Infection</li></ul></li></ul> |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |

### 3.2.3 Aetiology of nephrotic syndrome

- The glomerulus in nephrotic syndrome can be damaged by diseases that affect only the kidney (primary glomerular disease) or by diseases that can affect other organs (systemic diseases).
- In adults, identification of glomerular diseases requires a kidney biopsy.

Table 6: Classification of Common Primary and Secondary Glomerular Diseases

| Common primary glomerular diseases                                                                                                                                                                                                                                                                                                                                                                       | Common secondary glomerular diseases (systemic diseases)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• Focal segmental glomerulosclerosis (FSGS) <ul style="list-style-type: none"> <li>• In adults, FSGS accounts for between 35% and 50% of nephrotic syndrome causes</li> </ul> </li> <li>• Membranous nephropathy (MN)</li> <li>• Minimal change disease (MCD)</li> <li>• In adults, FSGS accounts for between 35% and 50% of nephrotic syndrome causes</li> </ul> | <ul style="list-style-type: none"> <li>• Diabetic kidney disease</li> <li>• Infections: <ul style="list-style-type: none"> <li>• Viral infections: Hepatitis B, Hepatitis C, HIV</li> <li>• Bacterial: Syphilis</li> <li>• Protozoal: malaria (chronic)</li> </ul> </li> <li>• Autoimmune: <ul style="list-style-type: none"> <li>• Systemic lupus erythematosus (SLE)</li> </ul> </li> <li>• Malignancy: <ul style="list-style-type: none"> <li>• Haematological: myeloma, leukaemia, lymphoma</li> <li>• Solid: breast, gynaecological</li> </ul> </li> <li>• Drugs: NSAIDs, interferon, lithium, bisphosphonates</li> </ul> |

### 3.2.4 Diagnostic criteria for nephrotic syndrome

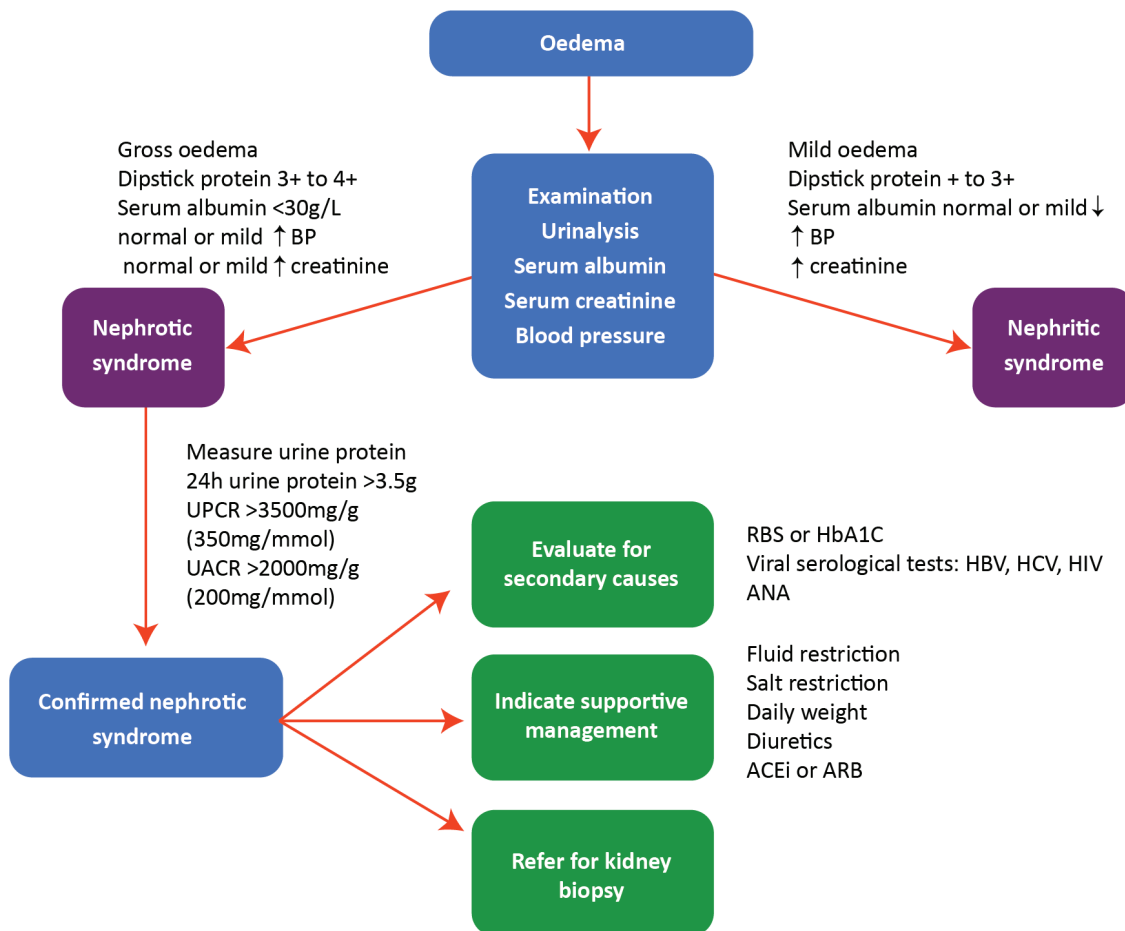
| Condition         | Values                                                                                                                                                             |
|-------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Proteinuria       | <ul style="list-style-type: none"> <li>• 24h urine protein &gt;3.5g</li> <li>or</li> <li>• UACR* &gt;2000mg/g</li> <li>or</li> <li>• UPCR* &gt;3500mg/g</li> </ul> |
|                   |                                                                                                                                                                    |
| Hypo-albuminaemia | <ul style="list-style-type: none"> <li>• Serum albumin &lt;30g/L</li> </ul>                                                                                        |
| Peripheral oedema |                                                                                                                                                                    |

UACR - Urine Albumin-Creatinine Ratio

UPCR - Urine Protein-Creatinine Ratio

### 3.2.5. Clinical approach

- Nephrotic syndrome is suspected when a patient presents with peripheral oedema (ankles, legs or periorbital)
- Subsequent evaluation is according to the flow chart below (Figure 2):



↑: increased; ↓: decreased; ACEi: angiotensin converting enzyme inhibitor; ANA: antinuclear antibody; ARB: angiotensin receptor blocker; BP: blood pressure; HbA1C: glycosylated haemoglobin; HBV: hepatitis B virus; HCV: hepatitis C virus; HIV: human immunodeficiency virus; RBS: random blood sugar; UACR: urine albumin: creatinine ratio; UPCR: urine protein: creatinine ratio

Figure 2: Approach to Nephrotic Syndrome

## 3.2.6 Management of nephrotic syndrome

### A. Management of Oedema

#### i. Create negative fluid balance (fluid input to be less than fluid output):

- Restrict fluids to less than 1.0-1.5L/24h (tell patient to drink water only when thirsty)
- Restrict salt to NaCl less than 5g/d (1 teaspoon of salt) (salt for cooking only, no salt added on food on the table)
- Increase urine output with diuretics



#### ii. Diuretics:

- Powerful diuretics (loop diuretics) preferred to mild diuretics (e.g. thiazide diuretics)
  - Loop diuretics:
    - Oral Furosemide 40-80mg once a day.
- IF:

| Adequate response                                                                                                                                                                              | Adequate urine response but inadequate diuresis                                            | Inadequate response                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Patient should pass more urine within 2-4h of dose</li><li>• Urine output should exceed input to enable weight loss of approximately 1kg/day</li></ul> | <ul style="list-style-type: none"><li>• Increase frequency of dose to BID or TID</li></ul> | <ul style="list-style-type: none"><li>• Double Furosemide dose (rather than increasing frequency of administration)</li><li>• Change to IV rather than oral Furosemide</li><li>• Add diuretic acting on different segment of tubule:<ul style="list-style-type: none"><li>◦ Thiazide diuretic: HCTZ or chlorthalidone 12.5-25mg OD</li><li>◦ Thiazide-like diuretic: Metolazone 2.5-5mg OD or BID</li><li>◦ Potassium-sparing diuretic (especially if hypokalaemia present): Spironolactone 25-100mg BID or TID</li></ul></li><li>• Switch to longer-acting loop diuretic: Torsemide 10-40mg OD</li><li>• Use IV infusion rather than bolus IV doses</li></ul> |



#### iii. Monitoring:

- Daily morning patient weight (aim for weight loss of  $\geq 1$ kg/d)
- Laboratory monitoring:
  - Serum potassium, sodium, (magnesium if hypokalaemia present)
  - Urea and creatinine
  - Check on alternate days initially, then weekly until patient is oedema-free

## B. Management of Proteinuria

| Category              | Details                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |
|-----------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Supportive Management | Low-salt diet (NaCl less than 5g/d = 1 teaspoon of salt)<br>BP control (if BP is elevated >140/90mmHg): <ul style="list-style-type: none"><li>• Target: systolic BP &lt;120mmHg in adults (caution to avoid hypotension in frail and elderly patients)</li><li>• Systolic BP ≤50th percentile in children</li><li>• Agents: angiotensin blockade + diuretic + other agents</li></ul>                                                                                    |
| Angiotensin blockade  | Agents: ACE inhibitor or Angiotensin receptor blocker (especial-ly if intolerant to ACE inhibitor) <ul style="list-style-type: none"><li>• ACE inhibitor e.g. Enalapril, Captopril, Ramipril, Lis-inopril, Perindopril</li><li>• Angiotensin receptor blocker e.g. Losartan, Irbesartan, Valsartan, Telmisartan</li></ul> Target: <ul style="list-style-type: none"><li>• Maximum tolerated dose</li><li>• Proteinuria reduction to &lt;1g/24h (UACR 100mg/g)</li></ul> |
| Monitoring            | <ul style="list-style-type: none"><li>• Monitor creatinine and potassium at start of and within 4 weeks after starting angiotensin blockade</li><li>• Stop angiotensin blockade only if:<ul style="list-style-type: none"><li>• Serum creatinine rises &gt;30%</li><li>• Serum potassium &gt;6mmol/L</li><li>• Hypotension occurs</li><li>• Patient feels sick (sick-day rule): resume when patient feels better</li></ul></li></ul>                                    |

## C. Management of Hyperlipidaemia

- Hyperlipidaemia will resolve if proteinuria is controlled. No need for lipid-lowering treatment if nephrotic syndrome is likely to respond to treatment
- If persistent nephrotic syndrome with hyperlipidaemia, decision for lipid-lowering treatment should be based on estimation of the total cardiovascular risk score
- If cardiovascular risk score is high (≥20% 10-year risk of a major cardiovascular event) OR more than 2 other cardiovascular risk factors present, e.g. diabetes, hypertension, tobacco use), start treatment:
  - o **Lifestyle modification:**
    - Diet: heart-healthy diet, low in saturated fats
    - Exercise
    - Weight loss
    - Smoking cessation
  - o **Medications:**
    - Statin: e.g. Rosuvastatin 10mg OD, Atorvastatin 20mg OD
    - Non-statin therapy if lipid levels not at target or statins not tolerated:
      - Ezetimibe 10mg OD
      - Fibrates: Fenofibrate 200mg OD
      - Bile acid sequestrants: Cholestyramine 4g OD or BID

## D. Management of Hypercoagulability and Thrombosis

| Risk Category                                      | Clinical Criteria                                                                                                                                                                                                                                                                                                                                                                | Management                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |
|----------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Low Risk</b><br>(No prophylaxis)                | Nephrotic syndrome with: <ul style="list-style-type: none"> <li>Albumin &gt;20 g/L</li> <li>No other risk factors for thrombosis</li> </ul>                                                                                                                                                                                                                                      | <ul style="list-style-type: none"> <li>No prophylaxis</li> <li>No treatment for thrombosis</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      |
| <b>Moderate Risk</b><br>(Give prophylaxis)         | Nephrotic syndrome with Albumin <20 g/L <b>plus any one of the following:</b> <ul style="list-style-type: none"> <li>Proteinuria &gt;10 g/24 h</li> <li>BMI &gt;35 kg/m<sup>2</sup></li> <li>Congestive heart failure (NYHA III –IV)</li> <li>Prolonged immobilization</li> <li>Recent orthopaedic or abdominal surgery</li> <li>Genetic predisposition to thrombosis</li> </ul> | <b>Prophylactic anticoagulation:</b> <ul style="list-style-type: none"> <li>Unfractionated Heparin 5000 units SC BD</li> </ul> <p style="text-align: center;"><b>OR</b></p> LMW Heparin (e.g., Enoxaparin 40 mg OD)<br>⚠ Consult nephrologist/pharmacist to adjust dose to renal function (eGFR)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
| <b>Thrombosis Present</b><br>(Treatment indicated) | Sites may include: <ul style="list-style-type: none"> <li>Deep venous thrombosis</li> <li>Arterial thrombosis</li> <li>Pulmonary embolism</li> <li>Non-valvular atrial fibrillation</li> </ul>                                                                                                                                                                                   | <b>i. Full-dose anticoagulation for 6 - 12 months</b> <ul style="list-style-type: none"> <li><b>Heparin followed by Warfarin:</b> <ul style="list-style-type: none"> <li>Unfractionated Heparin 10,000 units TID <b>OR</b> LMW Heparin (Enoxaparin 1 mg/kg BD) × 5 days + • Warfarin 5 mg OD</li> <li>Check INR day 3; target INR 2.0 – 3.0</li> <li>Stop heparin once target INR reached</li> <li>Monitor INR weekly, then monthly</li> </ul> </li> <li>LMW Heparin (Enoxaparin 1 mg/kg BD) for 6 –12 months</li> <li>New Oral Anticoagulant (NOACs) <ul style="list-style-type: none"> <li>Apixaban 10 mg BD for 7 days then 5 mg BD</li> <li>Rivaroxaban 15 mg BD for 3 weeks then 20 mg OD</li> </ul> </li> </ul> <b>ii. ⚠ Consult nephrologist/pharmacist to adjust dose to renal function (eGFR)</b> |

## E. Infection Prevention:

High risk of infection in nephrotic syndrome patients due to depressed immune system response by the disease (e.g. loss of immunoglobulins into urine) and by treatment (immunosuppressive medication).

Prevent infections by:

- Basic hygiene
- Infection prevention protocols:
  - Hand washing
  - Sanitizer use
  - Face mask for patients at high risk and for patients when in environments with high risk for exposure to airborne or droplet pathogens (e.g. in crowded environments)

- Vaccination:
  - o Pneumococcal vaccination
  - o Influenza vaccine
  - o Hepatitis B vaccination: for CKD 4 or 5 patients
- Screening:
  - o For hepatitis B and C and HIV
- Minimizing exposure to immunosuppressive medications:
  - o Always refer adults with nephrotic syndrome for kidney biopsy: not all causes of nephrotic syndrome require immunosuppression
  - o Consult nephrologist for immunosuppressive regimens that:
    - Avert the immediate complications of the disease
    - Prevent disease progression
    - Minimize harmful side-effects of the medications (including infections, metabolic diseases, cancer, and infertility)

## F. Prevent Complications in Patients on High-dose Steroids

### i. Classification of potential complications associated with high-dose steroid

- High-dose corticosteroid therapy is associated with a wide range of potential adverse effects affecting multiple organ systems. These complications may occur immediately, as idiosyncratic reactions, or develop with prolonged use, necessitating structured monitoring and prevention strategies.
- Classification of these complications supports early identification, risk mitigation, and safe long-term management of patients receiving steroid therapy.

*Table 7: Classification of Potential Complications Associated with High-Dose Steroid*

| Immediate                                                                                                                                                                                                                  | Idiosyncratic                                                                                                                                      | Chronic                                                                                                                                     |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• Fluid retention with oedema</li> <li>• Blurriness of vision</li> <li>• Mood impairment</li> <li>• Immune response modulation</li> <li>• Steroid induced hyperglycaemia</li> </ul> | <ul style="list-style-type: none"> <li>• Avascular bone necrosis</li> <li>• Cataract formation</li> <li>• Glaucoma</li> <li>• Psychosis</li> </ul> | <ul style="list-style-type: none"> <li>• Osteoporosis</li> <li>• Dyslipidaemia</li> <li>• Obesity</li> <li>• Adrenal suppression</li> </ul> |

### ii. Prevention of complications in patients on high-dose steroids

- Empiric administration of immunosuppressants/steroids should be clearly restricted to exceptional circumstances (not routine) where kidney biopsy can't be done.
- Patients receiving high-dose corticosteroid therapy require proactive measures to prevent treatment-related complications.
- Preventive strategies should address bone protection, opportunistic infection prophylaxis, and gastrointestinal protection to reduce morbidity.

The following recommendations outline standard pharmacologic interventions to mitigate these risks while ensuring safe long-term steroid use.

- Bone protection with
  - o Calcium 500mg to 1000mg BID
  - o Vitamin D 800 units PO OD or 60,000 units PO once a week
  - o If high risk for osteoporosis or if osteoporosis present, give Bisphosphonates:
    - Alendronate 35mg PO once a week
    - Ibandronate 150mg PO once a month
    - Zoledronic acid 5mg IV every 2 years
- Pneumocystis pneumonia prophylaxis:
  - o Trimethoprim-Sulphamethoxazole 480mg OD or 960mg on alternate days (adjust dose to eGFR)
  - o If TMP-SMX intolerant: Dapsone 50mg OD
- Gastroprotection with:
  - o Proton-pump inhibitors e.g. Esomeprazole or Omeprazole 20-40mg OD
  - o Mist antacids 10-20mL TID

#### Treatment of Steroid-induced hyperglycaemia

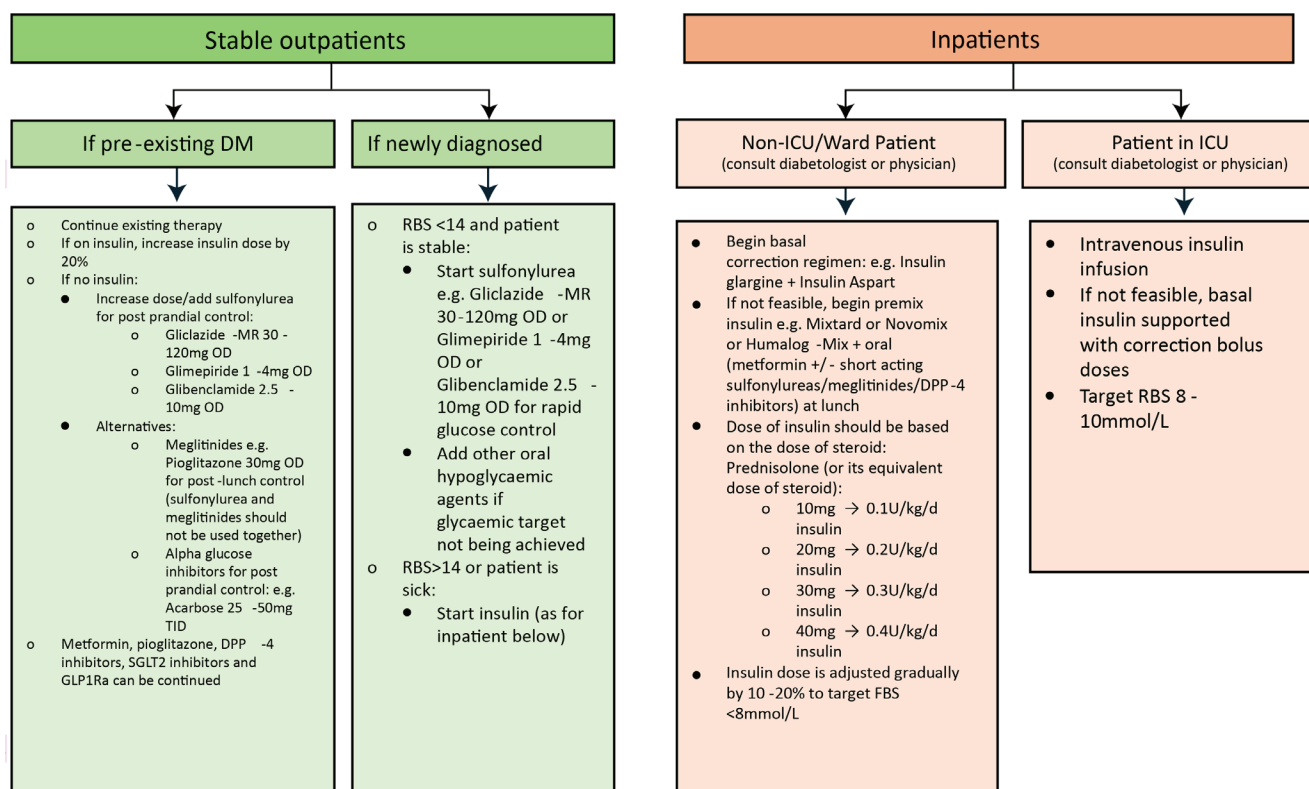


Figure 3: Treatment of Steroid-induced Hyperglycaemia

Steroid-induced hyperglycaemia (SIHG) is a common metabolic complication defined as abnormally elevated blood glucose associated with the use of steroids in patients with or without pre-existing diabetes mellitus. It occurs due to the effects of corticosteroids on glucose metabolism and is diagnosed using standard diabetes criteria:

- Random blood glucose  $\geq 11.1$  mmol/L
- Fasting blood glucose  $\geq 7$  mmol/L
- 2-hour glucose  $\geq 11.1$  mmol/L following a 75 g oral glucose tolerance test (OGTT)
- HbA1c  $\geq 6.5\%$

Early monitoring, preventive lifestyle measures, and timely pharmacologic management are essential to achieve glycaemic control and reduce acute and long-term complications.

## G. Dietary management in Nephrotic Syndrome

- Dietary modification is a key component of the comprehensive management of nephrotic syndrome and chronic kidney disease.
- Appropriate regulation of sodium, protein, caloric intake, and dietary fat helps control oedema and blood pressure, reduce proteinuria, slow disease progression, and lower cardiovascular risk.
- The table below outlines recommended targets and the clinical rationale for each dietary component.

*Table 8: Recommended Targets and the Clinical Rationale for Dietary Components*

| Component   | Recommendations / Targets                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
|-------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Sodium      | <p>Target: <math>&lt;5</math> g NaCl/day (<math>=2</math> g Na)/day; <math>\sim 1</math> teaspoonful/day</p> <p>Benefits / Clinical Considerations:</p> <ul style="list-style-type: none"> <li>• Blood pressure control</li> <li>• Oedema control: less fluid retention and improved response to diuretics</li> <li>• Fluid intake control: reduces thirst</li> <li>• Proteinuria control: lowers intraglomerular pressure and enhances response to angiotensin blockade</li> </ul>                                                                                                                                                                                                                                                  |
| Protein     | <ul style="list-style-type: none"> <li>• Restrict dietary protein according to the degree of proteinuria.</li> <li>• Excess protein intake increases intraglomerular pressure, leading to more proteinuria, greater glomerular and tubular damage, and a higher risk of progressing kidney disease (loss of GFR).</li> <li>• Over-restriction of protein can cause protein malnutrition and increase mortality risk.</li> <li>• Target: Limit protein to <math>0.8</math>–<math>1.0</math> g/kg/day.</li> <li>• Add <math>1</math> g of protein for each additional <math>1</math> g of urinary protein loss, up to a maximum of <math>5</math> g/day.</li> <li>• Prefer plant-based proteins over animal-based proteins.</li> </ul> |
| Calories    | <ul style="list-style-type: none"> <li>• Avoid excess caloric intake to avoid obesity and central adiposity that increase risk of CKD progression, cardiovascular events, and mortality Target: <math>35</math> kcal/kg/day</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
| Dietary Fat | <ul style="list-style-type: none"> <li>• Dietary fat to be <math>&lt;30\%</math> of total calories Mono- or poly-unsaturated fat to be <math>7</math>–<math>10\%</math> of total calories</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |

## H. Specific Management of Nephrotic Syndrome

### i. The aetiologies of nephrotic syndrome

- The aetiologies of nephrotic syndrome are:
  - o immune-mediated and may respond to immunosuppression guided by a nephrologist
  - o infection-related and so can be exacerbated by immunosuppression
  - o or have a genetic cause and therefore will not respond to immunosuppression.
- Treatment of nephrotic syndrome therefore depends on the aetiology.

### ii. The Diagnosis of Nephrotic Syndrome

The gold standard of diagnosis of nephrotic syndrome is a kidney biopsy specimen that is evaluated using:

- o Light microscopy with special staining
- o Immunohistology with immune staining for antibodies and complement
- o Occasionally, electron microscopy
- In children, especially if less than 12 years of age, more than 80% have minimal-change disease as the aetiology and respond to treatment with steroids.
  - o Minimal change disease does not cause deterioration of kidney function (creatinine remains normal)
  - o Therefore, kidney biopsy is not indicated in childhood nephrotic syndrome unless:
    - Disease is steroid-resistant
    - Kidney function is abnormal or deteriorating
    - There is family history for nephrotic syndrome (suggesting a genetic aetiology)
  - o Contraindications to kidney biopsy:
    - o Absolute:
      - Bleeding diathesis
      - Uncontrolled hypertension
      - Hydronephrosis
      - Uncooperative patient
    - o Relative:
      - Single kidney
      - Small kidneys
      - Anatomical abnormalities of kidney
      - Active urinary tract infection or local site infection
- Treatment of nephrotic syndrome involves two phases:
  - o Induction of remission: using potent immunosuppressive medications
  - o Maintaining of remission: using less potent or lower doses of immunosuppressive medications to prevent relapse
- Treatment if patient is less than 18 years of age (childhood nephrotic syndrome):
- Evaluate for secondary causes
- If no secondary cause: commence immunosuppression with glucocorticoids:
  - o Prednisone/Prednisolone 60 mg/m<sup>2</sup>/d or 2 mg/kg/d (maximum 60 mg/d) PO for 4 weeks followed by
  - o Prednisone/Prednisolone 40 mg/m<sup>2</sup> or 1.5 mg/kg (maximum of 50 mg) on alternate days for another 4 weeks
  - or
  - o Prednisone/Prednisolone 60 mg/m<sup>2</sup>/d (maximum 60 mg/d) for 6 weeks followed by
  - o Prednisone/Prednisolone 40 mg/m<sup>2</sup> or 1.5 mg/kg (maximum of 50 mg) on alternate days for another 6 weeks

### iii. Management of Nephrotic Syndrome in Adults (18 years and above)

Management of nephrotic syndrome in adults will depend on specific aetiology of nephrotic syndrome.

- o Evaluate for and manage secondary causes:
  - Obesity: BMI>35kg/m<sup>2</sup>
  - Viral infections: HIV, HBV, HCV
  - Chronic infections: malaria, schistosomiasis, syphilis
  - Autoimmune diseases: post-streptococcal, SLE, rheumatoid arthritis, small-vessel vasculitis
  - Diabetes
  - Severe hypertension: hypertensive crisis
- o Empirical immunosuppressive medications:
  - May be commenced only if:
    - Kidney biopsy contraindicated
    - Kidney biopsy difficult: e.g. late pregnancy
  - In such cases, commence empirical treatment, in consultation with a nephrologist, based on the fact that steroid-responsive aetiologies (minimal change disease and FSGS) account for a substantial proportion of nephrotic syndrome
  - Induction of remission:
    - Oral steroid: Prednisone or Prednisolone
    - Dose:
      - o 1mg/kg/d (maximum 80mg/d) or
      - o 2mg/kg on alternate days (maximum 120mg on alternate days)
        - Duration:
          - o Minimum of 4 weeks
          - o Maximum of 16 weeks
    - Maintenance of remission:
      - After remission, taper dose over at least 24 weeks
  - Goals for proteinuria reduction: refer to Table 9 below:

*Table 9: Nephrotic Syndrome Treatment Response Definitions*

|                    |                                                                                                                                              |
|--------------------|----------------------------------------------------------------------------------------------------------------------------------------------|
| Complete remission | Proteinuria <0.3g/day<br>or UACR <300mg/g (<30mg/mmol) or UPCR <500mg/g<br>Stable serum creatinine<br>Albumin >35g/L                         |
| Partial remission  | Proteinuria 0.3 to <3.5g/day<br>or UACR 300 to <2000mg/g (30–200mg/mmol)<br>or UPCR 300 to 3500mg/g<br>and a reduction of >50% from baseline |
| Relapse            | Proteinuria >3.5g/day or<br>UACR >2000mg/g (200mg/mmol)<br>or UPCR >3500mg/g after complete remission previously achieved                    |

UACR: urine albumin: creatinine ratio; UPCR: urine protein: creatinine ratio

## 3.3 Nephritic Syndrome

### 3.3.1 Definition

- Nephritic syndrome is a consequence of inflammation of the glomerulus.
- It is often sudden in onset, resulting in acute nephritic syndrome (acute glomerulonephritis)

### 3.3.2 Clinical presentation

- Acute nephritic syndrome is characterised by sudden onset of:
  - o Haematuria (glomerular haematuria):
    - Characterised by dysmorphic red blood cells and red cell casts on urine microscopy
    - Usually microscopic but can be macroscopic (grossly evident)
  - o Non-nephrotic range proteinuria (<3.5 g/d)
  - o Oedema: periorbital, pedal, sacral, pulmonary
  - o Hypertension
  - o Decreased GFR resulting in azotaemia (accumulation of nitrogenous waste) and even uraemia (clinical disease from azotaemia)

### 3.3.3 Aetiologies (main)

- Infection-related glomerulonephritis
- Lupus nephritis
- Membranoproliferative glomerulonephritis
- Antineutrophil cytoplasmic antibody (ANCA) – associated vasculitis
- IGA nephropathy

### 3.3.4 Laboratory evaluation

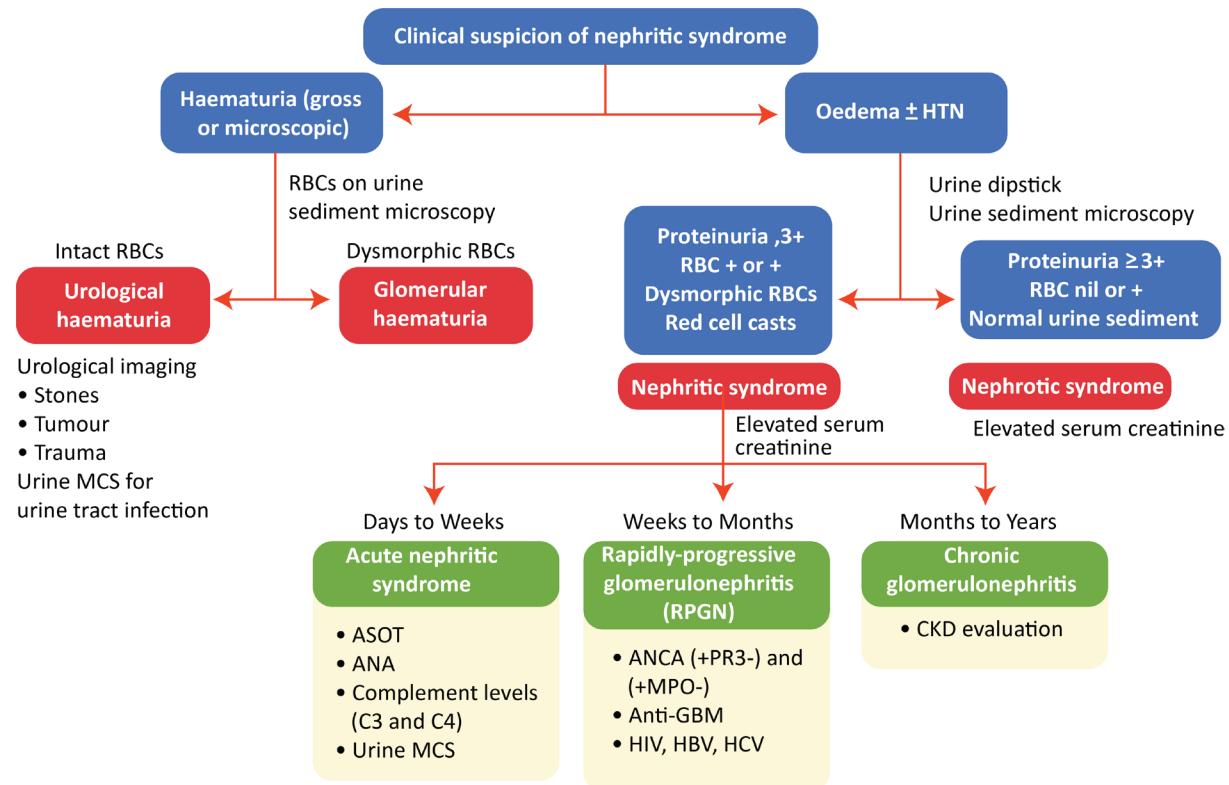
- Urine evaluation:
  - o Urine dipstick: proteinuria (+ to 3+), haematuria
  - o Urine sediment microscopy: dysmorphic RBCs, red cell casts
  - o Spot urine: UACR <300mg/g, UPCR <500mg/g

- UEC and eGFR
- Complete blood count
- Serum albumin
- C-reactive protein
- Blood culture

Others;

- Complement C3 and C4 levels
- ASOT titre, ANA, ANCA (MPO/p-ANCA and PR3/c-ANCA), antiGBM
- Serology for HIV, Hepatitis B surface antigen, Hepatitis C antibody
- Myeloma screen: Serum protein electrophoresis, Serum free light-chain assay, Urine Bence-Jones protein

### 3.2.5 Clinical approach to nephritic syndrome



ANA: antinuclear antibody; ANCA: anti-neutrophil cytoplasmic antibody; ASOT: anti-streptolysin O titre; CKD: chronic kidney disease; GBM: glomerular basement disease; HBV: hepatitis B virus; HCV: hepatitis C virus; HIV: human immunodeficiency virus; HTN: hypertension; MCS: microscopy, culture and sensitivity; RBC: red blood cell; UTI: urinary tract infection

Figure 4: Approach to nephritic syndrome

### 3.3.6 Management of nephritic syndrome

- Management of nephritic syndrome focuses on stabilizing the patient, controlling fluid overload and hypertension, identifying and treating the underlying cause, and preventing progression to acute or chronic kidney failure.
- Early nephrology consultation is essential, particularly in cases of worsening renal function or suspected rapidly progressive glomerulonephritis, to guide further investigations (including kidney biopsy) and initiate timely, targeted therapy.
- The table below outlines the structured management approach to nephritic syndrome, organized by clinical presentation and severity. It summarizes targeted interventions for key complications

Table 10: Management Approach to Nephritic Syndrome

| Condition                              | Management                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
|----------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Acute nephritic syndrome</b>        |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |
| Oedema                                 | <ul style="list-style-type: none"> <li>• Fluid restriction to less than 1.0-1.5L/day</li> <li>• Salt restriction to less than 5g NaCl (1 teaspoon) per day</li> <li>• Loop diuretic: e.g. Furosemide 40-80mg PO or IV</li> <li>• Target: daily morning weight to decrease by 1-2kg per day until patient is oedema-free</li> </ul>                                                                                                                                                      |
| Hypertension                           | <ul style="list-style-type: none"> <li>• Angiotensin blockade with ACE inhibitor or angiotensin receptor blocker: <ul style="list-style-type: none"> <li>o ACE inhibitor e.g. Enalapril 2.5-10mg BID</li> <li>o Angiotensin receptor blocker e.g. Losartan 25-50mg OD or BID</li> </ul> </li> <li>• Target BP: &lt;130/80mmHg</li> </ul>                                                                                                                                                |
| Gross haematuria:                      | <ul style="list-style-type: none"> <li>• Manage primary cause</li> <li>• Avoid tranexamic acid: may cause intravesical clots leading to urinary obstruction</li> <li>• Bladder 3-way irrigation with normal saline may be required</li> </ul>                                                                                                                                                                                                                                           |
| Azotaemia                              | <ul style="list-style-type: none"> <li>• Consult nephrologist: may require kidney biopsy</li> <li>• Nephrologist to guide management of primary cause</li> <li>• Avoid over-aggressive diuresis to prevent hypovolaemia</li> <li>• Remove and avoid nephrotoxins e.g. NSAIDs</li> <li>• Ensure no urinary obstruction</li> <li>• Monitor for and manage complications of acute kidney injury</li> <li>• Dialysis (haemodialysis or peritoneal dialysis) if indications exist</li> </ul> |
| Rapidly progressive glomerulonephritis | <ul style="list-style-type: none"> <li>• Consult nephrologist urgently: without treatment patient will progress to irreversible kidney failure</li> <li>• Will require kidney biopsy: crescentic glomerulonephritis will be seen</li> <li>• Monitor for and manage complications of kidney dysfunction</li> <li>• Dialysis (haemodialysis or peritoneal dialysis) if indications exist</li> </ul>                                                                                       |
| Chronic glomerulonephritis             | <ul style="list-style-type: none"> <li>• Manage as chronic kidney disease</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                    |

# Chapter 4: Acute Kidney Disease

## 4.1. Definition of Acute Kidney Injury (AKI)

A sudden loss (hours to days) of kidney function clinically manifested as increase in serum creatinine or reduced urine output:

- Increase in serum creatinine by  $26\mu\text{mol/L}$  within 48 hours  
or
- Increase in serum creatinine  $>1.5$  times baseline within 7 days (presuming the baseline is known)  
or
- Decreased urine volume  $<0.5\text{ml/kg/h}$  for 6 hours

## 4.2 Presentation of AKI

- Most patients with AKI have no clinical symptoms related to AKI.
- AKI diagnosed on the basis of routine laboratory blood tests.
- May have the following signs/symptoms:
  - o Reduced urine output
  - o Uraemic features:
    - Nausea and vomiting
    - Pruritus
    - Sleep Disturbance
    - Altered Mental Status
  - o Fluid overload features:
    - Facial Puffiness
    - Oedema
    - Hypertension
    - Shortness of Breath

## 4.3 Risk Factors for AKI

- o Hypovolaemia:
  - Volume depletion: vomiting, diarrhoea, blood loss, diuresis
  - Hypotension
  - Prolonged physical work in hot climate
- o Effective hypovolaemia:
  - Critically ill patient
  - Sepsis
  - Advanced liver disease
  - Heart failure
  - Cardiac surgery

- Wasp or bee stings
- Snake bites
- o For direct renal injury
  - Radio contrast imaging
  - Over the counter medication/herbal medication use/NSAIDs
  - Malignancies
  - Hyperuricemia
  - Hypercalcemia
- o For decreased renal reserve:
  - Advanced Age
  - Chronic kidney disease
- o Pregnancy (pregnancy-associated AKI (PrAKI)):
  - Early: abortal sepsis
  - Late: pre-eclampsia/eclampsia, post-partum haemorrhage

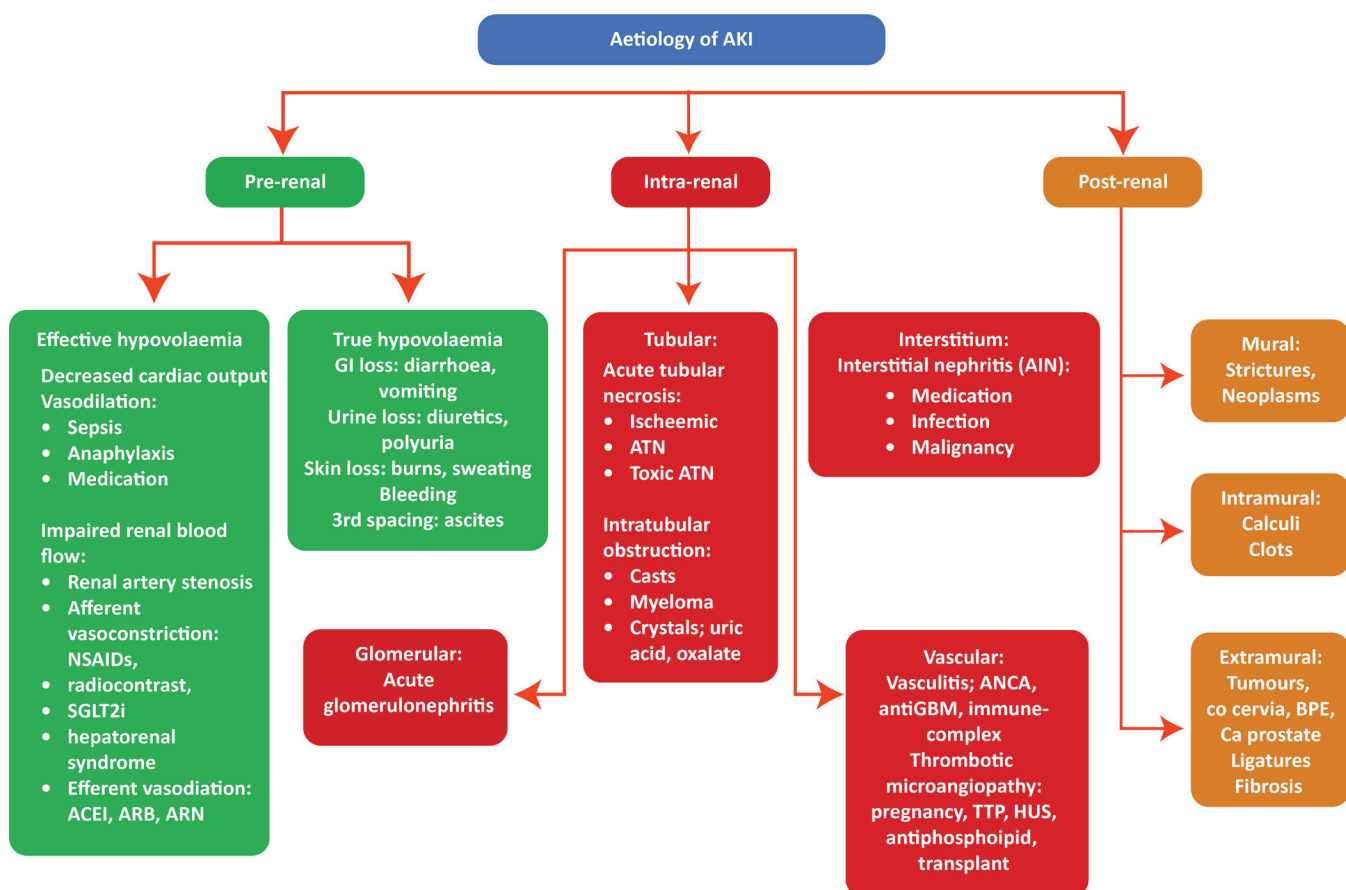
## 4.4 Staging of AKI

| STAGE    | SERUM CREATININE                                                               | URINE OUTPUT                                    |
|----------|--------------------------------------------------------------------------------|-------------------------------------------------|
| <b>1</b> | 1.5-1.9 X baseline or >26umol/L increase                                       | <0.5ml/kg/h for 6-12 h                          |
| <b>2</b> | 2.0-2.9 X baseline                                                             | <0.5ml/kg/h for ≥12h                            |
| <b>3</b> | ≥3.0 X baseline<br>or<br>Creatinine >350umol/L<br>or<br>Initiation of dialysis | <0.3ml/kg/h for >24hr<br>or<br>Anuria for >12hr |

## 4.5 Aetiology of AKI

Classified into:

1. Pre-renal: decreased blood supply to the glomerulus
2. Intra-renal: intrinsic injury to the glomerulus, tubules, interstitium, or intrarenal vessels
3. Post-renal: obstruction to urine flow



ACEi: angiotensin converting enzyme inhibitor; AKI: acute kidney injury; ANCA: anti-neutrophil cytoplasmic antibody; antiGBM: antiglomerular basement antibody; ARB: angiotensin receptor blocker; ARNI: angiotensin receptor – neprilysin inhibitor; ATN: acute tubular necrosis; BPE: benign prostatic enlargement; HUS: haemolytic-uraemic syndrome; NSAID: non-steroidal anti-inflammatory drug; SGLT2i: sodium-glucose-linked transporter 2 inhibitor; TTP: thrombotic thrombocytopenic purpura

Figure 5: Aetiology of AKI

## 4.6 Clinical Approach to AKI:

- The clinical approach to AKI begins with confirming the diagnosis using urine output and serum creatinine, followed by staging its severity based on established criteria.
- It requires early identification of complications, including electrolyte disturbances, acid–base imbalance, pulmonary oedema, and uraemic features.
- A systematic assessment of the underlying cause is essential, focusing on volume status, obstruction, nephrotoxic exposures, and systemic conditions.
- Appropriate laboratory and imaging investigations are then performed to guide management and address the precipitating cause.

|                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
|-------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>1. Confirm diagnosis of AKI</b><br> | <ul style="list-style-type: none"> <li>• <b>Measure urine output</b> <ul style="list-style-type: none"> <li>◦ If patient can follow instructions: <ul style="list-style-type: none"> <li>◦ Ask to void in measuring can</li> <li>◦ Check volume after 6h and divide by 6 to get hourly output</li> <li>◦ Divide hourly output by patient's weight to get urine flow rate in mL/kg/h</li> </ul> </li> <li>◦ If above not feasible or if urine obstruction suspected: <ul style="list-style-type: none"> <li>◦ Place indwelling urethral catheter</li> <li>◦ Monitor hourly urine output</li> </ul> </li> </ul> </li> <li>• <b>Measure creatinine:</b> <ul style="list-style-type: none"> <li>◦ Admission or last review creatinine</li> <li>◦ Repeat creatinine within 48h</li> <li>◦ Get difference between the 2 readings: confirm if &gt;26μmol/L</li> <li>◦ Get baseline creatinine if available:</li> <li>◦ Confirm if current creatinine is &gt;1.5 times baseline creatinine</li> </ul> </li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |
| <b>2. Stage AKI</b><br>                | <ul style="list-style-type: none"> <li>◦ Based on urine criteria and/or creatinine criteria</li> <li>◦ AKI stage is the worse of the two</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       |
| <b>3. Identify Complications</b><br>   | <ul style="list-style-type: none"> <li>◦ Electrolytes: <ul style="list-style-type: none"> <li>▪ Hyperkalaemia (serum K &gt;5.5mmol/L)</li> <li>▪ Hyponatraemia (serum Na &gt;145mmol/L)</li> <li>▪ Hyponatraemia (serum Na &lt;135mmol/L)</li> </ul> </li> <li>◦ BGA: <ul style="list-style-type: none"> <li>▪ Severe metabolic acidosis (arterial blood pH &lt;7.20, serum HCO<sub>3</sub> &lt;15mEq/L)</li> <li>▪ Severe metabolic alkalosis (arterial blood pH &gt;7.55, serum HCO<sub>3</sub> &gt;38mmol/L)</li> </ul> </li> <li>◦ Pulmonary oedema</li> <li>◦ Uraemic organ damage: <ul style="list-style-type: none"> <li>▪ Encephalopathy</li> <li>▪ Pericarditis</li> </ul> </li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
| <b>4. Assess for aetiology</b><br>   | <ul style="list-style-type: none"> <li>• <b>Assess for aetiology:</b> <ul style="list-style-type: none"> <li>◦ Assess volume status: <ul style="list-style-type: none"> <li>▪ Evidence of dehydration: dry oral mucosa, sunken eyes, decreased skin turgor, reduce urine volume</li> <li>▪ Evidence of fluid overload: oedema (ankle, periorbital, sacral), respiratory distress, lung crepitations or rhonchi</li> </ul> </li> <li>◦ Assess for urine obstruction: <ul style="list-style-type: none"> <li>▪ History of lower urinary tract symptoms</li> <li>▪ Palpate for full bladder</li> <li>▪ If in doubt, site urethral catheter</li> <li>▪ POCUS (point-of-care ultrasound) or conventional ultrasound KUB (kidneys, ureter, bladder) to assess bladder volume, evidence of hydronephrosis or hydroureter</li> </ul> </li> <li>◦ Assess for nephrotoxins: <ul style="list-style-type: none"> <li>▪ Recent nephrotoxic medication use: <ul style="list-style-type: none"> <li>• NSAIDs, angiotensin blockers, chemotherapeutic agents, herbal medication, intravenous contrast media, targeted therapy</li> </ul> </li> <li>▪ Get medication history <ul style="list-style-type: none"> <li>• Evaluate all recent treatment charts</li> <li>• History or health record assessment for intravenous contrast media use</li> </ul> </li> </ul> </li> <li>◦ Evaluate for particular diseases/syndromes: <ul style="list-style-type: none"> <li>▪ Heart failure</li> <li>▪ Advanced liver disease</li> <li>▪ Sepsis</li> <li>▪ Vasculitis</li> <li>▪ Thrombocytopenia-related (thrombotic microangiopathies)</li> <li>▪ Infections: malaria, streptococcal, gastroenteritis</li> <li>▪ Pregnancy-related: pre-eclampsia/eclampsia, post-partum haemorrhage</li> </ul> </li> <li>◦ Laboratory evaluation: <ul style="list-style-type: none"> <li>▪ Serial renal function tests (UEC): at least daily</li> <li>▪ Urinalysis and microscopy</li> <li>▪ Complete blood count</li> <li>▪ Septic screen: CRP, PCT, cultures</li> <li>▪ Liver function test</li> <li>▪ Serum calcium, phosphorous</li> <li>▪ Uric acid</li> <li>▪ Blood gas analysis</li> </ul> </li> <li>◦ KUB ultrasound</li> <li>◦ Other relevant tests based on suspected aetiology</li> </ul> </li> </ul> |
| <b>5. Investigations</b><br>         | <ul style="list-style-type: none"> <li>• Investigations guided by suspected aetiology and severity of AKI</li> <li>• Investigate/treat complications</li> <li>• Review medication and stop nephrotoxins</li> <li>• Monitor urine output, renal function and electrolytes</li> <li>• Consider early referral to nephrologist for severe AKI (stage 3) or if the cause is unclear</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                |

Figure 6: Clinical Approach to AKI

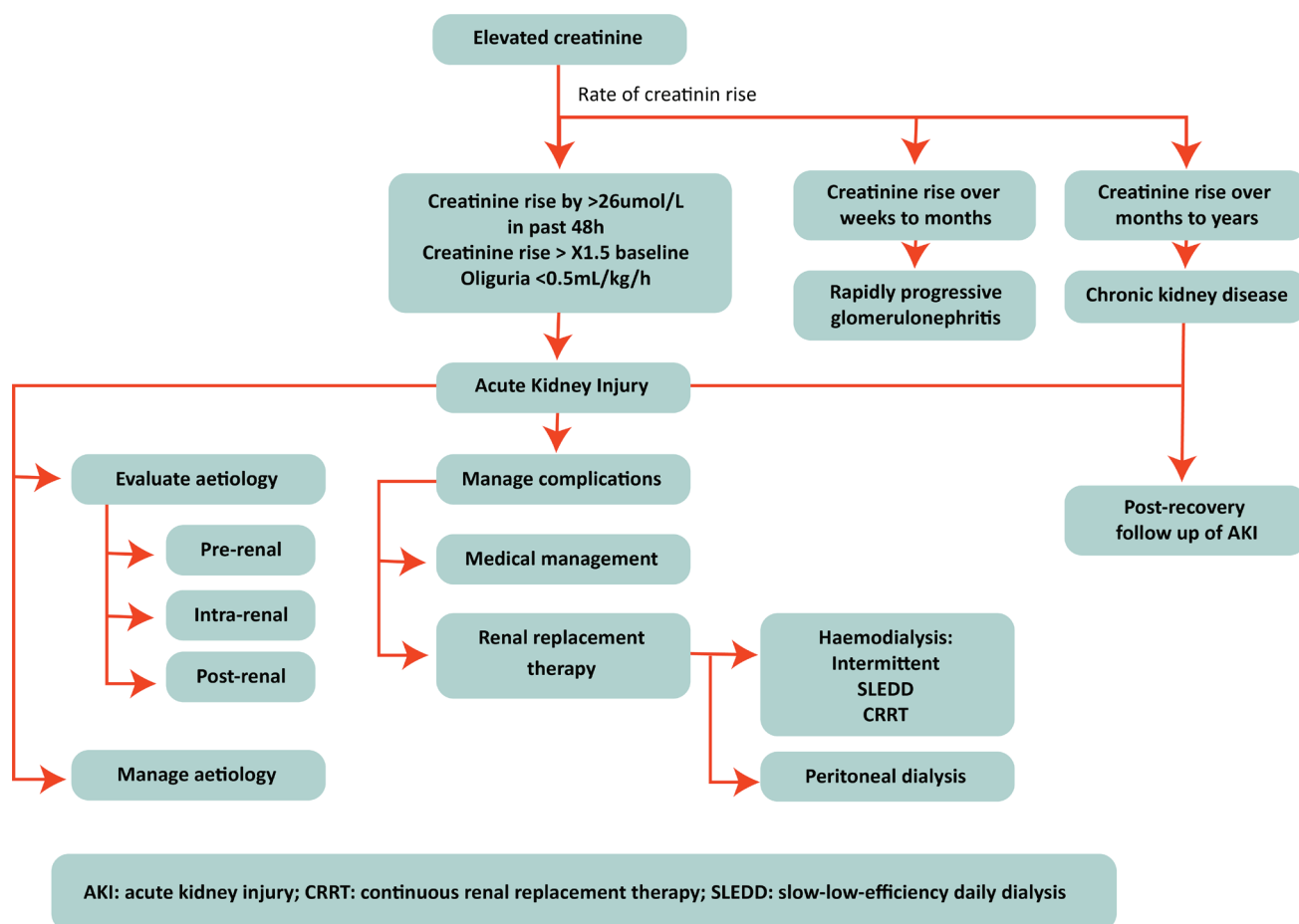


Figure 7: Clinical Approach to AKI

#### AKI emergencies include:

- Hyperkalaemia
- Metabolic acidosis
- Pulmonary oedema
- Uraemic encephalopathy
- Uraemic pericarditis
- Other severe electrolyte disorders:
  - o Hyper- or hypo-natraemia
  - o Hyper- or hypocalcaemia
  - o Hyper- or hypomagnesaemia
  - o Hyper- or hypo-phosphataemia

## 4.6.2 Management of emergencies

| Type of Emergency         | Management                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
|---------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Hyperkalemia</b>       | <ul style="list-style-type: none"> <li>Stabilize myocardium from malignant arrhythmias: <ul style="list-style-type: none"> <li>Calcium intravenously: <ul style="list-style-type: none"> <li>Calcium gluconate</li> <li>Calcium chloride</li> </ul> </li> </ul> </li> <li>Shift potassium into cells: <ul style="list-style-type: none"> <li>Insulin</li> <li>50% Dextrose</li> <li>Others: <ul style="list-style-type: none"> <li>B2 agonist: nebulized salbutamol</li> <li>Sodium bicarbonate</li> </ul> </li> </ul> </li> <li>Bind potassium in GIT: <ul style="list-style-type: none"> <li>Potassium-binding resins: <ul style="list-style-type: none"> <li>Sodium polystyrene 15-30g TID</li> <li>Calcium polystyrene 15-30g TID</li> </ul> </li> <li>Newer agents: <ul style="list-style-type: none"> <li>Sodium zirconium cyclocilate</li> <li>Patiromer</li> </ul> </li> </ul> </li> <li>If medical therapy fails, for emergency dialysis</li> </ul> |
| <b>Metabolic acidosis</b> | <ul style="list-style-type: none"> <li>Improve tissue perfusion: <ul style="list-style-type: none"> <li>Intravenous volume replacement</li> <li>Red blood cell transfusion for anaemia (Hb &lt;7.0g/dL)</li> </ul> </li> <li>Intravenous sodium bicarbonate: <ul style="list-style-type: none"> <li>Given if: <ul style="list-style-type: none"> <li>pH &lt;7.10</li> <li>Serum bicarbonate &lt; 15mmol/L</li> </ul> </li> <li>Goal is to raise the serum pH&gt;7.1</li> <li>Formula for calculating the required amount of sodium bicarbonate: <ul style="list-style-type: none"> <li>0.4 X Base Excess (24- serum bicarbonate level) x patient's weight</li> <li>½ of deficit - given within 30min</li> <li>½ of deficit - given within the next 4hours</li> </ul> </li> <li>Repeat BGAs after 4hours</li> <li>If not improving, for HD</li> </ul> </li> </ul>                                                                                             |

|                               |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
|-------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Pulmonary oedema</b>       | <ul style="list-style-type: none"> <li>• Prop up in bed</li> <li>• O2 supplementation if <math>SO_2 &lt; 92\%</math></li> <li>• Fluid restriction to <math>&lt; 1.5L/24h</math></li> <li>• Furosemide: <ul style="list-style-type: none"> <li>o Intravenous preferred to oral</li> <li>o 40-80mg IV stat</li> <li>o If poor diuresis (less than 200mL in next 2h), repeat Furosemide at double the dose</li> <li>o If good diuresis but patient still in distress, repeat Furosemide dose after 4h</li> </ul> </li> <li>• If respiratory failure imminent (severe distress, persisting hypoxia): refer to a facility with ICU for: <ul style="list-style-type: none"> <li>o Ventilation: invasively or non-invasively</li> <li>o Urgent dialysis for ultrafiltration</li> </ul> </li> <li>• If medical therapy fails, for urgent haemodialysis</li> </ul> |
| <b>Uraemic encephalopathy</b> | <ul style="list-style-type: none"> <li>• Nurse patient in calm environment</li> <li>• Reassure patient</li> <li>• Sedation if agitated</li> <li>• Antipsychotics</li> <li>• Urgent dialysis</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    |
| <b>Uraemic pericarditis</b>   | <ul style="list-style-type: none"> <li>• Urgent dialysis</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       |

### 4.6.3 Manage the primary cause of AKI

Primary causes can be categorised as:

- Pre-renal AKI
- Sepsis
- Post renal AKI
- Intrarenal AKI

## A. Manage pre-renal AKI

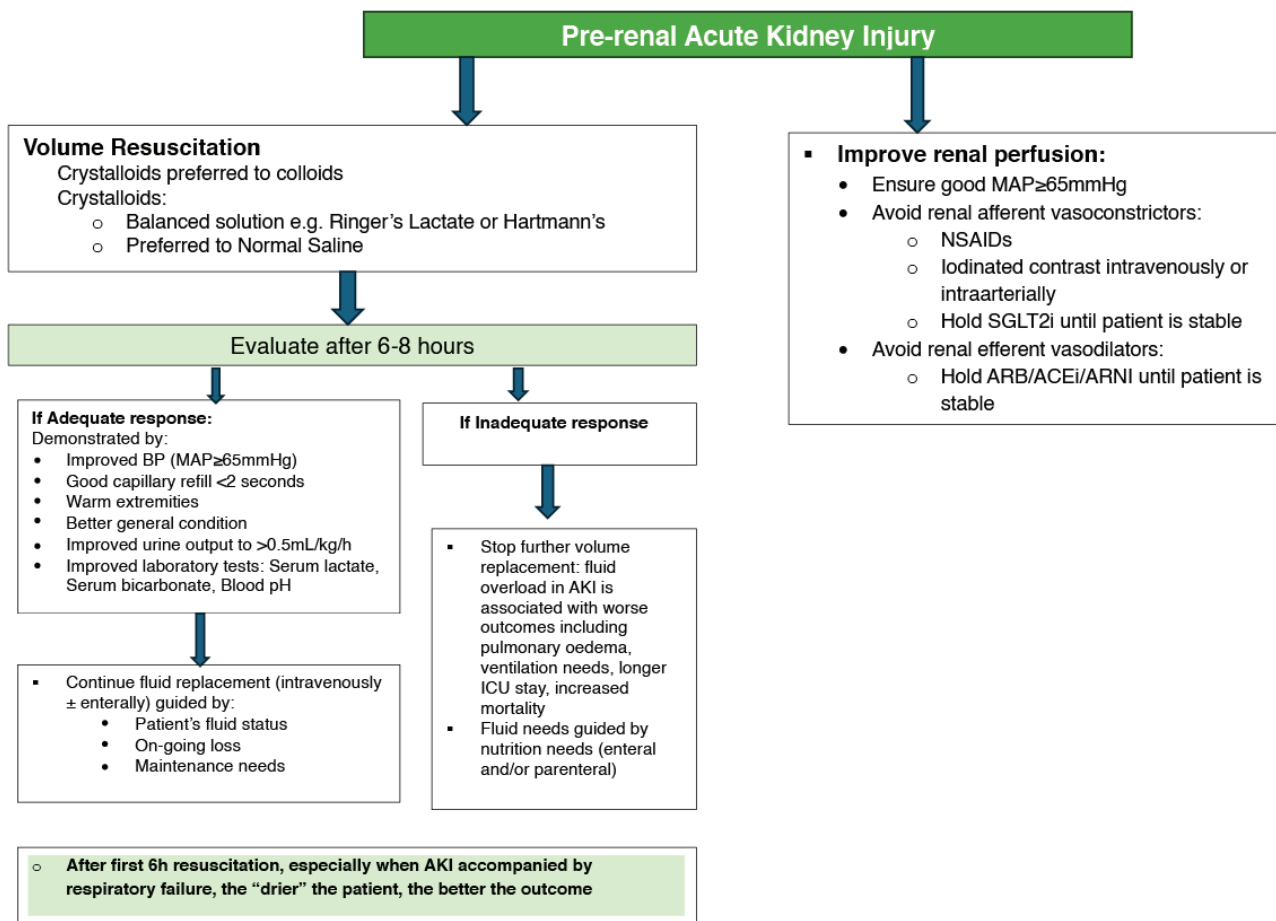


Figure 8: Management of AKI

## B. Manage sepsis

- Identify focus
- Appropriate sampling for culture: blood, urine, respiratory sample, stool, wound swab, CSF
- Empirical broad-spectrum antimicrobial:
- Follow institution guidelines
- Hit hard:
  - Ensure antibiotic cover for both Gram negative (especially Enterobacteriaceae) and Gram positive (consider MRSA)
  - Administer highest dose of antibiotic
  - Consider pharmacokinetics: time dependent or concentration dependent action, renal-dose (eGFR) adjustment
  - Avoid nephrotoxic antibiotics
- Hit fast:
  - Ensure antibiotic given within first 2h of medical contact
- Manage focus, e.g. drainage of abscess, removal of foreign body

### C. Manage post-renal AKI

- Identify location and cause of obstruction
- Renal imaging:
  - Ultrasound KUB
  - CT KUB
- Relieve obstruction (urinary decompression)
  - Bladder outlet obstruction:
    - Urethral catheter
    - Suprapubic catheter
  - Ureteric obstruction:
    - Percutaneous nephrostomy (PCN)
    - Ureteric stenting with double-J (DJ) stents
- Involve urologist early: get definitive management of obstruction
- In women, actively look for ca cervix
- In men, actively look for prostatic enlargement

### D. Manage intra-renal AKI

- Acute tubular necrosis (ATN) is the most common cause
  - Usually ischaemic from progression of pre-renal AKI
  - Also, septic ATN or toxic ATN
- Consult nephrologist if non-ATN cause is a consideration

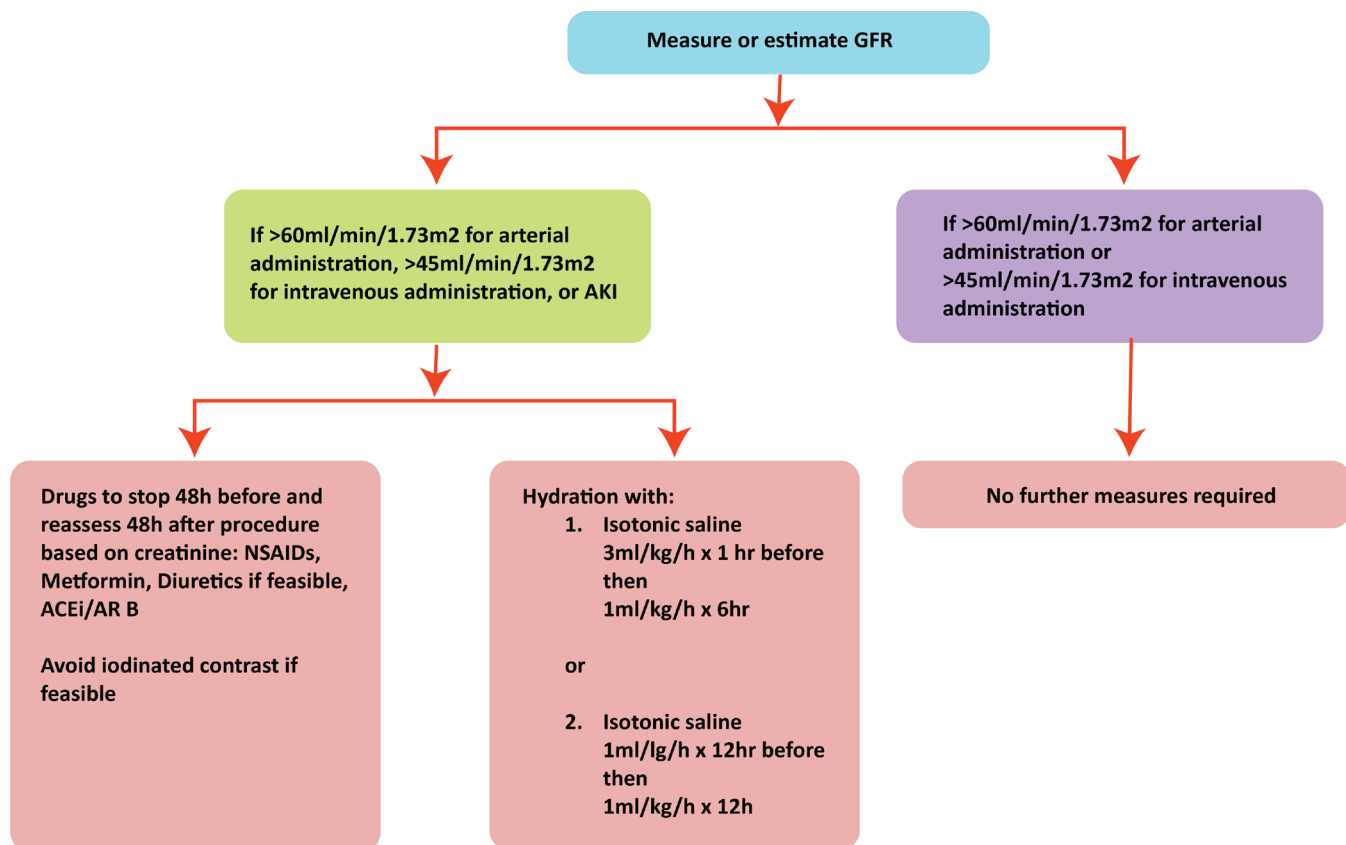
## 4.6.4 Approach to manage AKI

| Pre-renal                                                                                                                                                                                                                                                                                         | Intra-renal                                                                                                                                                                                                                                                       | Post Dialysis                                                                                                                                                                                                                                                                                                                                                                                |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Correct hemodynamic derangement</li><li>• If hypovolemic - IV fluids</li><li>• if hypervolemic - Diuretic</li><li>• If septic and non responsive to fluids give vasopressors</li><li>• if low contractility - Afterload reduction +/- inotropes</li></ul> | <ul style="list-style-type: none"><li>• Treat the underlying disease</li><li>• Acute Tubular Nephritis - No specific treatment</li><li>• Acute Interstitial Nephritis - consider steroids if not improving with discontinuation of causative medication</li></ul> | <ul style="list-style-type: none"><li>• Relieve obstruction</li><li>• Ureter obstruction - nephrostomy tubes and or ureteral stent</li><li>• Neurogenic bladder - intermittent straight catheter or long term foley</li><li>• UTI - antibiotics +/- temporary catheter</li><li>• medication - stop any nephrotoxic agent</li><li>• BPH - alpha blockers + temporary foley catheter</li></ul> |

BPH: benign prostatic hypertrophy; IV: intravenous; UTI: urinary tract infection

## 4.7 Prevention of Contrast AKI

- The figure below presents a simple algorithm for preventing contrast-associated acute kidney injury.
- Renal function (GFR) is assessed before contrast exposure, and patients are stratified by risk.
- Those with reduced GFR or AKI require preventive measures—temporary cessation of nephrotoxic drugs, consideration of avoiding iodinated contrast, and isotonic saline hydration—while patients with adequate GFR need no additional intervention.



ACEi: angiotensin converting enzyme inhibitor; AKI: acute kidney injury; ARB: angiotensin receptor blocker; GFR: glomerular filtration rate; NSAIDs: non-steroidal anti-inflammatory drugs

Figure 9: Prevention of Contrast AKI

## 4.8 Renal Replacement Therapy in AKI (Acute RRT)

Always consult nephrologist for guidance on the indication, choice, and prescription of RRT

### 4.8.1 Indications for acute dialysis

- Refractory metabolic Acidosis
- Refractory Hyperkalemia
- Refractory Volume overload
- Uremic complications:
  - o Uraemic encephalopathy
  - o Uraemic pericarditis
    - Uraemic gastritis
    - Uraemic pruritus
- Anuria >24h

### 4.8.2 Dialysis modalities

Various modalities of dialysis can be utilized for AKI, and include Intermittent Haemodialysis (IHD), sustained slow efficiency dialysis (SLEDD), Continuous renal replacement therapy CRRT and Peritoneal Dialysis.

**Refer to Chapter 10 on dialysis**

# Chapter 5: Chronic Kidney Disease

## 5.1. Definition of Chronic Kidney Disease (CKD)

CKD is defined as abnormalities of kidney structure or function, present for a minimum of 3 months, with implications for health.

|                        |                                                                                                              |
|------------------------|--------------------------------------------------------------------------------------------------------------|
| Structural Abnormality | Abnormalities on imaging<br>Histological abnormalities<br>Kidney transplant                                  |
| Functional Abnormality | Decreased GFR $< 60 \text{ mL/min/1.73m}^2$<br>Albuminemia<br>Urine sediment abnormalities (Haematuria/Cast) |

## 5.2. Aetiology of CKD

Commonest aetiologies of CKD:

- Diabetes:
  - Diabetic nephropathy
  - Other diabetic kidney diseases
- Hypertension:
  - Benign nephrosclerosis
  - Malignant nephrosclerosis
- Chronic glomerulonephritis
- Cystic kidney diseases:
  - Polycystic kidney disease:
    - Autosomal dominant
    - Autosomal recessive
  - Other cystic kidney diseases
- Urinary tract obstruction:
  - Congenital: Congenital Anomalies of the Kidney and Urinary Tract (CAKUT)
  - Acquired:
    - Benign: strictures, stones, BPH, retroperitoneal fibrosis
    - Malignant: ca prostate, ca cervix

### Complications of CKD

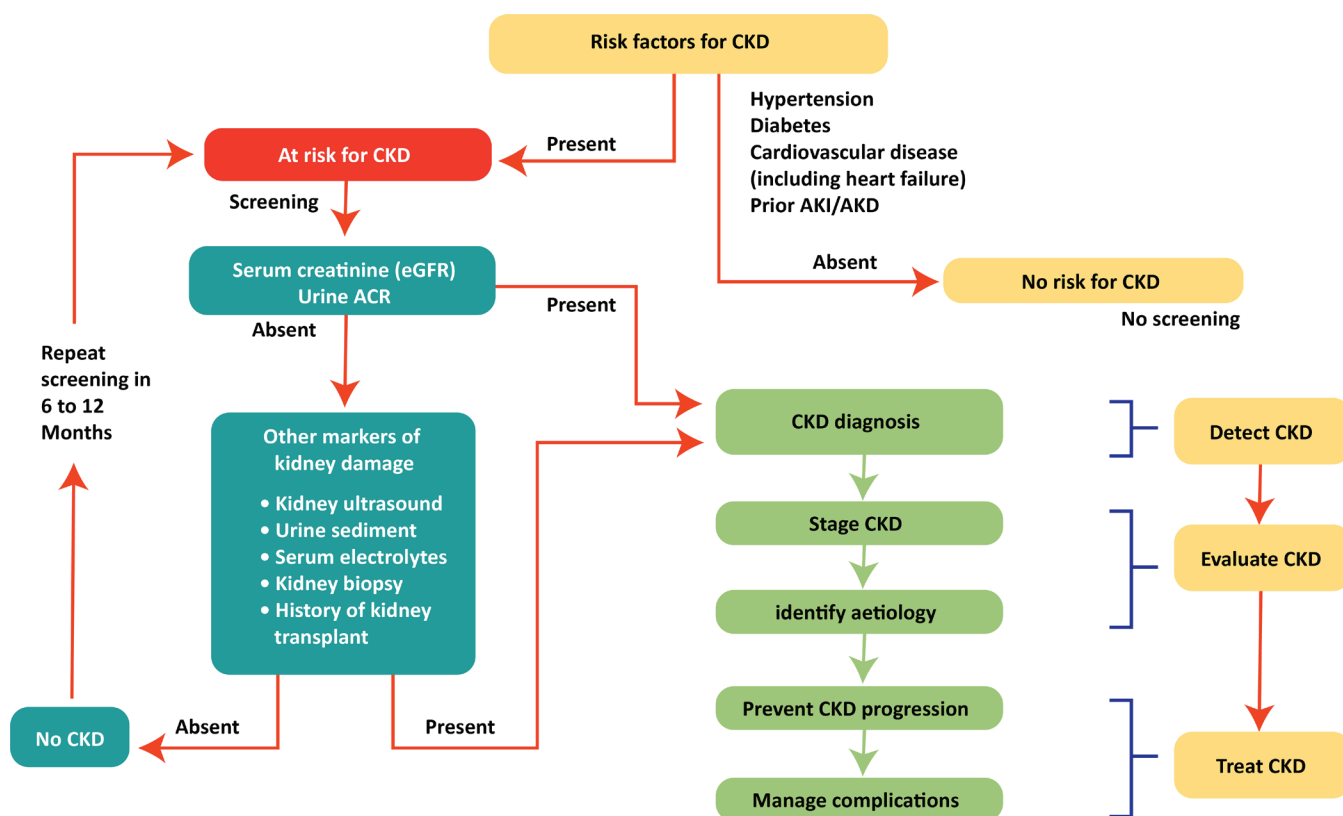
- The complications of CKD are as a result of failure of kidney function.
- Fluid and salt balance:
  - Fluid overload:
    - Peripheral oedema
    - Pulmonary oedema
    - 3rd space fluid collection: ascites, pleural effusion
  - Hypertension

- Nitrogenous waste accumulation (azotaemia)
  - o Uraemia:
    - Nausea, vomiting, anorexia
    - Pruritus
    - Insomnia
  - o Uraemic encephalopathy
  - o Uraemic pericarditis
- Electrolyte and acid-base abnormalities:
  - o Hyperkalaemia
  - o Metabolic acidosis
  - o Hyperphosphataemia
- Endocrine abnormalities:
  - o CKD bone and mineral disorders:
    - Hyperparathyroidism:
    - Secondary hyperparathyroidism: renal osteodystrophy
    - Tertiary hyperparathyroidism
    - Hypocalcaemia
  - o Renal anaemia

## 5.3 Clinical Approach to CKD

Clinical approach to CKD involves a systematic pathway aimed at ensuring early detection, appropriate evaluation, prevention of disease progression, and comprehensive management of CKD and its complications, with periodic rescreening for individuals who remain at risk. This approach includes the following key steps:

- Identify individuals at risk: Hypertension, diabetes, cardiovascular disease, prior AKI/ AKD
- Screen appropriately: Serum creatinine (eGFR) and urine ACR
- Confirm kidney damage: Additional markers (urine sediment, imaging, electrolytes, biopsy if indicated)
- Diagnose and stage CKD
- Identify underlying aetiology
- Prevent progression: Control BP, proteinuria, and modifiable risk factors
- Manage complications: Anaemia, mineral bone disorder, acidosis, cardiovascular risk
- Repeat screening in at-risk individuals every 6–12 months



AKD: acute kidney disease; AKI: acute kidney injury; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate

Figure 10: Clinical Approach to CKD

## 5.4 Screening for CKD

### Key Facts

- Approximately 1 in 10 individuals has CKD.
- Universal population screening is not cost-effective and is unlikely to significantly improve health outcomes.
- Targeted screening of high-risk individuals is recommended.

### High-Risk Groups Requiring Testing

- Individuals with:
  - o Hypertension
  - o Diabetes mellitus
  - o Cardiovascular disease
  - o Family history of kidney disease
  - o History of acute kidney injury (AKI)
  - o Medical conditions affecting kidney function, including:
    - o Systemic lupus erythematosus
    - o HIV infection
    - o Obesity

- o Genetic risk factors
- High-risk occupations or environmental exposure to nephrotoxins

#### Screening Tools

- Urine albumin measurement (urine albumin-to-creatinine ratio [ACR])
- Assessment of glomerular filtration rate (eGFR)

#### Confirmatory Testing

Repeat testing is required following incidental detection of:

- Elevated urine ACR or positive urine dipstick
- Persistent haematuria
- Reduced eGFR

## 5.5 Diagnosis of CKD

CKD is diagnosed using the diagnostic criteria below:

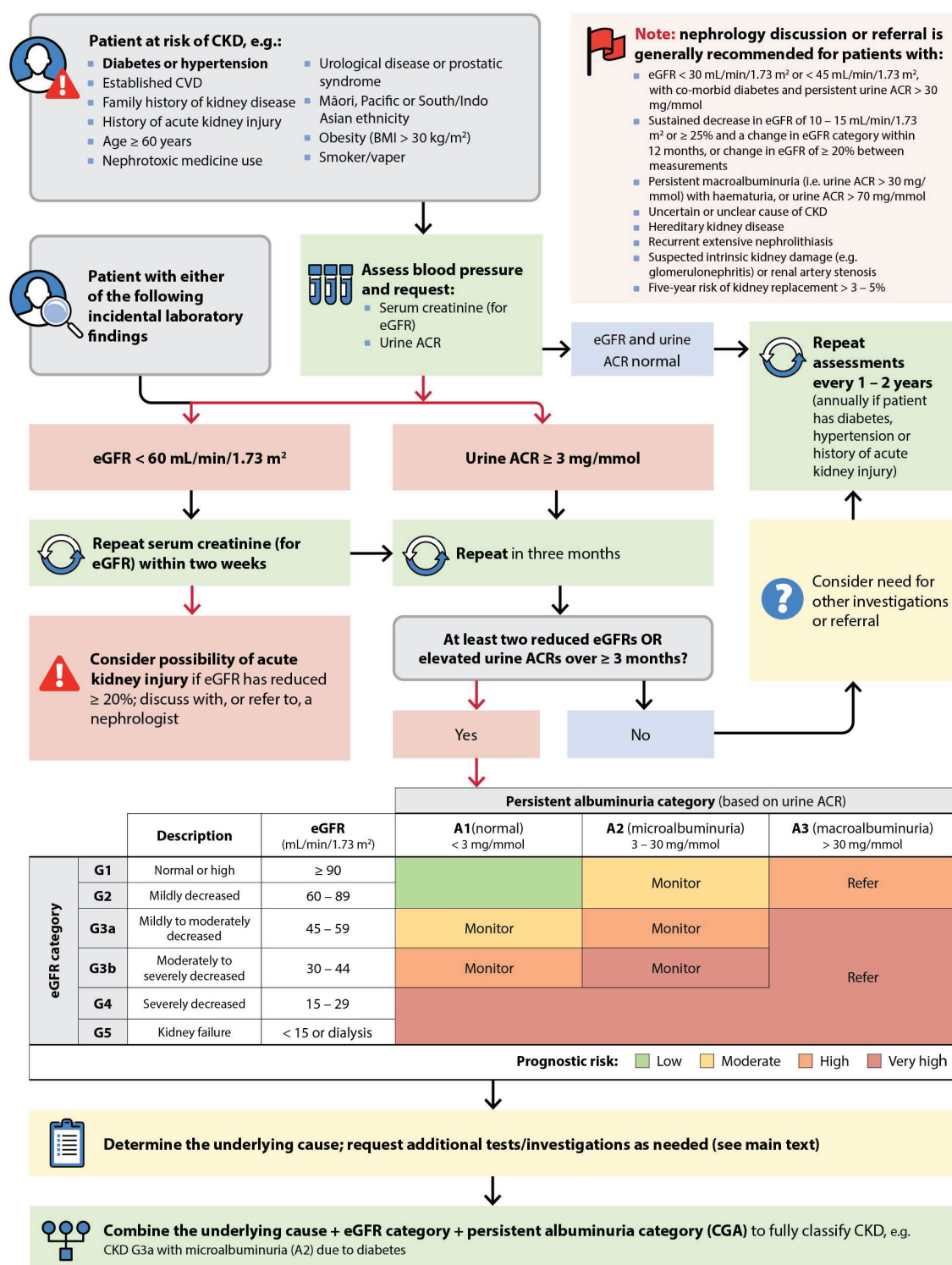
*Table 11: Criteria for Chronic Kidney Disease*

| Either of the following presents for a minimum of 3 months |                                                                                                                                                                                                                                                                                                                                                                                                                                   |
|------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Category                                                   | Criteria                                                                                                                                                                                                                                                                                                                                                                                                                          |
| Markers of kidney damage (1 or more)                       | <ul style="list-style-type: none"> <li>• Albuminuria (ACR <math>\geq 30</math> mg/g [<math>\geq 3</math> mg/mmol])</li> <li>• Urine sediment abnormalities</li> <li>• Persistent hematuria</li> <li>• Electrolyte and other abnormalities due to tubular disorders</li> <li>• Abnormalities detected by histology</li> <li>• Structural abnormalities detected by imaging</li> <li>• History of kidney transplantation</li> </ul> |
| Decreased GFR                                              | GFR $< 60$ ml/min per $1.73 \text{ m}^2$<br>(GFR categories G3a–G5)                                                                                                                                                                                                                                                                                                                                                               |

ACR = albumin-to-creatinine ratio; GFR = glomerular filtration rate •

- Proof of chronicity of kidney disease can be established by:
  - o review of past measurements:
    - eGFR
    - albuminuria or proteinuria
    - urine microscopic examinations
  - o imaging findings:
    - reduced kidney size
    - reduction in cortical thickness
    - loss of corticomedullary differentiation
  - o kidney pathological findings such as fibrosis and atrophy
  - o medical history, especially conditions known to cause or contribute to CKD
  - o repeat measurements within and beyond the 3-month point
- **Do not assume chronicity based upon a single abnormal level for eGFR and ACR, as the finding could be the result of a recent acute kidney injury (AKI) event or acute kidney disease (AKD).**

## 5.6 Clinical Approach to Diagnosing CKD



ACR: albumin: creatinine ratio; BMI: body mass index; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate

Retrieved from <https://bpac.org.nz/2022/docs/ckd.pdf> on 17th February 2026

Figure 11: Clinical approach to Diagnosing CKD

## 5.7 CGA Staging of Chronic Kidney Disease (CKD)

### 5.7.1 CGA staging classifies CKD using three components:

- C – Cause (aetiology of kidney disease)
- G – GFR category (level of kidney function)
- A – Albuminuria category (degree of kidney damage)

### 5.7.2 C – Cause of CKD

|                                                                                                                                                                                                                                                                                                                                              |                                                                                                                                                                                                                                                                                                                                                                                                                |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Establish the cause of CKD using:</b> <ul style="list-style-type: none"><li>• Clinical context</li><li>• Personal and family history</li><li>• Social and environmental factors</li><li>• Medications</li><li>• Physical examination</li><li>• Laboratory measures</li><li>• Imaging</li><li>• Genetic and pathologic diagnosis</li></ul> | <b>Identify the underlying aetiology whenever possible</b> , cause informs prognosis and treatment strategy: <ul style="list-style-type: none"><li>• Diabetes mellitus</li><li>• Hypertension</li><li>• Glomerulonephritis</li><li>• Tubulointerstitial disease</li><li>• Polycystic kidney disease</li><li>• Obstructive uropathy</li><li>• Congenital/hereditary disorders</li><li>• Unknown cause</li></ul> |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|

### 5.7.3 G – GFR categories (ml/min/1.73 m<sup>2</sup>)

| G Category | GFR (ml/min/1.73 m <sup>2</sup> ) | Description              |
|------------|-----------------------------------|--------------------------|
| G1         | ≥90                               | Normal or high           |
| G2         | 60–89                             | Mildly decreased         |
| G3a        | 45–59                             | Mild–moderate decrease   |
| G3b        | 30–44                             | Moderate–severe decrease |
| G4         | 15–29                             | Severe decrease          |
| G5         | <15                               | Kidney failure           |

### 5.7.4 A – Albuminuria categories

| A Category | ACR (mg/g) | ACR (mg/mmol) | Description                |
|------------|------------|---------------|----------------------------|
| A1         | <30        | <3            | Normal to mildly increased |
| A2         | 30–300     | 3–30          | Moderately increased       |
| A3         | >300       | >30           | Severely increased         |

Table 12 Staging of CKD

| Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012            |     |                                  |       | Persistent albuminuria categories                    |                                                     |                                                |
|---------------------------------------------------------------------------|-----|----------------------------------|-------|------------------------------------------------------|-----------------------------------------------------|------------------------------------------------|
|                                                                           |     |                                  |       | Description and range                                |                                                     |                                                |
|                                                                           |     |                                  |       | A1                                                   | A2                                                  | A3                                             |
|                                                                           |     |                                  |       | Normal to mildly increased<br><30 mg/g<br><3 mg/mmol | Moderately increased<br>30–300 mg/g<br>3–30 mg/mmol | Severely increased<br>>300 mg/g<br>>30 mg/mmol |
| GFR categories (mL/min per 1.73 m <sup>2</sup> )<br>Description and range | G1  | Normal or high                   | ≥90   | Green                                                | Yellow                                              | Orange                                         |
|                                                                           | G2  | Mildly decreased                 | 60–89 | Green                                                | Yellow                                              | Orange                                         |
|                                                                           | G3a | Mildly to moderately decreased   | 45–59 | Yellow                                               | Orange                                              | Red                                            |
|                                                                           | G3b | Moderately to severely decreased | 30–44 | Orange                                               | Red                                                 | Red                                            |
|                                                                           | G4  | Severely decreased               | 15–29 | Red                                                  | Red                                                 | Red                                            |
|                                                                           | G5  | Kidney failure                   | <15   | Red                                                  | Red                                                 | Red                                            |
|                                                                           | G5  |                                  |       | Red                                                  | Red                                                 | Red                                            |

Green, low risk (if no other markers of kidney disease, no CKD); yellow, moderately increased risk; orange, high risk; red, very high risk (pgeat:-1)

Green, low risk (if no other markers of kidney disease, no CKD); yellow, moderately increased risk; orange, high risk; red, very high risk

Yellow, moderately increased risk; orange, high risk; red, very high risk

Red, very high risk

## 5.8 Principles of CKD Management

- Recognize GFR stages at which interventions are important:

|                     |                                                                                                                                                                                                                                                                                                             |
|---------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| At eGFR <60 ml/min: | <ul style="list-style-type: none"> <li>Interventions to retard or prevent the progression of CKD should be instituted.</li> </ul>                                                                                                                                                                           |
| At eGFR <30 ml/min: | <ul style="list-style-type: none"> <li>Options for renal replacement need to be considered.</li> <li>Consider preparing vascular or peritoneal dialysis access</li> <li>Discuss transplantation option</li> </ul>                                                                                           |
| At GFR <15 ml/min:  | <ul style="list-style-type: none"> <li>Watch for and manage complications such as hypertension, fluid overload, electrolyte disturbances and malnutrition</li> <li>Ensure vascular access or PD access is ready</li> <li>Discuss pre-emptive kidney transplantation if potential donor available</li> </ul> |

- Appropriately refer to a nephrologist when there is:
  - Abnormal renal imaging e.g., cystic kidney disease, at any GFR
  - Persistent proteinuria and/or haematuria
  - Low eGFR:
    - eGFR <60 ml/min (recommended)
    - eGFR <30 ml/min (mandatory)

## 5.9 Prevention of Progression of CKD

### **i) CKD progresses towards worse stages of CKD because of:**

- Ongoing kidney injury from initial aetiology
- CKD-related hypertension
- Intraglomerular hypertension from compensatory hyperfiltration of remaining glomeruli
- Episodes of acute kidney injury occurring in the background of CKD
- Continued NSAID use

### **ii) Aim to prevent progression of CKD into end-stage kidney failure by addressing the factors above**

### **iii) Monitor for progression of CKD based upon GFR and ACR categories:**

- At least annually:
  - Albuminuria
  - GFR
- More frequently:

◦ For individuals at higher risk of CKD progression when measurement will impact therapeutic decisions

### **iv) Warning thresholds during monitoring (subsequent test) that warrant evaluation:**

- For GFR monitoring:
  - A reduction in eGFR of >20%
  - A reduction in eGFR of >30% in people with CKD who initiate hemodynamically active therapies, e.g. ACEi or ARB or SGLT2i
- For albuminuria monitoring:
  - A doubling of the ACR
- If these warning thresholds occur, evaluate for possible:
  - AKI occurring in CKD patient:
    - Pre-renal from:
      - Over-diuresis
      - Over-control of BP
      - Haemodynamic effect (decreased intraglomerular pressure) of ACEi, ARB, ARNI, SGLT2i, NSAIDs
    - Intra-renal from: acute TIN (drug-related)
    - Post-renal from new urinary tract obstruction
  - Progression of primary aetiology, e.g.:
    - Failed immunosuppression of immune-mediated kidney disease
    - Poorly controlled diabetes in diabetic kidney disease
  - Uncontrolled hypertension

Table 13: Frequency of Repeat Testing of Creatinine and Albumin in CKD1

| GFR (ml/min/1.73M2)      | Albuminuria          | Parameter                                                                  | Frequency |
|--------------------------|----------------------|----------------------------------------------------------------------------|-----------|
| >= 60 (CKD 1 & 2)        | <30mg/g              | Creatinine, microalbumin                                                   | yearly    |
|                          | 30- 300 mg/g         | Creatinine, microalbumin                                                   | 6-monthly |
| 30 - 60 (CKD 3)          | <30mg/g              | Creatinine, microalbumin                                                   | 6-monthly |
|                          | 30 - 300mg/g         | Creatinine, microalbumin                                                   | 6-monthly |
|                          | >300mg/g             | Creatinine, microalbumin                                                   | 3-monthly |
| 15 - 29 (CKD 4)          | any albumin level    | UECs, microalbumin, hemoglobin, calcium                                    | 3-monthly |
| <15 (CKD 5 pre-dialysis) | any level of albumin | UECs<br>hemoglobin,<br>calcium, phosphorus,<br>bicarbonate<br>Iron profile | 6-weekly  |

## 5.10 Management of CKD

Management of CKD involves:

- Delaying CKD progression
- Managing CKD complications
- Identifying the need for and initiating RRT

CKD patients should be managed with a comprehensive treatment strategy to reduce risks of progression of CKD and its associated complications using:

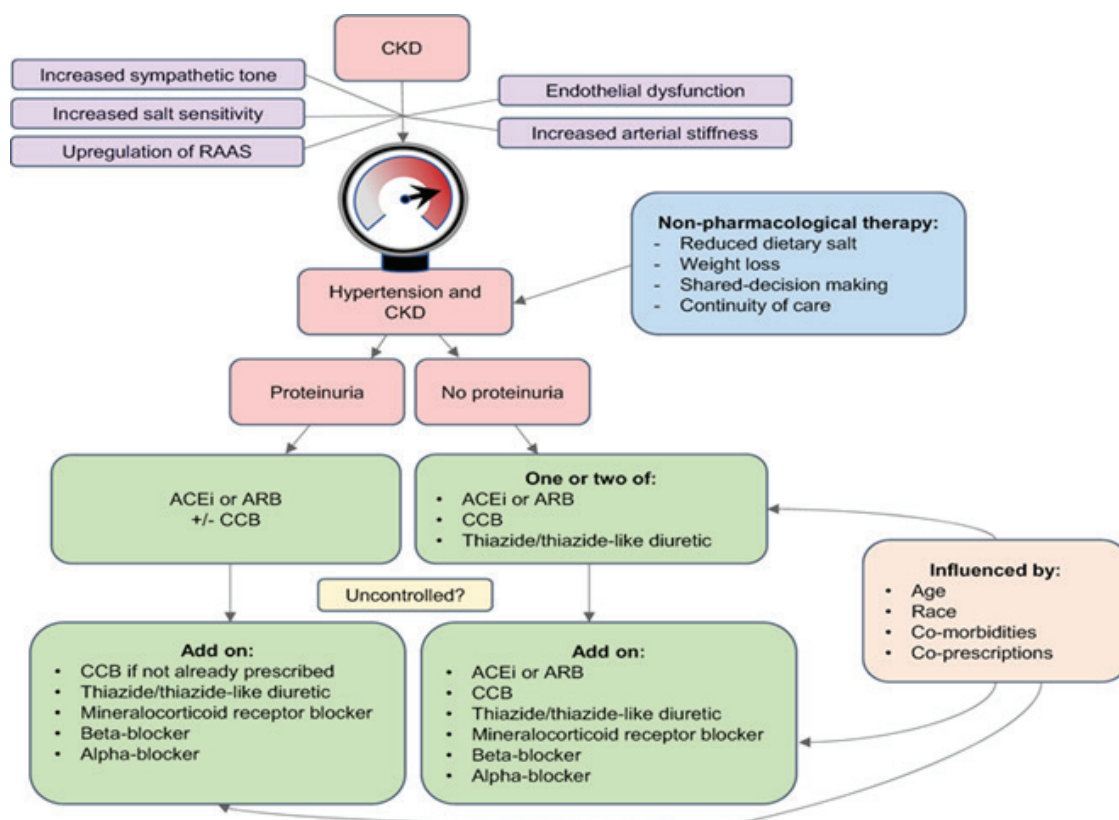
- Lifestyle modification
- Medications

### 5.10.1 Lifestyle modification

- Physical activity:
  - o Moderate-intensity physical activity for a cumulative duration of at least 150 minutes per week, or to a level compatible with their cardiovascular and physical tolerance
- Optimal body mass index (BMI):
  - Encourage weight loss in people with obesity and CKD
- Diet:
  - Adopt healthy and diverse diets with:
    - o a higher consumption of plant-based foods compared to animal-based foods
    - o a lower consumption of ultra-processed foods.
- Use renal dietitians or accredited nutrition providers
  - Protein intake:
    - Maintain protein intake of 0.8 g/kg body weight/day in adults with CKD G3–G5
    - Avoid high protein intake (>1.3 g/kg body weight/d)
    - Very low–protein diet (0.3–0.4 g/kg body weight/d) used only if:
      - o Patient is willing and able
      - o Diet will be supplemented with essential amino acids or ketoacid analogs (up to 0.6 g/kg body weight/d)
      - o Close supervision is possible
    - Do not prescribe low- or very low–protein diets in metabolically unstable people with CKD.
- Salt intake:
  - o Sodium intake <2 g of sodium per day (or <90 mmol of sodium per day, or <5 g of sodium chloride per day)
- Avoid tobacco products

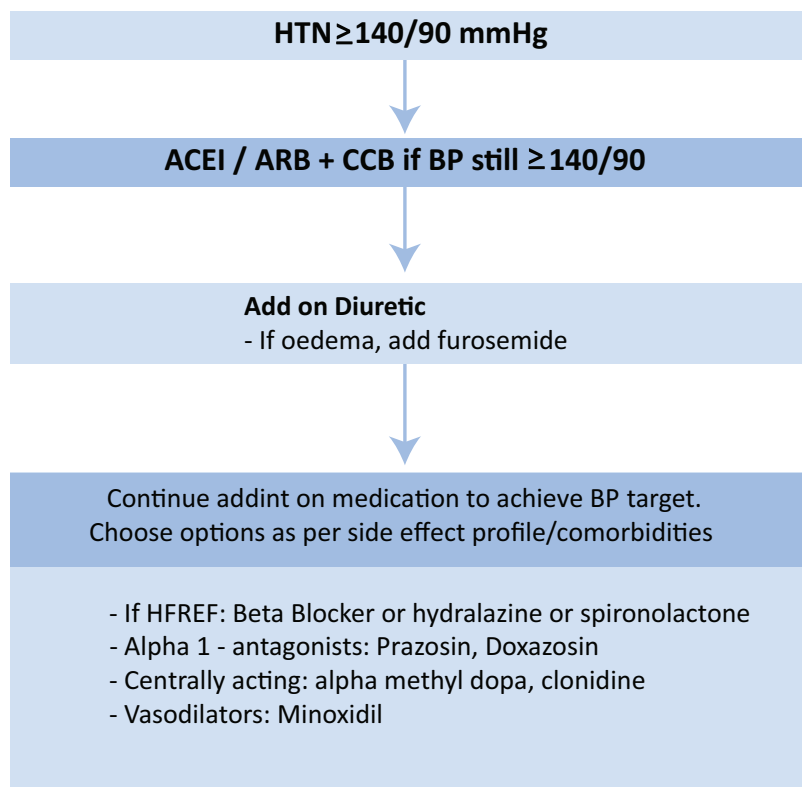
### 5.10.2 Blood pressure control

- CKD patients tend to develop resistant hypertension as GFR falls. Resistant hypertension is defined as BP>140/90mmHg despite the use of more than three medications, including a diuretic, at their maximal dose.
- **Use ACEi or ARB as first-line antihypertensives in these patient populations for renal protection:**
  - o Adjust to maximal dose while monitoring for risk of hyperkalemia.
  - o Halve the dose when K<sup>+</sup> >5.1mmol/l and stop if K<sup>+</sup>>5.5mmol/l.
  - o ACEi / ARB may increase creatinine levels:
    - o If creatinine increase is >25% from the baseline, stop ACEi / ARB and monitor.
    - o Consider challenging with half the dose after a return to baseline creatinine.
    - o Do not combine ACEi and ARB due to increased adverse effects without added benefits
- Add other anti-hypertensives as per Kenya National Guidelines For Cardiovascular Disease Management to achieve target BP <130/80mmHg (Figure 12).
- Use fixed dose combinations as much as possible to reduce pill burden and encourage adherence to medication.
- Consider less intensive BP-lowering therapy in CKD with:
  - o Frailty
  - o High risk of falls and fractures
  - o Very limited life expectancy
  - o Symptomatic postural hypotension



ACEi: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker; CCB: calcium channel blocker; CKD: chronic kidney disease; RAAS: renin-angiotensin-aldosterone system

Figure 12: Algorithm for the Management of Hypertension in Chronic Kidney Disease



\*\*\*HFREF: Heart failure with reduced ejection fraction

Figure 13: Algorithm for the Sequential Addition of Antihypertensive Therapy in Chronic Kidney Disease

### 5.10.3 Angiotensin blockade

#### Angiotensin blockade

- Start renin-angiotensin-system inhibitors (RASi) (ACEi or ARB) for people with CKD and:
  - o Diabetes with moderately-to severely increased albuminuria (G1–G4, A2 and A3)
  - o No diabetes and moderately-to severely increased albuminuria (G1–G4, A2 and A3)
  - o Normal to mildly increased albuminuria (A1) for specific indications, e.g. hypertension or heart failure with low ejection fraction
- Administer using the highest approved dose that is tolerated
- Avoid any combination of ACEi, ARB, and direct renin inhibitor (DRI) therapy
- Within 2–4 weeks of initiation or increase in the dose of a RASi, check for changes in:
  - o Blood Pressure
  - o Serum creatinine
  - o Serum potassium
- Continue ACEi or ARB therapy unless serum creatinine rises by > 30% within 4 weeks following initiation of treatment or an increase in dose.
- Continue ACEi or ARB in people with CKD even when the eGFR falls below 30 ml/min per 1.73 m<sup>2</sup>.
- Hyperkalemia associated with use of RASi should first be managed by measures to reduce the serum potassium levels rather than decreasing the dose or stopping RASi.
- Reduce the dose or discontinue ACEi or ARB if:
  - o Symptomatic hypotension
  - o Uncontrolled hyperkalemia despite medical treatment
  - o Uraemic symptoms need to be reduced while treating kidney failure

- Be aware of common side effects of medications and watch out for them (see table 14)

Table 14: Common Side Effects of Medications Used for BP Control in CKD

| Category                            | Medication                 | Usual Starting Dose  | Maximum Dose                | Common Side Effects                                                                           |
|-------------------------------------|----------------------------|----------------------|-----------------------------|-----------------------------------------------------------------------------------------------|
| Calcium Channel Blockers (CCB)      | Amlodipine                 | 5mg OD               | 10mg OD                     | Oedema, headache, palpitations                                                                |
|                                     | Nifedipine                 | 10mg BD (LA 30mg OD) | 30mg BD (Long acting: 90mg) |                                                                                               |
|                                     | Cilnidipine                | 10mg OD              | 20mg OD                     |                                                                                               |
| Thiazides                           | Hydrochlorothiazide (HTCZ) | 12.5mg OD            | 25mg OD                     | Hyponatraemia, Hypokalemia, Hyperuricemia, Hypercalcemia, Hyperglycemia                       |
|                                     | Chlorthalidone             | 25mg OD              | 50mg OD                     |                                                                                               |
|                                     | Indapamide                 | 1.5mg OD             | 5mg OD                      |                                                                                               |
| ACE Inhibitors                      | Enalapril                  | 5mg BD               | 10mg BD                     | Cough, Angioedema, hyperkalemia, Increased creatinine                                         |
|                                     | Perindopril                | 5mg OD               | 10 mg OD                    |                                                                                               |
|                                     | Captopril                  |                      |                             |                                                                                               |
|                                     | Ramipril                   | 5mg OD               | 10mg OD                     |                                                                                               |
| Angiotensin Receptor Blockers (ARB) | Losartan                   | 50mg OD              | 100mg OD                    | Increased creatinine, hyperkalemia                                                            |
|                                     | Valsartan                  | 80mg OD              | 160mg OD                    |                                                                                               |
|                                     | Irbesartan                 | 150mg OD             | 300mg OD                    |                                                                                               |
|                                     | Candesartan                | 8mg OD               | 32mg OD                     |                                                                                               |
|                                     | Olmesartan                 |                      |                             |                                                                                               |
|                                     | Azilsartan                 |                      |                             |                                                                                               |
|                                     | Telmisartan                | 40mg OD              | 80mg OD                     |                                                                                               |
| Beta-Blockers                       | Atenolol                   | 25mg OD              | 100mg OD                    | Bradycardia, Cough                                                                            |
|                                     | Bisoprolol                 | 5mg OD               | 20mg OD                     |                                                                                               |
|                                     | Carvedilol                 | 3.25mg BD            | 25mg BD                     |                                                                                               |
|                                     | Labetalol                  | 100mg BD             | 400mg BD                    |                                                                                               |
|                                     | Metoprolol                 | 25mg OD              | 100mg OD                    |                                                                                               |
|                                     | Nebivolol                  | 5mg OD               | 20mg OD                     |                                                                                               |
| Centrally Acting                    | Methyldopa                 | 250mg BD             | 1000mg/day in divided doses | Angina, Orthostatic hypotension, Gynecomastia                                                 |
|                                     | Clonidine                  | 0.1mg BD             | 2.4mg/day in divided doses  |                                                                                               |
| Loop diuretics                      | Torsemide                  | 5mg OD               | 20mg BD                     | Dehydration, hypokalemia                                                                      |
|                                     | Furosemide                 | 10mg OD              | 80mg TDS                    |                                                                                               |
| Vasodilators                        | Hydralazine                | 25mg TDS             | 150mg/day                   | Palpitations                                                                                  |
|                                     | Minoxidil                  | 2.5mg OD             | 20mg BD                     | Hypertrichosis, Pericardial effusions, Tachycardia (must be used with a B-blocker ± diuretic) |
| Aldosterone Antagonists             | Spirolactone               | 25mg OD              | 100mg OD                    | Gynecomastia,                                                                                 |
|                                     | Eplerenone                 | 25mg OD              | 50mg BD                     | Hyperkalemia                                                                                  |
| α1 Receptor blockers                | Prazocin                   | 1mg BD / TDS         | 20mg/day                    | Diarrhea, tachycardia                                                                         |
|                                     | Doxazocin                  | 1mg OD               | 8mg/day                     |                                                                                               |

\*\*\*OD: Once daily, BD Twice daily,, TDS thrice daily

## 5.10.4 Glycaemic control

- Monitoring glycaemic control:
  - Use hemoglobin A1c (HbA1c)
  - Frequency:
    - Every 3-6 months per year
    - As often as 4 times per year if the glycaemic target is not met or after a change in antihyperglycemic therapy.
  - Accuracy and precision of HbA1c measurement declines with advanced CKD (G4–G5), particularly among patients treated by dialysis, in whom HbA1c measurements have low reliability
- Glycaemic target:
  - Individualise the HbA1c target to range from <6.5% to <8.0%
  - Factors guiding decisions on individualised HbA1C targets are in Figure 14 and Table 24

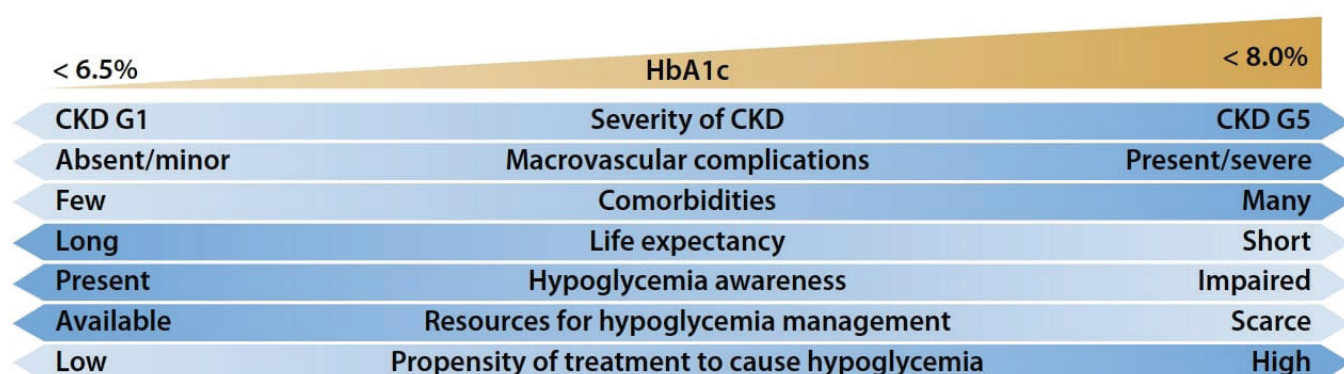


Figure 14: Determinants of HbA1C Target for CKD Patients with Diabetes

Table 15: Individualised HbA1C Targets According to Patient Profile

| A1c Target | Patient type                                                                                                          |
|------------|-----------------------------------------------------------------------------------------------------------------------|
| 4-6.5%     | Long life expectancy, No episodes of hypoglycemia                                                                     |
| < 7%       | Optimal for most adults                                                                                               |
| <8%        | CKD stage 4 and lower, elderly, high risk of hypoglycemia, reduced life expectancy, Significant medical comorbidities |

For more information, refer to the chapter on CKD in diabetes.

### 5.10.5 Mineralocorticoid receptor antagonists

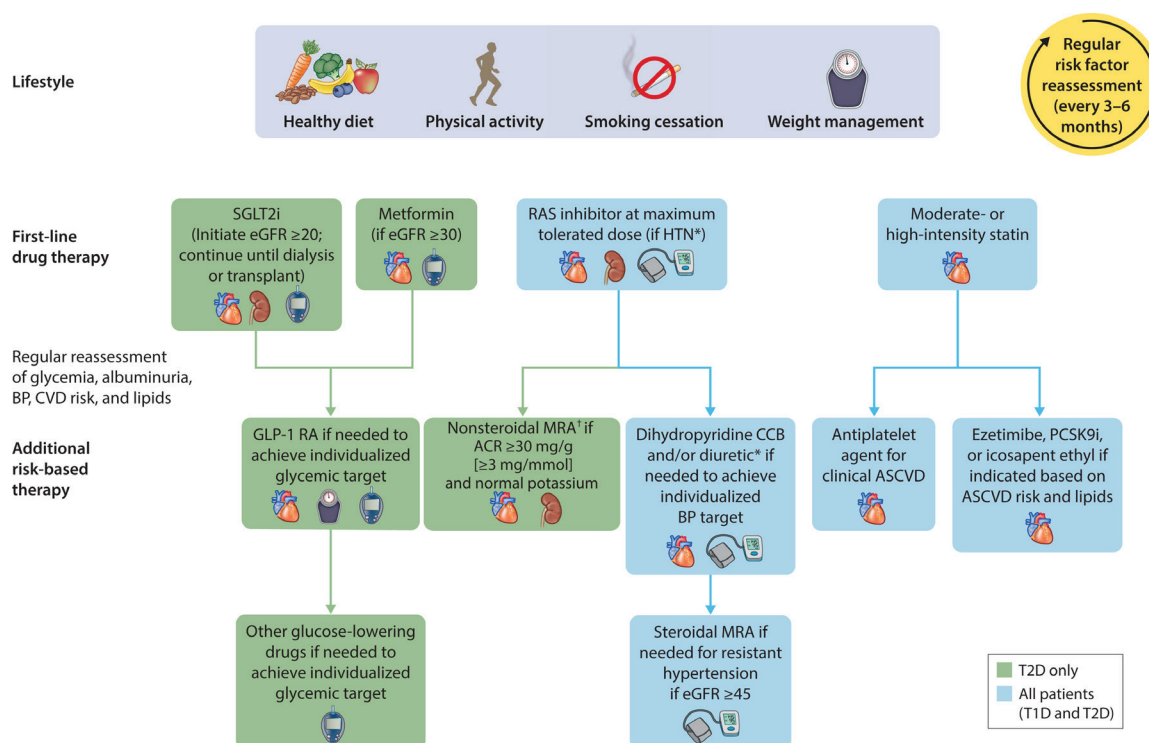
- Use a nonsteroidal mineralocorticoid receptor antagonist with proven kidney or cardiovascular benefit for adults with:
  - o T2DM
  - o eGFR >25 ml/min per 1.73 m<sup>2</sup>
  - o normal serum potassium concentration, and
  - o albuminuria (>30 mg/g [>3 mg/mmol]) despite maximum tolerated dose of RAS inhibitor (RASi)
- Example: Finerenone 10-20mg once a day

### 5.10.6 Statins

- CKD patients are at high risk of cardiovascular events
- Driven by both traditional and non-traditional risk factors
- Statins are recommended for cardiovascular event prevention for:
  - o Adults aged ≥50 years with CKD and eGFR ≥60 ml/min per 1.73 m<sup>2</sup> (GFR categories G1–G2)
  - o Adults aged ≥50 years with eGFR <60 ml/min per 1.73 m<sup>2</sup> but not treated with chronic dialysis or kidney transplantation (GFR categories G3a–G5)
  - o In adults aged 18–49 years with CKD but not treated with chronic dialysis or kidney transplantation, if:
    - Known coronary artery disease
    - Diabetes mellitus,
    - Prior ischemic stroke
    - Estimated 10-year incidence of coronary death or nonfatal myocardial infarction >10%.

## 5.10.7 Holistic CKD management

The figure below presents a comprehensive, risk-based approach to managing CKD in patients with diabetes. It integrates lifestyle measures, first-line therapies (SGLT2 inhibitors, metformin, RAS blockade, and statins), and additional treatments guided by glycaemia, albuminuria, blood pressure, and cardiovascular risk, with reassessment every 3–6 months.



ACR: albumin: creatinine ratio; ASCVD: atherosclerotic cardiovascular disease; BP: blood pressure; CCB: calcium channel blocker; CVD: cardiovascular disease; eGFR: estimated glomerular filtration rate; GLP-1 RA: glucagon-like peptide 1 receptor agonist; HTN: hypertension; MRA: mineralocorticoid receptor antagonist; PCSK9i: proprotein convertase subtilisin/kexin type 9 inhibitor; RAS: renin-angiotensin system; SGLT2i: sodium-glucose cotransporter type 2 inhibitor; T1D: type 1 diabetes; T2D: type 2 diabetes

## 5.10.8 Management of symptoms in CKD

The 25 outlines a structured approach to the management of common symptoms in patients with chronic kidney disease (CKD). It links each symptom to its likely underlying causes and provides both non-pharmacological measures and stepwise pharmacological options, promoting comprehensive, patient-centred supportive care alongside disease-directed treatment.

Table 16: Management of Symptoms in CKD

| Symptoms                                           | Likely causes                                                                                                                                                                                                                                                 | Management                                                                                                                                                                                                                                                                             |                                                                                                                                                                                                                                                                                                                                                                                                                                     |
|----------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|                                                    |                                                                                                                                                                                                                                                               | Non-pharmacological                                                                                                                                                                                                                                                                    | Pharmacological<br>(stepwise approach)                                                                                                                                                                                                                                                                                                                                                                                              |
| <b>Nausea, vomiting, bitter taste and anorexia</b> | <ul style="list-style-type: none"> <li>Metabolic acidosis</li> <li>Drugs (e.g. opiates)</li> <li>Constipation</li> <li>Delayed gastric emptying</li> <li>Uraemic toxins</li> </ul>                                                                            | <ul style="list-style-type: none"> <li>Exclude offending drugs</li> <li>Small, frequent meals</li> <li>Avoid alcohol, greasy, spicy, sweet and bitter-tasting food and drinks</li> <li>Postprandial upright position</li> </ul>                                                        | <ul style="list-style-type: none"> <li>Metoclopramide (low dose) 5–10 mg TID PO half-an-hour before meals. Start with 5 mg TID and titrate to maximal dose of 10 mg TID</li> <li>Ondansetron 4–8 mg TID (sublingually, orally or IV)</li> </ul>                                                                                                                                                                                     |
| <b>Constipation</b>                                | <ul style="list-style-type: none"> <li>Phosphate binders (calcium carbonate)</li> <li>Oral iron</li> <li>Ondansetron</li> <li>Fluid restriction</li> </ul>                                                                                                    | <ul style="list-style-type: none"> <li>Mild exercise</li> <li>Bulking agents like fibre are not recommended for patients using constipating medication or with decreased fluid/food intake or decreased activity</li> </ul>                                                            | <ul style="list-style-type: none"> <li>Lactulose syrup 15mL PO OD to TID</li> <li>Bisacodyl 5–20 mg PO OD nocte</li> </ul>                                                                                                                                                                                                                                                                                                          |
| <b>Uraemic pruritis</b>                            | <ul style="list-style-type: none"> <li>Exacerbating factors:</li> <li>Non-dermatological</li> <li>Anaemia</li> <li>Iron deficiency</li> <li>Dermatological</li> <li>Xerosis</li> <li>Drug related</li> <li>Contact dermatitis</li> <li>Infestation</li> </ul> | <ul style="list-style-type: none"> <li>Appropriate skin care</li> <li>Bath in lukewarm water and use fragrance-free soap</li> <li>Moisturize with aqueous emollients</li> <li>Patients on dialysis: improve dialysis adequacy</li> <li>Manage calcium and phosphate balance</li> </ul> | <ul style="list-style-type: none"> <li>Gabapentin 100–200 mg nocte (initiate 100 mg nocte alternate days and titrate up slowly. If the patient is on dialysis this should be given after dialysis)</li> <li>Pregabalin: initiate at 25 mg OD nocte, titrate up slowly to 75mg OD nocte.</li> <li>Antihistamines (limited role, but can help with nocturnal pruritus): Chlorpheniramine 10mg nocte, Cetirizine 10mg nocte</li> </ul> |
| <b>Neuropathic Pain</b>                            |                                                                                                                                                                                                                                                               |                                                                                                                                                                                                                                                                                        | <ul style="list-style-type: none"> <li>Pregabalin 25–75 mg (initiate at 25 mg OD nocte and titrate up slowly)</li> <li>Amitriptyline 10 - 25mg OD nocte</li> <li>Gabapentin 100–200 mg nocte (initiate 100 mg nocte alternate days and titrate up slowly. If the patient is on dialysis, this should be given after dialysis)</li> </ul>                                                                                            |

|                           |                                                                                                                                                                                                                                                                                                       |                                                                                                                                                                                                                                                                                                                                                |                                                                                                                                                               |
|---------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Sleep disturbances</b> | <ul style="list-style-type: none"> <li>• Anaemia</li> <li>• Vitamin D deficiency</li> <li>• Metabolic acidosis</li> <li>• Hypo/hyperthyroidism</li> <li>• Adrenal insufficiency</li> <li>• Anxiety</li> <li>• Depression</li> <li>• Medications: benzodiazepines, antidepressants, opioids</li> </ul> | <ul style="list-style-type: none"> <li>• Psychosocial and spiritual support</li> <li>• Cognitive and psychological therapies, e.g. relaxation therapy, meditation</li> <li>• Optimize sleep hygiene (no stimulants, reserve bedroom for sleep)</li> <li>• Manage restless leg syndrome, pruritus, pain and obstructive sleep apnoea</li> </ul> | <ul style="list-style-type: none"> <li>• Amitriptyline 10 - 25mg OD nocte</li> <li>• Zolpidem 5-10mg OD nocte</li> <li>• Melatonin 1-10mg OD nocte</li> </ul> |
|---------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------|

## 5.11 Referral to a Nephrologist/Consultant Physician

Late referral to a nephrologist is associated with poorer outcomes for patients with CKD. Early referral: -

- Allows identification of multi-factorial causes of CKD
- Enables staging of the severity of illness
- Enables creation of a treatment plan in conjunction with the primary physician, who can then institute the directives.
- Enables planning early for vascular access
- Commences management of complications for pre-dialysis patients (GFR <20ml/ min/1.73m<sup>2</sup>), which helps avoid unplanned or emergency starts to dialysis associated with worse outcomes

*Table 17: Indications for Mandatory Referral to a Nephrologist*

- eGFR < 30mL/min/1.73m<sup>2</sup> sustained for ≥3 months with no values rising above the threshold
  - Accelerated eGFR decline: eGFR decline by ≥25% or ≥15 mL/min/1.73 m<sup>2</sup> within 12 months, sustained for ≥3 months with no values rising above the threshold
- Substantial proteinuria:
  - UACR ≥70 mg/mmol
  - UACR >30mg/mmol + hematuria
- Haematuria
- Isolated sustained electrolyte abnormalities:
  - Hyperkalaemia >5.5mmol/l
  - Hyponatraemia <135mmol/l
- Resistant hypertension
- Recurring kidney stones
- Hereditary kidney diseases

Table 18: Levels of Health Care in Relation to CKD Management

| Levels of Care | Health Care Providers                                                                                                 | Services to be Offered                                                                                                                                                                                                                                                          |
|----------------|-----------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Level 1</b> | Community health nurses,<br>Community health promoters                                                                | <ul style="list-style-type: none"> <li>Continuous patient education on medication and clinic adherence and individualised diet</li> <li>Refer to a higher level of care as per patient's clinical presentation</li> </ul>                                                       |
| <b>Level 2</b> | Community health nurses,<br>Clinical officers                                                                         | <ul style="list-style-type: none"> <li>Continuous patient education on medication adherence and individualised diet</li> <li>Refer to a higher level of care as per patient's clinical presentation</li> </ul>                                                                  |
| <b>Level 3</b> | Physicians, Clinical officers,<br>Nurses, Nutritionist                                                                | <ul style="list-style-type: none"> <li>Routine investigations such as UECs, Hemoglobin and albumin</li> <li>Continuous patient education on medication adherence and individualised diet</li> </ul>                                                                             |
| <b>Level 4</b> | Physicians, Registered nurses,<br>Clinical officers, Nutritionist                                                     | <ul style="list-style-type: none"> <li>Continuous patient education on medication adherence and individualised diet</li> <li>Referral to a nephrologist when recommended and mandatory</li> </ul>                                                                               |
| <b>Level 5</b> | Physicians, Registered nurses,<br>Nephrology nurse(s), Nephrologist,<br>Clinical nutritionists, Clinical psychologist | <ul style="list-style-type: none"> <li>Continuous patient education on medication adherence and individualised diet</li> <li>Routine investigations such as UECs, Hemoglobin, albumin and iron profile if available</li> <li>Psychological counselling</li> </ul>               |
| <b>Level 6</b> | Nephrologist, Physicians,<br>Nephrology nurses, Clinical nutritionists,<br>Clinical psychologist                      | <ul style="list-style-type: none"> <li>Nephrologist review as per scheduled visits</li> <li>Continuous patient education on medication adherence and individualised diet</li> <li>Investigations such as creatinine, microalbumin</li> <li>Psychological counselling</li> </ul> |

# Chapter 6: Kidney Disease in Children

## 6.1 Introduction

- Kidney diseases in children represent a significant yet often under-recognized contributor to childhood morbidity and long-term non-communicable disease burden.
- Renal disorders in childhood range from acute, potentially reversible conditions such as Acute Kidney Injury (AKI) to chronic and progressive disorders including Nephrotic Syndrome and Chronic Kidney Disease (CKD).
- Early identification and timely intervention are critical to prevent irreversible kidney damage, growth impairment, cardiovascular complications, and premature mortality.
- Children presenting with any of these warning signs require prompt evaluation and appropriate referral to prevent progression and long-term renal complications.

## 6.2 Acute Kidney Injury in Children

### 6.2.1 Overview of AKI in children

Acute Kidney Injury is defined functionally as a rapid decline in glomerular filtration rate (GFR) that leads to accumulation of waste products such as blood urea nitrogen (BUN) and creatinine. It also leads to the inability to maintain fluid, acid and electrolyte balance.

AKI is the most common reason for consulting a nephrologist for kidney disease in children. The commonest causes of AKI include viral infections, malaria, streptococcal infections, crush injuries and burns.

### 6.2.2 Approach to diagnosis of AKI in children

1. Always have a high index of suspicion
2. Perform a good medical history and physical examination
3. Evaluate hydration status of the child
4. Check the blood pressure
5. Perform laboratory evaluation:

| Level 3 health facility laboratory                                                                                                         | Level 4 health facility and above laboratory                                                                                                                                                                                                                              |
|--------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Full blood count</li><li>• UECs</li><li>• Urinalysis</li><li>• BS for MPS (if indicated)</li></ul> | <ul style="list-style-type: none"><li>• Level 3 lab evaluations</li><li>• Extended electrolytes</li><li>• Viral serology: HIV, HBsAg, HCV</li><li>• Autoimmune serology: ANA, ANCA, antiGBM, ASOT</li><li>• Complement C3 and C4 level</li><li>• Ultrasound KUB</li></ul> |

6. Refer to Level 4 or above for further management by a specialist

## 6.2.3 Management of AKI in children

### A. Supportive Therapy:

#### 1. Fluid management

i. Daily clinical assessment and monitoring is essential:

- Vital signs including blood pressure
- Weight
- Hydration status
- Neurological assessment
- RBS

#### ii. Replacing fluids

A safe approach is:

Fluid replacement = Maintenance fluid + Insensible loss + Urine output + ongoing fluid losses

- Insensible loss = 300-400 ml/m<sup>2</sup>/day
- Maintenance fluid = 20-30mL/kg in neonates and infants  
=10-15mL/kg in children above one year

#### ii. Bolus fluid if in shock

Type of fluid:

- Intravenous fluid: ¼ strength saline:
  - o For 500mL volume, mix 100ml of 5% Dextrose into 400ml of Normal Saline
- If not available, hydrate via NGT with ORS

### B. Management at Level 4 or Higher Hospital

#### 1. Management of hypernatraemia

Hypernatraemic dehydration is associated high morbidity and mortality

Management is based on assumption that children with hypernatremia will be dehydrated increasingly with increasing level of serum sodium

- o Serum Na 150mmol/L - Fluid loss 5% body weight
- o Serum Na 160mmol/L - Fluid loss 10% body weight
- o Serum Na 170mmol/L - Fluid loss 15% body weight
- o Serum Na 180mmol/L - Fluid loss 20% body weight
- Replace fluid at 60mL/kg + maintenance fluid every 24 hours
- Use 5% Dextrose or ¼ strength saline
- Replace as long as required to resolve serum sodium excess
- Correct sodium at a rate of 0.5mmol/h
  - o E.g. if sodium is:
    - 160mmol/L - correct over 40h
    - 170mmol/L - correct over 60h
    - 180mmol/L – correct over 80h

## **2. Management of hyperkalemia (serum K >6.5 mmol/L)**

- A medical emergency that can lead to cardiac arrhythmia and death
- Management:
  - o Avoid any potassium-containing medications and IV fluids
  - o Maintain patient on low-potassium or potassium-free diet
  - o Stabilize myocardium against arrhythmias:
    - Calcium gluconate 0.5mL/kg slow IVI over 15 minutes
  - o Shift potassium from plasma into cells:
    - Soluble insulin 0.1 units/kg + 10% dextrose (10g/100mL =5g) over 1 hour
      - E.g. for a 10kg child give 1 unit of insulin + 50ml of 10% dextrose over 1 hour
    - Sodium bicarbonate 1ml/kg intravenously over 1 hour
    - Salbutamol nebulization: based on child's weight:
      - <10kg: 1.25mg
      - 10-25kg: 2.5mg
      - >25kg: 5mg

## **3. Avoid nephrotoxic drugs: e.g. gentamicin, amikacin, macrolides, NSAIDs, cotrimoxazole**

## **4. Fluid and blood pressure management**

- Aim for normal BP
- Avoid rapid BP fluctuations (keep BP <90th percentile)
- Fluid overload may contribute to hypertension. Manage by:
  - o salt and water restriction
  - o diuretics in higher doses

## **5. Prevent and treat any infection adequately**

### **i. Medications:**

- Adjust drug dosage to renal function as determined by eGFR
- Monitor drug levels where possible

### **ii. Nutrition:**

- Obtain nutritionist review
- Maintain adequate nutrition
- Enteral feeding is preferred
- Parenteral nutrition if enteral feeding is not possible

## **6. Refer for dialysis if indication arises**

## 6.3 Nephrotic Syndrome in Children

### 6.3.1 Introduction

Nephrotic syndrome results from wastage of proteins in urine characterised by

1. oedema
2. frothy urine,
3. proteinuria of 3+

### 6.3.2 Clinical manifestations of nephrotic syndrome in children

- Oedema: periorbital (first to appear), ankle and leg, scrotal and penile, labial
- Third spacing: ascites, pleural effusion
- Reduced circulating volume: tachycardia, oliguria, reduced GFR
- Hypercoagulable state: DVT, pulmonary embolism, renal vein thrombosis, CVA
- Infections: pneumonia, UTI, sepsis and peritonitis
- Others: haematuria, AKI, anorexia, lethargy, irritability

### 6.3.3 Evaluation of nephrotic syndrome in children

| At Level 3 hospital:                                                                                                                                                                                                                                                                                                          | At Level 4 or higher hospital:                                                                                                                                                                                                                                                                                                                                               |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Urinalysis:<ul style="list-style-type: none"><li>• Use first morning void</li><li>• Urine appears frothy</li><li>• Nephrotic range proteinuria: 3+ or 4+</li><li>• May have microscopic hematuria</li></ul></li><li>• UECs</li><li>• Total Blood Count - rule out infection</li></ul> | <p>Level 3 facility laboratory tests plus:</p> <ul style="list-style-type: none"><li>• Serum albumin and total protein level</li><li>• Lipid profile</li><li>• Complement C3 and C4 levels</li><li>• ANA</li><li>• Triple serology: HIV, HBV and HCV</li></ul> <p><b>Imaging:</b></p> <ul style="list-style-type: none"><li>• Ultrasound KUB</li><li>• Chest X-ray</li></ul> |

## 6.3.4 Supportive management of nephrotic syndrome in children

| Level 3 Health Facility                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>Blood pressure measurements must be done.</li> <li>Fluid restriction is not recommended. Should consume normal amounts of fluids.</li> <li>Start prednisolone at 2 mg/kg or maximum of 60 mg.</li> <li>Refer for specialist management by a paediatrician or paediatric nephrologist</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
| Level 4 or higher Hospital                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
| <ol style="list-style-type: none"> <li>All level 3 measures PLUS</li> <li>Albumin <ul style="list-style-type: none"> <li>For patients with refractory oedema +/- symptomatic hypovolemia)</li> <li>Dose: 20% albumin infusion given at 1 g/kg over 4–8 hrs</li> <li>with diuretic given mid-infusion</li> <li>Avoid if BP is not low (use diuretics only)</li> </ul> </li> <li>Diet <ul style="list-style-type: none"> <li>Balanced diet with adequate protein</li> <li>Salt restriction not necessary but in persistent oedema - no added salt</li> </ul> </li> <li>Diuretics <ul style="list-style-type: none"> <li>Give furosemide</li> <li>Monitor tissue perfusion</li> <li>on refractory oedema with no intravascular depletion, add thiazide diuretic or a potassium sparing diuretic</li> </ul> </li> <li>Infections: treat common infections including peritonitis and chest infections</li> <li>Hypertension <ul style="list-style-type: none"> <li>Amlodipine 5mg once a day</li> <li>Hydralazine 12.5 to 25mg BID to TID</li> <li>Refer to a specialist</li> </ul> </li> <li>Vaccination <ul style="list-style-type: none"> <li>Withhold all live vaccines until 3 months off steroids or defer administration until the steroid dose is below 1mg/kg/d</li> <li>Avoid live vaccines during relapse</li> <li>Killed/Inactivated vaccines are safe</li> <li>For all children with nephrotic syndrome: <ul style="list-style-type: none"> <li>Pneumococcal vaccines</li> <li>Annual influenza vaccine</li> </ul> </li> </ul> </li> </ol> | <ol style="list-style-type: none"> <li>DVT prophylaxis <ul style="list-style-type: none"> <li>Encourage mobilization</li> <li>Avoid prolonged bed rest</li> <li>Use compression stockings</li> <li>Avoid hypovolemia</li> </ul> </li> <li>Hyperlipidaemia <ul style="list-style-type: none"> <li>Decrease dietary fat &lt;30% of total calories</li> <li>There is no role for statins in hyperlipidaemia</li> </ul> </li> <li>Close monitoring: daily charting for <ul style="list-style-type: none"> <li>Urine dipstick</li> <li>Blood pressure</li> <li>Daily weight</li> <li>Input and output chart</li> </ul> </li> <li>Definitive management</li> <li>Refer to a paediatric nephrologist</li> <li>When to refer to a paediatric nephrologist <ul style="list-style-type: none"> <li>Extremes of age &lt;1yr and &gt;10yr</li> <li>Gross hematuria</li> <li>Systemic disease</li> <li>Renal failure</li> <li>Frequent relapses and steroid dependence</li> <li>Worsening renal function</li> <li>Hypertension</li> </ul> </li> </ol> |

## 6.4 Chronic Kidney Disease in Children

### 6.4.1 Definition of CKD

Chronic kidney disease is characterised by an irreversible deterioration of renal function that gradually progresses to end-stage renal disease and eventually requires renal replacement therapy.

### 6.4.2 Prevalence of CKD in children

- Children, adolescents, and young adults constitute less than 5% of the end-stage renal disease population
- CKD in children is reported at 15 to 74.7 per million children
- Number of children with paediatric CKD is underestimated especially in resource-limited areas

### 6.4.3 Aetiology of CKD in children

#### CKD in children results from a heterogeneous group of disorders:

- Primary glomerular disease – 22%
- Congenital anomalies of the kidney and urinary tract (CAKUT) – 22%
- Cystic/hereditary/congenital diseases – 12%
- Secondary glomerular disease/vasculitis – 11%
- Interstitial nephritis/pyelonephritis – 3%
- Transplant complications – 1%
- Diabetes – 1%
- Neoplasms/tumors – 0.5%
- Miscellaneous conditions – 14%
- Uncertain aetiology – 5%

#### Congenital disease:

- Accounts for 60% of CKD cases
- Glomerular disorders:
- 2nd largest cause of childhood CKD
- More common in children >12 years of age
- Includes congenital anomalies of the kidney and urinary tract (CAKUT)
- As part of CAKUT, the most common obstructive lesions are posterior urethral valves and prune belly syndrome. Both of these conditions only occur in boys; hence, boys have higher prevalence of CKD than girls

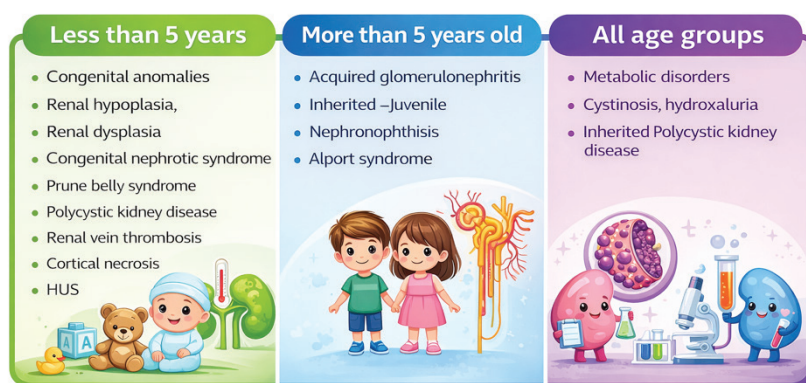


Figure 16: Aetiology of CKD in Children

### 6.4.4 Diagnosis of CKD in children

- In children, the diagnosis of CKD is often missed or delayed
- Note that:
  - o Normal levels of GFR vary with age, gender, and body size.
  - o GFR increases with maturation from infancy and approaches the adult mean value by 2 years of age
  - o GFR of children less than 2 years of age is lower than GFR of older people
  - o The KDIGO classification of CKD is not applicable to children less than 2 years of age
- CKD is present if either of the following criteria are present:
  - o GFR <60 mL/min per 1.73 m<sup>2</sup> for >3 months with implications for health regardless of whether other CKD markers are present.
  - o GFR >60 mL/min per 1.73 m<sup>2</sup> accompanied by:
    - Evidence of structural damage or other markers of kidney function abnormalities, including proteinuria, albuminuria, renal tubular disorders, or pathologic abnormalities detected by histology or inferred by imaging
    - This category also includes patients with functioning kidney transplants

### 6.4.5 Estimating GFR in children

- An estimated GFR is used as a measure of kidney function
- In children, eGFR is calculated using height, serum creatinine, and a constant “k”
- For children 1-17 years, use the Beside Schwarz formula:  

$$eGFR = \text{“k”} \times \text{height (in cm)} / \text{serum creatinine (in umols/l)}$$
 where k=40

## 6.4.6 Progression of CKD in children

- Children with CKD experience progressive deterioration of kidney function leading to ESRD
- The rate of CKD progression is variable and often unpredictable
- Control of BP and proteinuria control has a beneficial effect on slowing CKD progression
- Risk factors associated with an increased risk of progression of CKD include:

|                            |                                                                                                                                                                |
|----------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Neonatal Factors           | Prematurity & fetal growth restriction                                                                                                                         |
|                            | Nephron development completed at 35 weeks gestation                                                                                                            |
| Puberty Factors            | Rate of CKD progression is usually greatest during periods of rapid growth                                                                                     |
|                            | Sudden increase in body mass results in rise in filtration demands of remaining nephrons                                                                       |
|                            | Adolescence may get rapid decline in kidney function                                                                                                           |
| Underlying primary disease | More rapid CKD progression is observed in children with glomerular disease versus those with non-glomerular disease uncontrolled hypertension and proteinuria. |

## 6.4.7 Staging and risk stratification of CKD

CKD staging guides management by stratifying risk for progression & complications of CKD

Risk stratification guides the intensity of monitoring & management for CKD progression

As in adults, in children CKD staging is based on aetiology GFR and degree of albuminuria

| Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012            |     |                                  |       | Persistent albuminuria categories                    |                                                     |                                                |
|---------------------------------------------------------------------------|-----|----------------------------------|-------|------------------------------------------------------|-----------------------------------------------------|------------------------------------------------|
|                                                                           |     |                                  |       | Description and range                                |                                                     |                                                |
|                                                                           |     |                                  |       | A1                                                   | A2                                                  | A3                                             |
|                                                                           |     |                                  |       | Normal to mildly increased<br><30 mg/g<br><3 mg/mmol | Moderately increased<br>30–300 mg/g<br>3–30 mg/mmol | Severely increased<br>>300 mg/g<br>>30 mg/mmol |
| GFR categories (mL/min per 1.73 m <sup>2</sup> )<br>Description and range | G1  | Normal or high                   | ≥90   |                                                      |                                                     |                                                |
|                                                                           | G2  | Mildly decreased                 | 60–89 |                                                      |                                                     |                                                |
|                                                                           | G3a | Mildly to moderately decreased   | 45–59 |                                                      |                                                     |                                                |
|                                                                           | G3b | Moderately to severely decreased | 30–44 |                                                      |                                                     |                                                |
|                                                                           | G4  | Severely decreased               | 15–29 |                                                      |                                                     |                                                |
|                                                                           | G5  | Kidney failure                   | <15   |                                                      |                                                     |                                                |
|                                                                           | G5  |                                  |       |                                                      |                                                     |                                                |

Green, low risk (if no other markers of kidney disease, no CKD); yellow, moderately increased risk; orange, high risk; red, very high risk

Green, low risk (if no other markers of kidney disease, no CKD); yellow, moderately increased risk; orange, high risk; red, very high risk

Source: KDIGO. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013;3(1):1–150.

Figure 17: CKD Staging by GFR and Albuminuria Categories

## Stages of chronic kidney disease for children based on the KDIGO 2012 Clinical Practice Guideline

| GFR Category | GFR (mL/min/1.73 m <sup>2</sup> ) | Terminology                      |
|--------------|-----------------------------------|----------------------------------|
| G1           | ≥ 90                              | Normal or high                   |
| G2           | 60–89                             | Mildly decreased*                |
| G3a          | 45–59                             | Mildly to moderately decreased   |
| G3b          | 30–44                             | Moderately to severely decreased |
| G4           | 15–29                             | Severely decreased               |
| G5           | < 15                              | Kidney failure                   |

Note: In the absence of evidence of kidney damage, neither GFR category G1 nor G2 fulfill the criteria for CKD.

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### 6.4.8 Pathophysiology of CKD in children

- Chronic kidney disease (CKD) in children results from progressive nephron loss leading to multisystem physiological disturbances that affect growth, metabolism, endocrine regulation, bone health, and pubertal development.
- Unlike adults, congenital and developmental causes such as **intrauterine growth restriction (IUGR)**, **genetic disorders**, and **prematurity** are major contributors. The pathophysiology is complex and involves interlinked nutritional, hormonal, inflammatory, and metabolic pathways.

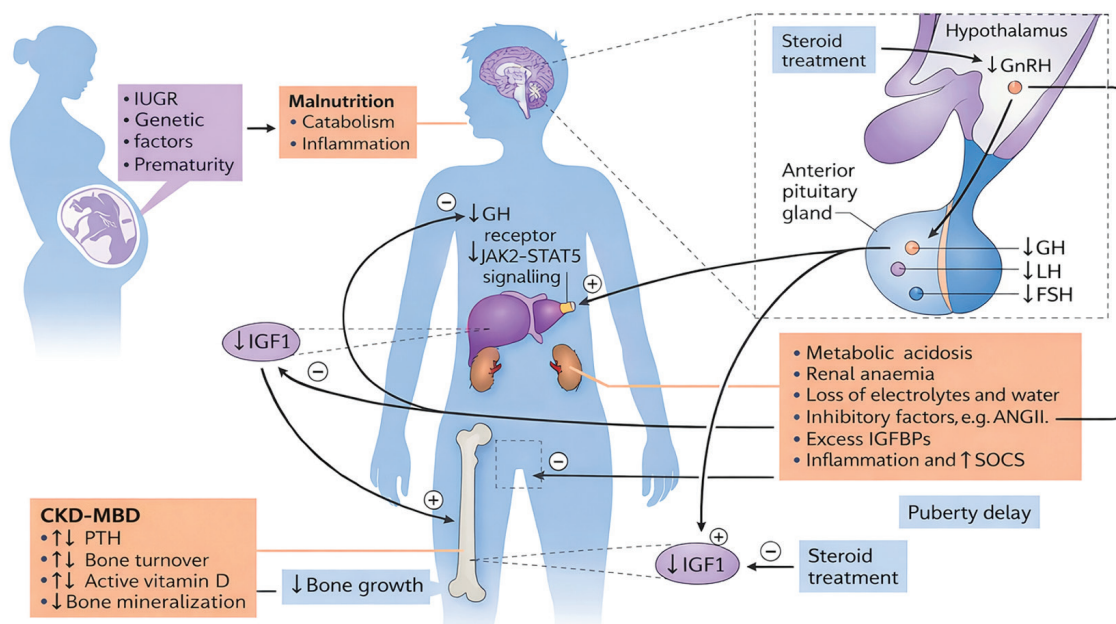


Figure 18: Pathophysiology of CKD in Children

| Pathophysiological Domain                         | Mechanisms and Clinical Consequences                                                                                                                                                                                                                                                                                                                                                         |
|---------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. Early-Life Risk Factors                        | <ul style="list-style-type: none"> <li>• Intrauterine growth restriction (IUGR), genetic disorders, and prematurity reduce nephron number.</li> <li>• Lower nephron endowment increases lifelong susceptibility to CKD.</li> <li>• Predisposes to impaired growth and progressive renal dysfunction.</li> </ul>                                                                              |
| 2. Malnutrition and Inflammation                  | <ul style="list-style-type: none"> <li>• CKD causes malnutrition, catabolism, and chronic inflammation.</li> <li>• Uremic toxins and inflammatory cytokines suppress appetite.</li> <li>• Increased protein breakdown → muscle wasting.</li> <li>• Results in poor linear growth and reduced growth velocity.</li> </ul>                                                                     |
| 3. Growth Hormone (GH)–IGF-1 Axis Dysfunction     | <ul style="list-style-type: none"> <li>• GH resistance despite normal/elevated circulating GH.</li> <li>• ↓ GH receptor expression.</li> <li>• Impaired JAK2–STAT5 signaling.</li> <li>• ↑ IGF-binding proteins (IGFBPs).</li> <li>• Inflammation and metabolic acidosis reduce IGF-1 bioavailability.</li> <li>• Leads to reduced bone growth and short stature.</li> </ul>                 |
| 4. CKD–Mineral and Bone Disorder (CKD-MBD)        | <ul style="list-style-type: none"> <li>• ↑ Parathyroid hormone (PTH).</li> <li>• ↑ Bone turnover.</li> <li>• ↓ Active vitamin D production.</li> <li>• ↓ Bone mineralisation.</li> <li>• Causes skeletal deformities and worsens growth failure.</li> </ul>                                                                                                                                  |
| 5. Metabolic and Hematologic Complications        | <ul style="list-style-type: none"> <li>• Metabolic acidosis impairs growth and bone buffering.</li> <li>• Renal anemia (↓ erythropoietin) reduces oxygen delivery.</li> <li>• Electrolyte and water imbalance.</li> <li>• Inhibitory factors (e.g., angiotensin II).</li> <li>• Inflammation and ↑ SOCS proteins.</li> <li>• Amplifies GH resistance and reduces growth velocity.</li> </ul> |
| 6. Hypothalamic–Pituitary–Gonadal Axis Disruption | <ul style="list-style-type: none"> <li>• ↓ GnRH (hypothalamus).</li> <li>• ↓ LH and FSH (anterior pituitary).</li> <li>• ↓ GH secretion.</li> <li>• Leads to delayed puberty.</li> <li>• Steroid therapy further suppresses growth and pubertal progression.</li> </ul>                                                                                                                      |

## 6.4.9 Clinical features and presentation of CKD in children

| Category                         | Key Clinical Features / Presentation                                                                                                                                                                                                                                                                                                                                                                              |
|----------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Early Stages CKD (G1–G2)         | <ul style="list-style-type: none"> <li>Usually asymptomatic</li> <li>Abnormal urinalysis (proteinuria/hematuria)</li> <li>Elevated blood pressure</li> <li>Polydipsia and nocturia (impaired urine concentrating ability)</li> <li>Incidental elevated serum creatinine</li> <li>Congenital or structural anomalies detected on imaging</li> </ul>                                                                |
| Later Stages CKD (G3–G5)         | <ul style="list-style-type: none"> <li>Poor growth</li> <li>Symptoms/signs of severe renal impairment</li> <li>Systemic manifestations from underlying disease</li> </ul>                                                                                                                                                                                                                                         |
| Advanced CKD                     | <ul style="list-style-type: none"> <li>Anemia</li> <li>Anorexia, nausea, vomiting</li> <li>Failure to thrive / short stature</li> <li>Renal osteodystrophy (bone disease)</li> <li>Cardiovascular disease</li> </ul>                                                                                                                                                                                              |
| Disease-Specific Presentations   | <ul style="list-style-type: none"> <li>CAKUT: Polyuria, polydipsia, recurrent UTIs</li> <li>Glomerular disease: Hematuria, proteinuria, oedema, hypertension</li> </ul>                                                                                                                                                                                                                                           |
| Common Patterns of Presentation  | <ul style="list-style-type: none"> <li>New-born baby born with kidney failure or with kidney enlargement detected on a prenatal ultrasound</li> <li>A normal four-year-old suddenly develops fluid retention with blood in the urine</li> <li>A six-year-old presents with muscle cramps and failure to thrive</li> <li>A 16-year-old girl complains of short stature and has never experienced menses</li> </ul> |
| General Symptoms                 | <ul style="list-style-type: none"> <li>Poor appetite</li> <li>Vomiting</li> <li>Stunted growth</li> <li>Malaise</li> <li>Headache</li> <li>Bone pain</li> <li>Pale skin</li> <li>Uremic breath</li> <li>Detectable abdominal mass</li> <li>Tissue swelling (oedema)</li> </ul>                                                                                                                                    |
| Urinary Symptoms                 | <ul style="list-style-type: none"> <li>Polyuria or oliguria/anuria</li> <li>Recurrent urinary tract infections</li> <li>Urinary incontinence</li> </ul>                                                                                                                                                                                                                                                           |
| Neurological / Systemic Symptoms | <ul style="list-style-type: none"> <li>Hearing deficit</li> <li>Altered mental alertness</li> <li>Irritability</li> <li>Poor muscle tone</li> </ul>                                                                                                                                                                                                                                                               |

## 6.4.10 Clinical approach to the evaluation and management of chronic kidney disease in children

### A. Take a Complete Medical History (from Antenatal, Birth to Childhood and through to Adolescence)

#### i. Antenatal history

- Oligohydramnios
- Maternal disease (e.g. hypertension, infections, diabetes mellitus)
- Drug exposure (e.g. nonsteroidal anti-inflammatory drugs (NSAIDs) and angiotensin-
- Converting enzyme (ACE) inhibitors)
- Routine antenatal ultrasound scanning to diagnose CAKUT

#### ii. Perinatal and infant periods

- Other preceding illnesses and investigations
- Current symptoms of all the major organ systems

#### iii. Family history of kidney diseases:

- Polycystic kidney disease
- Other genetic kidney diseases
- Renal dysplasia or hypoplasia
- Urologic disorders—especially obstructive uropathies

### B. Identify the Seven Red Flags for Kidney Disease in Children

- Failure to thrive
- Elevated blood pressure
- Chronic anemia
- Voiding problems
- Unusual bone disease
- Proteinuria
- Pre-existing kidney disease

## 6.4.11 Investigations of chronic kidney disease (CKD) in children

### A. Laboratory Evaluation

The following laboratory tests are recommended for the assessment and monitoring of CKD in children:

- Urea, Electrolytes, and Creatinine (UEC)
- Full Blood Count (FBC)
- Urinalysis

Table 19: Laboratory Tests and Recommended Frequency for Children With CKD (stages 2–5)

| Laboratory Test            | CKD Stage 2       | CKD Stage 3       | CKD Stage 4       | CKD Stage 5       |
|----------------------------|-------------------|-------------------|-------------------|-------------------|
| Electrolytes               | Every 12 months   | Every 6–12 months | Every 3–6 months  | Every 1–3 months  |
| Calcium / Phosphorus       | Every 12 months   | Every 6–12 months | Every 3–6 months  | Every 1–3 months  |
| Parathyroid Hormone (PTH)  | –                 | Every 6 months    | Every 6–12 months | Every 3–6 months  |
| Alkaline Phosphatase (ALP) | –                 | –                 | Every 12 months   | Every 12 months   |
| 25-Hydroxy Vitamin D       | Every 6–12 months | Every 6–12 months | Every 6–12 months | Every 6–12 months |
| Lipid Profile              | Every 12 months   | Every 12 months   | Every 12 months   | Every 12 months   |
| Hemoglobin                 | –                 | Every 12 months   | Every 6 months    | Every 6 months    |

Source: Adapted from UpToDate recommendations on laboratory monitoring in paediatric CKD stages 2–5.

## B. Renal and Bladder Imaging

### i. Ultrasonography (Ultrasound KUB)

- Shows anatomy of kidneys and bladder
- Diagnoses obstructions, developmental abnormalities, tumors, and stones

### ii. Computerized tomography scan

- Shows better images of kidneys or bladder
- Good at detecting kidney stones and tumours

### iii. Renal nuclear scan Diethylenetriamine Pentaacetic Acid (DTPA) scan and Dimercaptosuccinic Acid (DMSA) scan

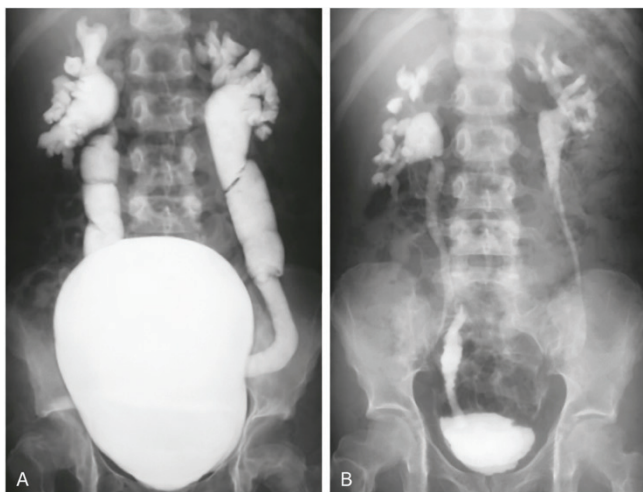
- DTPA: shows and compares both kidneys in size, shape, and function
- DMSA: detects renal scarring due recurrent urinary infection

### iv. Voiding (micturating) cystourethrogram (VCUG or MCU)

- To evaluate the bladder and the ureters

## C. Refer for Kidney Biopsy where Indicated:

- To evaluate kidney histology
- Useful for diagnosis of causes of nephritic and nephrotic syndromes



A micturating cystourethrogram (MCU) in a 5-year-old girl with recurrent urinary tract infections demonstrates a large, smooth-walled urinary bladder with bilateral high-grade (Grade V) vesicoureteric reflux.

During voiding, contrast is seen refluxing freely into grossly dilated and tortuous ureters and severely dilated renal pelvicalyceal systems.

Post-void images demonstrate persistent bilateral hydroureteronephrosis with retained contrast within the upper urinary tracts, findings consistent with severe vesicoureteric reflux associated with significant functional and anatomical abnormality.

Ref: Lebowitz RL, Olbing H, Parkkulainen KV, Smellie JM, Tamminen-Möbius TE. International system of radiographic grading of vesicoureteric reflux. *Pediatric Radiology*. 1985;15(2):105–109.

*Figure 19: Micturating Cystourethrogram of Vesicoureteric Reflux*

## 6.4.12 Management of CKD in children

### A. Manage the Risk Factors for CKD Progression

- Hypertension: Keep blood pressure below the 90th percentiles
- Proteinuria: ACE inhibitors or ARBs

Target UACR <3.5mg/mmol in boys and <2.5mg/mmol in girls

### B. Ensure Adequate Calorie and Protein Intake

- Dietary protein restriction is not recommended in children

### C. Renal Anaemia

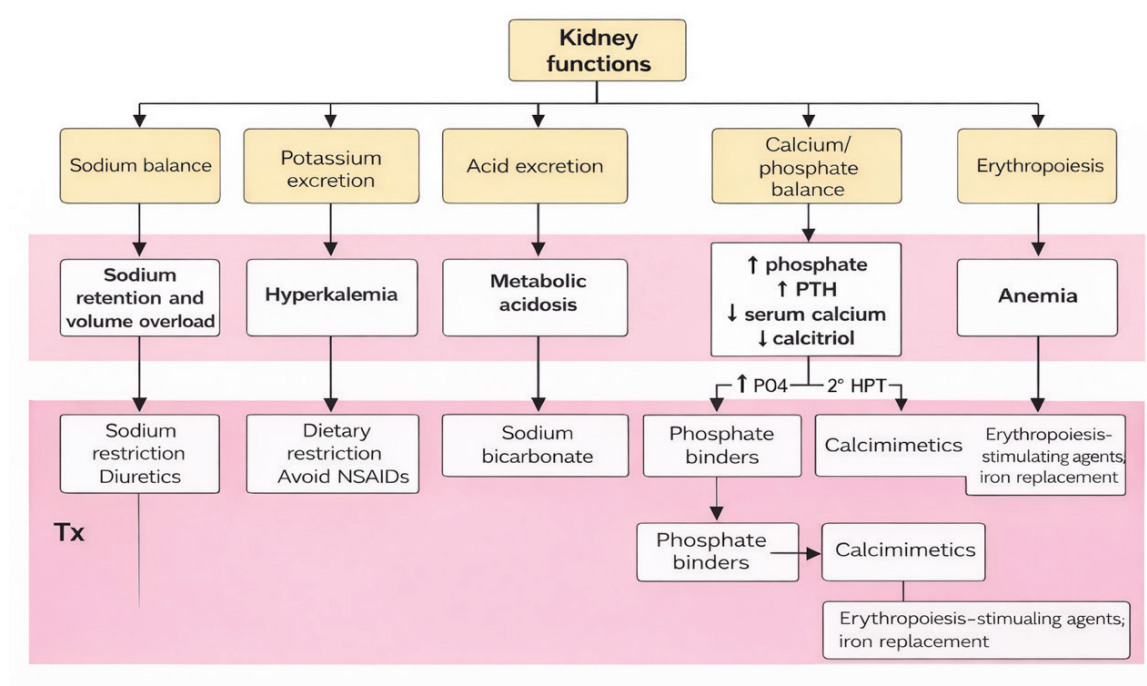
- Oral iron or IV iron
- Erythropoietin stimulating agents

### D. Manage CKD Complications:

- Control hyperlipidemia
- Manage metabolic acidosis: sodium bicarbonate to target serum  $\text{HCO}_3^- > 20 \text{ mmol/L}$
- Secondary hyperparathyroidism:
  - o Supplemental calcium carbonate or acetate
  - o Treat vitamin D deficiency: with vitamin D3
  - o Use vit D analogs: calcitriol

Table 20: Treatment of Complications of CKD

| Complication                                                                                     | Treatment / Management                                                                                                                                                                                                                                                                                                            |
|--------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. Poor growth                                                                                   | <ul style="list-style-type: none"> <li>Ensure adequate caloric intake</li> <li>Correct metabolic acidosis</li> <li>Treat renal osteodystrophy</li> <li>Consider recombinant human growth hormone (rhGH) where indicated</li> </ul>                                                                                                |
| 2. Anaemia                                                                                       | <ul style="list-style-type: none"> <li>Iron supplementation (oral or IV)</li> <li>Erythropoiesis-stimulating agents (e.g., erythropoietin)</li> </ul>                                                                                                                                                                             |
| 3. Renal osteodystrophy / Secondary hyperparathyroidism                                          | <ul style="list-style-type: none"> <li>Active vitamin D (1,25-dihydroxyvitamin D; e.g., calcitriol)</li> <li>Calcium supplementation</li> <li>Dietary phosphorus restriction</li> <li>Phosphate binders</li> </ul>                                                                                                                |
| 4. Cardiovascular complications<br>4a. Hypertension<br>4b. Left ventricular hypertrophy          | <ul style="list-style-type: none"> <li>Antihypertensive medications (ACE inhibitors, ARBs, others as indicated)</li> <li>Volume control (salt restriction, diuretics as appropriate)</li> </ul>                                                                                                                                   |
| 5. Electrolyte abnormalities<br>5a. Hyperkalaemia<br>5b. Hyponatraemia<br>5c. Metabolic acidosis | <ul style="list-style-type: none"> <li>Low potassium diet</li> <li>Loop diuretics (e.g., furosemide)</li> <li>Potassium-binding resins (e.g., sodium polystyrene sulfonate)</li> <li>Sodium supplementation (for hyponatraemia as indicated)</li> <li>Alkali therapy (e.g., sodium bicarbonate) for metabolic acidosis</li> </ul> |



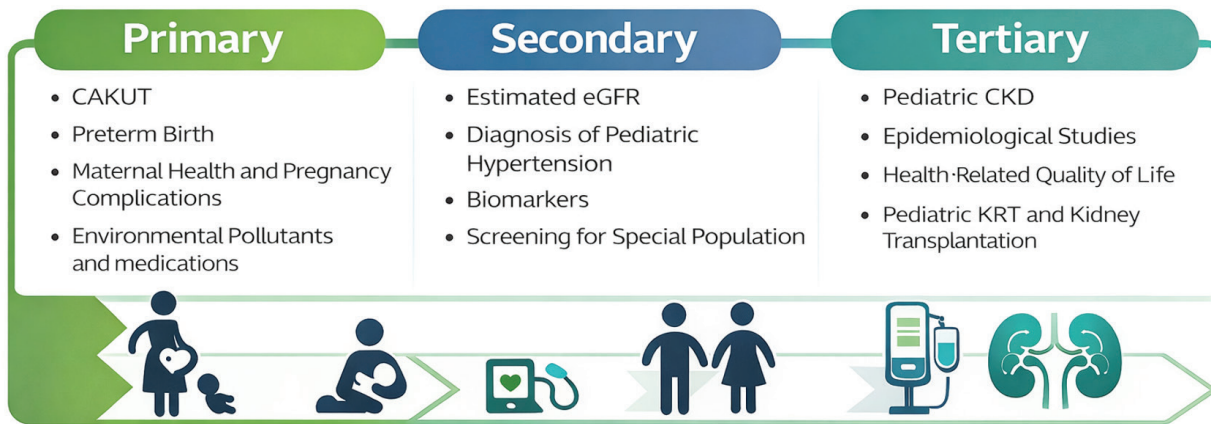
Ref: Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney International Supplements. 2013;3(1):1–150.

Figure 20: Complications and Management of CKD

## 6.4.13 CKD Prevention and Education

### A. CKD Prevention

Prevention of CKD requires a life-course approach to pediatric kidney health—from pregnancy through adulthood—integrating primary prevention, early detection and intervention, and comprehensive tertiary management, including CKD treatment and kidney replacement therapy.



### B. Patient and Family Education in CKD

#### i. Educate the caregivers and, if appropriate, the child with CKD about:

- Natural progression of CKD
- Importance of medical follow up especially with a paediatric nephrologist
- Compliance with secondary preventive measures
- Compliance with prescribed medications
- Compliance with the prescribed diet

#### ii. Highlight types of dialysis and eventually kidney transplantation

- Prepare for renal replacement therapy when child is in CKD stage 4
- Provide information on peritoneal dialysis, haemodialysis and kidney transplant
- Educate regarding preemptive kidney transplant

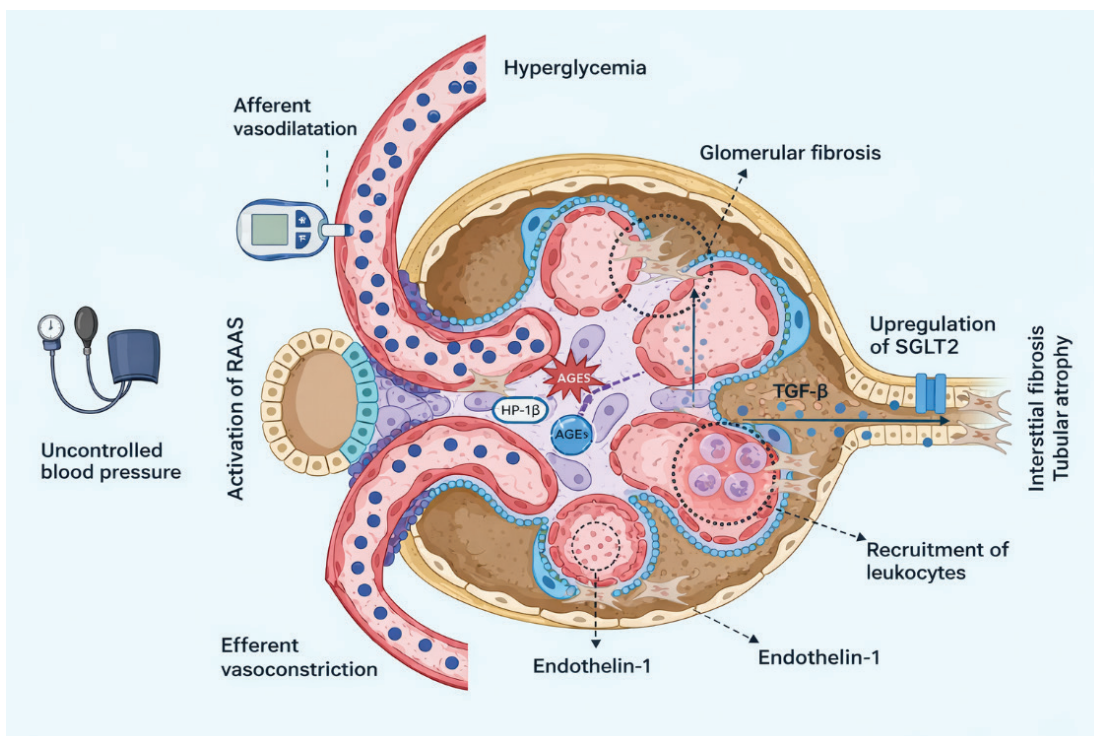
# Chapter 7: Chronic Kidney Disease in Patients with Diabetes Mellitus

## 7.1 Background

- Chronic kidney disease (CKD) is a progressive loss of kidney function caused by sustained kidney damage for more than three months, from various causes.
- Diabetes mellitus is a chronic metabolic disorder characterised by sustained elevated blood glucose (hyperglycemia) resulting from defects in insulin secretion, action, or both.
- Diabetes, if not well controlled, leads to multiple body organ disorders, including diabetic kidney diseases, increasing morbidity and mortality.
- Diabetes mellitus (DM) is classified into Type 1 (DM1) or Type 2 (DM2)

## 7.2 Pathophysiology

- Diabetes classically causes diabetic nephropathy characterised by:
  - o Proteinuria
  - o Worsening GFR
  - o Progressive glomerular hypertension



ROS Reactive oxygen species, HIF Hypoxia inducible factor, TGF-β Transforming Growth factor beta

Figure 21: Pathophysiology of Diabetic Kidney Disease

- However, many patients will still have CKD in DM2, without proteinuria. This is due to comorbid systemic diseases such as dyslipidemia, hyperuricemia, hypertension and other cardiovascular disorders.

## 7.3 Risk Factors for CKD in Diabetes

| MODIFIABLE RISK FACTORS                                                                                                                                                                                                                                                                                                                  | NON MODIFIABLE RISK FACTORS                                                                                                             |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• Alcohol use</li> <li>• Hyperlipidemia</li> <li>• Hypertension</li> <li>• Obesity</li> <li>• Lower Physical activity</li> <li>• Nephrotoxins eg Tobacco, NSAIDs, aspirin, recreational drug use, some nutritional supplements, some antibiotics</li> <li>• Poorly controlled diabetes</li> </ul> | <ul style="list-style-type: none"> <li>• Younger age at diagnosis</li> <li>• Family history of CKD in DM</li> <li>• Male sex</li> </ul> |

## 7.4 Screening for CKD in Diabetes

### 7.4.1 Baseline tests

- Creatinine (or UEC): estimate GFR using the CKD-Epi formula 2021
- Urine dipstick: check for proteinuria
- Urine protein quantification:
  - gold standard: 24h urine protein
  - clinically easier: urine albumin to creatinine ratio (UACR) or urine protein to creatinine ratio (UPCR)
- Kidney / Ureter / Bladder (KUB) ultrasound with post-void bladder volume: assess for chronicity (small kidney sizes, poor corticomedullary differentiation), check for obstruction, check for polycystic kidneys

### 7.4.2 Additional tests

- Full blood count: check haemoglobin level
- Uric acid
- Fasting cholesterol level
- HbA1C - may not be reliable if Hb is below 10g/dl

Primary health care considerations:

- Although dipsticks are insensitive to low levels of urine albumin and perform poorly when interpreted visually, dipstick proteinuria may be used as a CKD screening test in resource-limited settings
- With a positive urine dipstick analysis, primary health care providers should further screen for CKD using:
  - o Urine albumin-creatinine ratio (ACR)
  - o Serum creatinine and estimation of GFR using the CKD-EPI (2021) equation

Note:

- o Protein detected on a urine dipstick is equivalent to albuminuria >300mg/g and indicates severe damage
- o A urine dipstick should only be used where UACR testing is not available
- o A urine microalbumin >30mg/g indicates the beginning of glomerular damage secondary to DM2.
- o Intervention should be initiated immediately if UACR is greater than 30mg/g

## 7.5 Management of CKD in Diabetes

### 7.5.1 Lifestyle recommendations

Lifestyle changes are the cornerstone of DM2 management and should be emphasized.

This includes:

- Physical activity: Participate in at least 30 min of moderate-intensity dynamic aerobic exercise (walking, jogging, cycling, or swimming) on 5–7 days (total 150 min per week)
- Appropriate balanced diet (see table)
- Weight reduction if overweight/ obese
- Smoking cessation
- Cessation of alcohol consumption

## 7.5.2 Dietary recommendations

Where available, a nutritionist should be consulted to give a diet plan that is tailored to the individual needs of the patient.

- Use renal dietitians or accredited nutrition providers to educate people with CKD about dietary adaptations regarding sodium, phosphorus, potassium, and protein intake, tailored to their individual needs, and severity of CKD and other comorbid conditions.
- Consider patients' cultural background when recommending diets
- Patients should not be moved abruptly to a 'renal' diet. Rather, diet should be adjusted gradually to account for hyperkalemia, high urea, and high uric acid.
- Balanced diet:
  - o People with diabetes and CKD 1-3b should consume a balanced, healthy diet that is high in:
    - o Fruits and vegetables e.g. Spinach, kales
    - o Whole grains e.g. Maize, whole wheat grains
    - o Fiber
    - o Legumes e.g. Beans
    - o Plant-based proteins
    - o Unsaturated fats
    - o Nuts
  - o Avoid processed meats, refined carbohydrates, sweetened beverages and alcohol.
- Protein intake:
  - o Should be moderate at 0.8g/kg, which is the normal for the general population.
  - o Lower protein diets (0.6-0.8g/kg) may help reduce uremic symptoms in patients with GFR <20 ml/min/1.73m<sup>2</sup> who are pre-dialysis. In this case, increase starch consumption to prevent malnutrition
  - o Very low protein diets (0.4-0.6g/kg) need supplementation with ketoanalogues, as they may cause severe undernutrition
  - o Higher protein intake is recommended in dialysis patients
- Salt intake:
  - o Low salt diet helps control blood pressure and delays progression of CKD
  - o Sodium intake should be less than 2g/day.

**Note: Refer to Chapter 12 on Nutrition for more details**

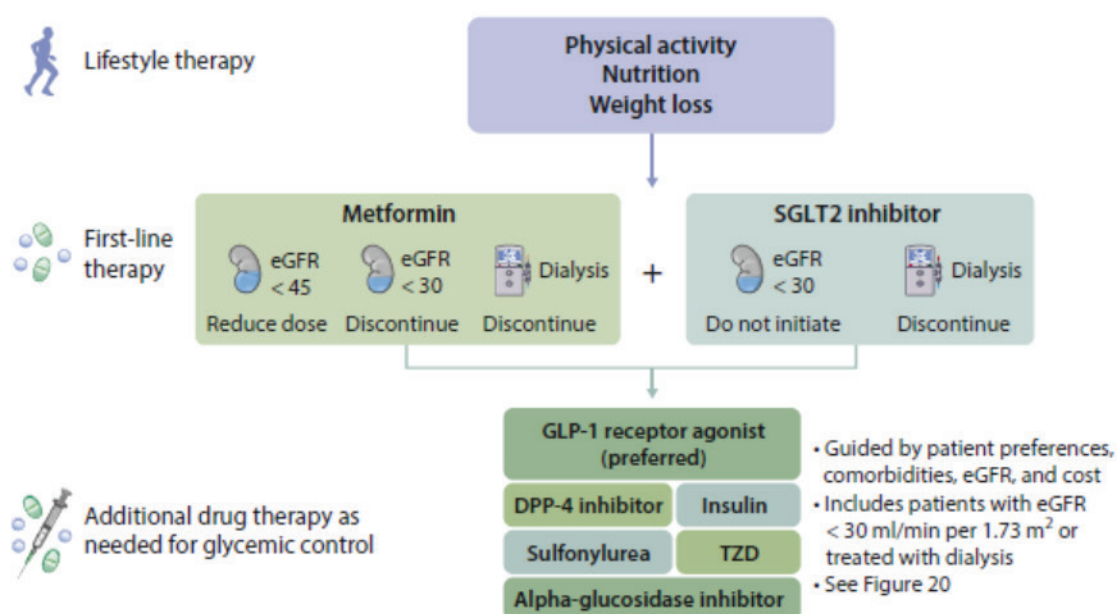
## 7.5.3 Control of hyperglycaemia

### A. Individualised HbA1C Targets in CKD

Individualise A1c targets based on patient profile. See the table below: -

| A1c Target | Patient type                                                                                                          |
|------------|-----------------------------------------------------------------------------------------------------------------------|
| 4-6.5%     | Long life expectancy, No episodes of hypoglycemia                                                                     |
| < 7%       | Optimal for most adults                                                                                               |
| <8%        | CKD stage 4 and lower, elderly, high risk of hypoglycemia, reduced life expectancy, Significant medical comorbidities |

- As GFR reduces, glycemic control improves due to reduced excretion of insulin by the kidneys.
- Monitor sugar control closely as GFR falls <30ml/min/1.73m<sup>2</sup> and taper insulin and oral hypoglycemic agents downwards to avoid hypoglycemic attacks.
- Medications that can safely be used in patients with DM2 and CKD will vary with the patient's GFR:
  - o Most medications will have predictable side effect profiles with a GFR > 45ml/min/1.73m<sup>2</sup> (CKD Stage 3a).
  - o Below 45ml/min/1.73m<sup>2</sup>, some medications shall have to be renal-dosed and some are contraindicated (Figure 22)
  - o Insulin remains the mainstay of management for patients with Type 1 DM. However, with progression of kidney disease insulin requirements also reduce and should be individualised.



DPP-4: dipeptidyl peptidase-4; eGFR: estimated glomerular filtration rate; GLP-1: glucagon-like peptide-1; SGLT2: sodium glucose cotransporter 2; TZD: thiazolidinedione

Figure 22: Management of Diabetes in CKD Patients

- **B. Oral Hypoglycaemic Agents used in CKD**

- The table below summarises recommended dose adjustments and contraindications for commonly used glucose-lowering medications in patients with advanced chronic kidney disease (CKD Stages 3b–5).
- As kidney function declines, drug clearance is reduced and the risk of hypoglycaemia and adverse effects increases. Therefore, careful selection of agents, appropriate dose modification, and specialist oversight in advanced CKD are essential to optimise glycaemic control while minimising harm.

| Drug / Class       | Stage 3b                                                                           | Stage 4             | Stage 5 |  |
|--------------------|------------------------------------------------------------------------------------|---------------------|---------|--|
| Metformin          | Reduce dose to maximum 1g/day                                                      | Contraindicated     |         |  |
| Insulin            | Initiate & titrate cautiously to avoid hypoglycemia.                               |                     |         |  |
| SGLT2 Inhibitors   |                                                                                    |                     |         |  |
| Dapagliflozin      | 10mg OD (Stop at initiation of dialysis or transplant unless other indication)     |                     |         |  |
| Empagliflozin      | 10mg OD (Stop at initiation of dialysis or transplant unless other indication)     |                     |         |  |
| GLP1 agonists      |                                                                                    |                     |         |  |
| Liraglutide        | No dose adjustment required                                                        |                     |         |  |
| Semaglutide        | No dose adjustment required                                                        |                     |         |  |
| DPP4i              |                                                                                    |                     |         |  |
| Linagliptin        | No dose adjustment required                                                        |                     |         |  |
| Sitagliptin        | Max. 50mg daily                                                                    | Max 25mg Once daily |         |  |
| Vildagliptin       | Reduce dose to 50mg daily                                                          |                     |         |  |
| Teneligliptin      | 20mg Once daily                                                                    |                     |         |  |
| Saxagliptin        | 2.5mg OD                                                                           | Contraindicated     |         |  |
| Sulfonylureas      |                                                                                    |                     |         |  |
| Gliclazide         | Initiate cautiously at 30mg daily & titrate slowly to avoid hypoglycemia           |                     |         |  |
| Glibenclamide      | Contraindicated                                                                    |                     |         |  |
| Glimepiride        | Maximum 1mg per day                                                                | Contraindicated     |         |  |
| Thiozolidinediones |                                                                                    |                     |         |  |
| Pioglitazone       | No dose adjustment required (caution in patients with heart failure,liver failure) |                     |         |  |

DPP-4: dipeptidyl peptidase-4; eGFR: estimated glomerular filtration rate; GLP-1: glucagon-like peptide-1; SGLT2: sodium glucose cotransporter 2; TZD: thiazolidinedione

Adapted from the American Diabetes Association guidelines

## 7.5.4 SGLT2i (Sodium-Glucose Co-Transporter 2 Inhibitors)

- Recommended for patients with:
  - o Type 2 diabetes, CKD, and an eGFR  $\geq 20$  ml/min per 1.73 m<sup>2</sup> (diabetic kidney disease)
  - o No diabetes and:
    - eGFR  $\geq 20$  ml/min per 1.73 m<sup>2</sup> with urine ACR  $\geq 200$  mg/g ( $\geq 20$  mg/mmol) (proteinuric kidney disease)
    - eGFR 20 to 45 ml/min per 1.73 m<sup>2</sup> with urine ACR  $< 200$  mg/g ( $< 20$  mg/mmol) (moderately severe non-proteinuric kidney disease CKD G3b-4)
    - heart failure, irrespective of level of albuminuria.
- Continue SGLT2i even if the eGFR falls below 20 ml/min per 1.73 m<sup>2</sup>, unless it is not tolerated or KRT is initiated.
- Withhold SGLT2i (to avoid euglycaemic ketoacidosis) during:
  - o Prolonged fasting
  - o Surgery
  - o Critical medical illness
- Recommended SGLT2i:
  - o Dapagliflozin 5-10mg once a day
  - o Empagliflozin 10-25mg once a day
  - o Canagliflozin 100-300mg once a day

## 7.5.5 Blood pressure control

- CKD patients tend to develop resistant hypertension as GFR falls. Resistant hypertension is defined as BP  $> 140/90$  mmHg despite the use of more than three medications, including a diuretic, at their maximal dose. (See chapter 5)

## 7.5.6 Renal protection

- In all patients with DM2 and CKD, a renal protective agent must be added on, to the maximal tolerated dose (Table 21)
- These can be used singly or added on sequentially to achieve ACR  $< 30$  mg/g

Table 21: Reno-protective Mediations used in CKD

| Renal protective agent                               | Mechanism of action                                                                                                                                                                                                                                                                                                                                                                                                       |
|------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| ACEi / ARB                                           | <ul style="list-style-type: none"> <li>Causes efferent arteriolar vasodilation, reducing Intraglomerular hypertension.</li> <li>Also have antifibrotic, anti-inflammatory, and nephroprotective effects through BP reduction.</li> </ul>                                                                                                                                                                                  |
| SGLT2 inhibitor                                      | <ul style="list-style-type: none"> <li>Reduce sodium reabsorption in the proximal convoluted tubule, causing, through tubule-glomerular feedback, afferent arteriolar vasoconstriction and reduction in hyperfiltration.</li> <li>Also reduces BP, promotes effective energy substrate utilization in the kidney, regulates autophagy, and maintains cellular homeostasis</li> </ul>                                      |
| Nonsteroidal Mineralocorticoid inhibitor: Finerenone | <ul style="list-style-type: none"> <li>Mineralocorticoid receptor (MR) overactivation leads to glomerular hypertrophy, sclerosis, and renal fibrosis.</li> <li>Finerenone reduces endothelial cell apoptosis, attenuates smooth muscle cell proliferation, decreases leukocyte recruitment, and reduces inflammatory responses after vascular injury. It blocks the kidney damage caused by MR overactivation.</li> </ul> |
| GLP1A: Semaglutide                                   | <ul style="list-style-type: none"> <li>Reduces inflammation, oxidative stress, and fibrosis. Renal &amp; immune cells express GLP1 Receptors, and GLP1A reduces profibrotic and proinflammatory mediators expressed by these cells. Causes weight reduction.</li> </ul>                                                                                                                                                   |

### 7.5.7 Lipid management

- Statin therapy does not alter the natural progression of CKD in DM2 but it does reduce the risk of cardiac events.
- All DM2 patients above the age of 40 should be on a high-intensity statin, preferably atorvastatin.
- Aim for LDL cholesterol of <1.8mmol/l.

### 7.5.8 General recommendations

Table 22 summarises general recommendations on management of patients with type 2 diabetes and CKD

Table 22: General management Recommendations for Patients with Type 2 Diabetes and CKD

| Care Domain                                    | Key Recommendations / Details                                                                                                                                                                                                                                                                                                                                                         |
|------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Multidisciplinary team based care              | <p>A multidisciplinary approach should be used for optimal care of patients with type 2 diabetes and CKD. The team should include:</p> <ul style="list-style-type: none"> <li>• Primary care physician</li> <li>• Nephrologist</li> <li>• Endocrinologist</li> <li>• Nutritionist / dietitian</li> <li>• Nursing and management support</li> <li>• Psychologist</li> </ul>            |
| Avoidance of nephrotoxins                      | <p>Nephrotoxic agents should be avoided to prevent further kidney damage. These include:</p> <ul style="list-style-type: none"> <li>• Non steroidal anti inflammatory drugs (NSAIDs)</li> <li>• Nephrotoxic antimicrobials (e.g. aminoglycosides, deoxycholate amphotericin B)</li> <li>• Iodinated contrast agents (use with caution and only with nephrologist guidance)</li> </ul> |
| Patient education                              | <p>Continuous education should be provided on:</p> <ul style="list-style-type: none"> <li>• Glycaemic control</li> <li>• Lifestyle modification</li> <li>• Strategies to delay progression of chronic kidney disease</li> </ul>                                                                                                                                                       |
| Sick day rules – when to apply                 | <p>Sick day rules apply when patients are at risk of dehydration, including when the patient is:</p> <ul style="list-style-type: none"> <li>• Septic or febrile</li> <li>• Unable to eat or drink adequately</li> <li>• Experiencing diarrhoea (loose motions)</li> <li>• Vomiting</li> </ul>                                                                                         |
| Sick day rules – immediate actions             | <p>During sick days:</p> <ul style="list-style-type: none"> <li>• Rehydrate the patient adequately</li> <li>• Temporarily stop SADMANS medications to prevent acute kidney injury</li> </ul>                                                                                                                                                                                          |
| Medications to stop during sick days (SADMANS) | <ul style="list-style-type: none"> <li>• Sulphonylureas</li> <li>• ACE inhibitors (ACEi)</li> <li>• Diuretics</li> <li>• Metformin</li> <li>• Angiotensin receptor blockers (ARB)</li> <li>• NSAIDs</li> <li>• SGLT2 inhibitors (SGLT2i)</li> </ul>                                                                                                                                   |
| Restarting medications                         | <p>All temporarily stopped medications should be restarted only after dehydration has resolved and the patient is clinically stable</p>                                                                                                                                                                                                                                               |

# CHOOSING GLUCOSE-LOWERING MEDICATION IN THOSE WITH ESTABLISHED ATHEROSCLEROTIC CARDIOVASCULAR DISEASE (ASCVD) OR CHRONIC KIDNEY DISEASE (CKD)



Use principles in Figure 1



TO AVOID CLINICAL INERTIA REASSESS AND MODIFY TREATMENT REGULARLY (3-6 MONTHS)

Use metformin unless contraindicated or not tolerated

If not at HbA<sub>1c</sub> target:

- Continue metformin unless contraindicated (remember to adjust dose/stop metformin with declining eGFR)
- Add SGLT2i or GLP-1 RA with proven cardiovascular benefit<sup>1</sup> (See below)

If at HbA<sub>1c</sub> target:

- If already on dual therapy, or multiple glucose-lowering therapies and not on an SGLT2i or GLP-1 RA, consider switching to one of these agents with proven cardiovascular benefit<sup>1</sup> (See below)

OR reconsider/lower individualised target and introduce SGLT2i or GLP-1 RA

OR reassess HbA<sub>1c</sub> at 3 month intervals and add SGLT2i or GLP-1 RA if HbA<sub>1c</sub> goes above target

ASCVD predominates



HF or CKD predominates



EITHER/  
OR

GLP-1 RA with proven CVD benefit<sup>1</sup>

SGLT2i with proven CVD benefit<sup>1</sup>, if eGFR adequate<sup>2</sup>

PREFERABLY

SGLT2i with evidence of reducing HF and/or CKD progression in CVOTs if eGFR adequate<sup>2</sup>

OR

If SGLT2i not tolerated or contraindicated or if eGFR less than adequate<sup>2</sup> add GLP-1 RA with proven CVD benefit<sup>1,4</sup>

If HbA<sub>1c</sub> above target

If HbA<sub>1c</sub> above target

If further intensification is required or patient is unable to tolerate GLP-1 RA and/or SGLT2i, choose agents demonstrating CV safety:

- Consider adding the other class (GLP-1 RA or SGLT2i) with proven CVD benefit<sup>1</sup>
- DPP-4i if not on GLP-1 RA
- Basal insulin<sup>5</sup>
- TZD<sup>6</sup>
- SU<sup>7</sup>

- Avoid TZD in the setting of HF

Choose agents demonstrating CV safety:

- Consider adding the other class with proven CVD benefit<sup>1</sup>
- DPP-4i (not saxagliptin) in the setting of HF (if not on GLP-1 RA)
- Basal insulin<sup>5</sup>
- SU<sup>7</sup>

1. Proven CVD benefit means it has label indication of reducing CVD events. For GLP-1 RA strongest evidence for liraglutide > semaglutide > exenatide extended release. For SGLT2i evidence modestly stronger for empagliflozin > canagliflozin.  
2. Be aware that SGLT2i vary by region and individual agent with regard to indicated level of eGFR for initiation and continued use

3. Both empagliflozin and canagliflozin have shown reduction in HF and to reduce CKD progression in CVOTs  
4. Caution with GLP-1 RA in ESRD  
5. Degludec or U100 glargine have demonstrated CVD safety  
6. Low dose may be better tolerated though less well studied for CVD effects  
7. Use lowest generation SU to lower risk of hypoglycaemia

Figure 23: Algorithm for the Management of Type 2 Diabetes in CKD

### 1st line drug therapy

- Metformin if GFR >30ml/min/1.73m<sup>2</sup>
- SGLT2 inhibitor continued until dialysis or transplantation
- RAASi to maximum tolerated dose considering hyperkalaemia. Aim for SBP <130mmHg
- Moderate to high intensity statin

**ADD**

### Targeted therapies for complications

- Albuminuria: SGLT2i, GLP1 agonists, ns-MRA
- Heart Failure: SGLT2i, GLP1 agonists, ns-MRA
- Manage CKD-MBD Mineral bone disease, Acidosis, hyperkalaemia when indicated

Figure 24: Medication Choices for Diabetes Management in CKD

#### NOTE:

In patients with diabetes and advanced chronic kidney disease (CKD stages G4–G5), individualised glucose-lowering therapy is essential due to reduced renal function and the limited suitability of some antidiabetic agents. While SGLT2 inhibitors and GLP-1 receptor agonists remain preferred where clinically indicated because of their renal and cardiovascular benefits, DPP-4 inhibitors—particularly linagliptin, may be considered as a safe and effective option for glycaemic control in patients with severe renal impairment, including those receiving dialysis. Linagliptin does not require dose adjustment in renal dysfunction and has a neutral cardiovascular risk profile, making it a practical option in advanced CKD where treatment choices are often constrained

# Chapter 8: Infections and the Kidneys

## 8.1 Malaria-Associated Acute Kidney Injury

### 8.1.1 Definition of severe malaria

- Severe malaria is a life-threatening complication of *Plasmodium falciparum* infection, characterised by organ dysfunction or failure.
- It requires urgent medical intervention to prevent mortality.

### 8.1.2 Clinical presentation

**Patients with severe malaria may present with one or more of the following:**

- Impaired consciousness (e.g., confusion, coma, Glasgow Coma Scale <11).
- Prostration (inability to sit or stand without assistance).
- Multiple convulsions (>2 episodes in 24 hours).
- Severe anemia (hemoglobin <5 g/dL or hematocrit <15%).
- Acute kidney injury (AKI)- (oliguria or elevated creatinine).
- Hypoglycemia (blood glucose <40 mg/dL or 2.2 mmol/L).
- Metabolic acidosis (pH <7.3 or plasma bicarbonate <15 mmol/L).
- Hyperlactatemia (lactate >5 mmol/L).
- Pulmonary oedema or acute respiratory distress syndrome (ARDS).
- Circulatory collapse or shock (systolic blood pressure <80 mmHg).
- Abnormal bleeding or disseminated intravascular coagulation (DIC).
- Hemoglobinuria (dark urine due to intravascular hemolysis).

### 8.1.3 Diagnostic criteria for severe malaria:

1. Confirmed *Plasmodium falciparum* infection (by microscopy or rapid detection test (RDT))
2. Presence of one or more of the above clinical or laboratory features

### 8.1.4 Clinical approach to malaria-associated AKI (MAKI)

- Clinical suspicion:
  - o Consider MAKI in patients with severe malaria presenting with:
    - Oliguria (<0.5 mL/kg/hour for 6 hours) or anuria
    - Elevated serum creatinine or urea
    - Clinical signs of fluid overload (e.g., pulmonary oedema, peripheral oedema)
    - Electrolyte imbalances (e.g., hyperkalemia, metabolic acidosis)
- Diagnostic workup:
  - o Laboratory tests:
    - Rapid diagnostic test (RDT) or microscopy for malaria
    - Serum creatinine, urea, electrolytes (Na, K, Ca, Mg)
    - Arterial blood gas (ABG) analysis for acid-base status
    - Urinalysis (check for proteinuria, hematuria, or muddy brown casts)
  - o Imaging:
    - Renal ultrasound to rule out obstructive uropathy or chronic kidney disease.

### 8.1.5 Management of malaria-associated AKI

#### A. Prompt antimalarial treatment:

- First-line: IV artesunate (2.4 mg/kg at 0, 12, and 24 hours, then daily).
- If IV artesunate is unavailable, use IV artemether or quinine
- Transition to oral artemisinin-based combination therapy (ACT) once the patient can tolerate oral medications

#### B. Fluid Management:

- Guided by volume status of patient
- Avoid over-hydration, especially in children as this leads to pulmonary and cerebral oedema with attendant mortality
- Hypovolemic patients:
  - o Administer isotonic fluids (e.g., Ringer's Lactate or 0.9% saline) cautiously to restore intravascular volume.
  - o Monitor for signs of fluid overload (e.g., crackles on lung auscultation, jugular venous distension)

- Euvolaemic or Hypervolemic patients:
  - o Restrict fluids to insensible losses (approximately 500 mL/day in adults) plus urine output.
  - o Avoid excessive fluid administration to prevent pulmonary oedema.

### C. Hyperkalemia:

- If serum potassium >6.5 mmol/L or ECG changes (e.g., peaked T waves, widened QRS): Administer IV calcium gluconate (10 mL of 10% solution over 10 minutes) for cardiac stabilization.
- Give IV insulin-glucose (10 units regular insulin with 50 mL of 50% glucose) to shift potassium intracellularly.
- Consider oral or rectal potassium-binding resins (e.g., sodium polystyrene sulfonate)

### D. Metabolic Acidosis:

- Correct underlying causes (e.g., hypoxia, dehydration).
- Consider IV sodium bicarbonate (1-2 mEq/kg) only if pH <7.1 or severe bicarbonate deficiency.

## 8.1.6 Renal replacement therapy (RRT) for malaria-associated AKI

## 8.1.7 Supportive care for malaria-associated AKI

- Nutrition:
  - o Provide adequate caloric intake (e.g., 25-30 kcal/kg/day).
  - o Restrict protein intake to 0.8-1.0 g/kg/day in non-catabolic patients.
  - o Correct micronutrient deficiencies (e.g., iron, folate).
- Infection prevention:
  - o Strict aseptic techniques for catheter insertion and dialysis procedures.
  - o Monitor for secondary infections (e.g., bacterial sepsis).
- Pain and symptom management:
  - o Use paracetamol for fever or pain; avoid NSAIDs (nephrotoxic).
  - o Treat nausea/vomiting with antiemetics (e.g., metoclopramide).

Refer to dialysis chapter 10

### 8.1.8 Monitoring and follow-up of malaria-associated AKI

- Daily Monitoring:
  - o Vital signs, urine output, fluid balance, and weight.
  - o Serum creatinine, electrolytes, and acid-base status.
- Long-Term Follow-Up:
  - o Assess renal function recovery post-discharge.
  - o Educate patients on malaria prevention (e.g., insecticide-treated nets, chemoprophylaxis).

### 8.1.9 Prevention of malaria-associated AKI

- Community education:
  - o Promote early diagnosis and treatment of malaria.
  - o Encourage use of insecticide-treated nets and indoor residual spraying.
- Health System Strengthening:
  - o Ensure availability of antimalarial drugs and diagnostic tools.
  - o Train healthcare workers in early recognition and management of MAKI.

## 8.2. Kidney Disease in HIV

### 8.2.1 Introduction

The risk of both acute and chronic kidney disease is higher in people living with HIV (PLHIV) compared to the general population. Risk factors for kidney disease in PLHIV include: increasing age, co-morbid conditions like diabetes mellitus and hypertension, volume depletion as a consequence of gastroenteritis secondary to GIT opportunistic infections, sepsis, drug-induced nephrotoxicity, and direct effects of HIV on the kidney.

Holistic management of PLHIV must thus involve screening for risk factors of kidney disease and instituting appropriate and timely strategies to reduce progression of kidney disease in these patients.

### 8.2.2 Epidemiology of kidney disease in PLHIV

PLHIV are at a 2-20-fold higher risk of developing end-stage kidney disease compared to the general population, with the risk highest in patients of African origin.

### 8.2.3 Acute kidney injury (AKI) in PLHIV

- Acute kidney injury is defined as an absolute increase in serum creatinine of  $\geq 26.5 \mu\text{mol/L}$  ( $\geq 0.3 \text{mg/dl}$ ) within 48 hours, or more than 1.5 times increase from the baseline serum creatinine within the last 7 days
- Where urine output can be measured, AKI can also be defined as a reduction in urine output to less than  $0.5 \text{ml/kg/hour}$  for 6 hours

#### A. Risk factors for acute kidney injury in patients living with HIV include:

- Older age
- Low CD4 count
- High viral load
- Advanced HIV disease
- Certain antiretroviral medications
- Concomitant diabetes mellitus
- Presence of chronic kidney disease

#### B. Causes of acute kidney injury in PLHIV

| Causes of AKI in PLHIV |                                                                                                                                                                                                                                                                             |
|------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Prerenal               | <ul style="list-style-type: none"><li>• Dehydration due to vomiting, diarrhoea, reduced oral intake</li><li>• Hypotension</li><li>• Sepsis</li><li>• Liver disease</li><li>• Hypoalbuminemia (malnutrition, proteinuria)</li></ul>                                          |
| Intrarenal             | <ul style="list-style-type: none"><li>• Acute tubular necrosis due to hypovolemia, sepsis, nephrotoxic medications, radiocontrast</li><li>• Parenchymal infection e.g. TB, fungal, viral</li><li>• Glomerular disease e.g. HIVAN</li><li>• Interstitial nephritis</li></ul> |
| Postrenal              | <ul style="list-style-type: none"><li>• Tubular obstruction due to crystalluria, tumor lysis syndrome</li><li>• Ureter or bladder obstruction due to nephrolithiasis, tumor, fungus ball, blood clots</li></ul>                                                             |

### C. Management of acute kidney injury in PLHIV

Management of AKI will depend on the cause:

- Volume depletion should be managed with early fluid therapy and correction of electrolyte abnormalities.
- All nephrotoxic medications e.g. Tenofovir Disoproxil Fumarate (TDF), NSAIDs, aminoglycosides must be withdrawn.
- Drugs that reduce intraglomerular pressure e.g. ACE inhibitors and angiotensin receptor blockers should be temporarily stopped, as should diuretics that may further worsen volume depletion.
- Sepsis must also be treated aggressively using appropriate antibiotics since sepsis-associated AKI is associated with high mortality.

## 8.2.4 Chronic kidney disease (CKD) in PLHIV

### A. Definition

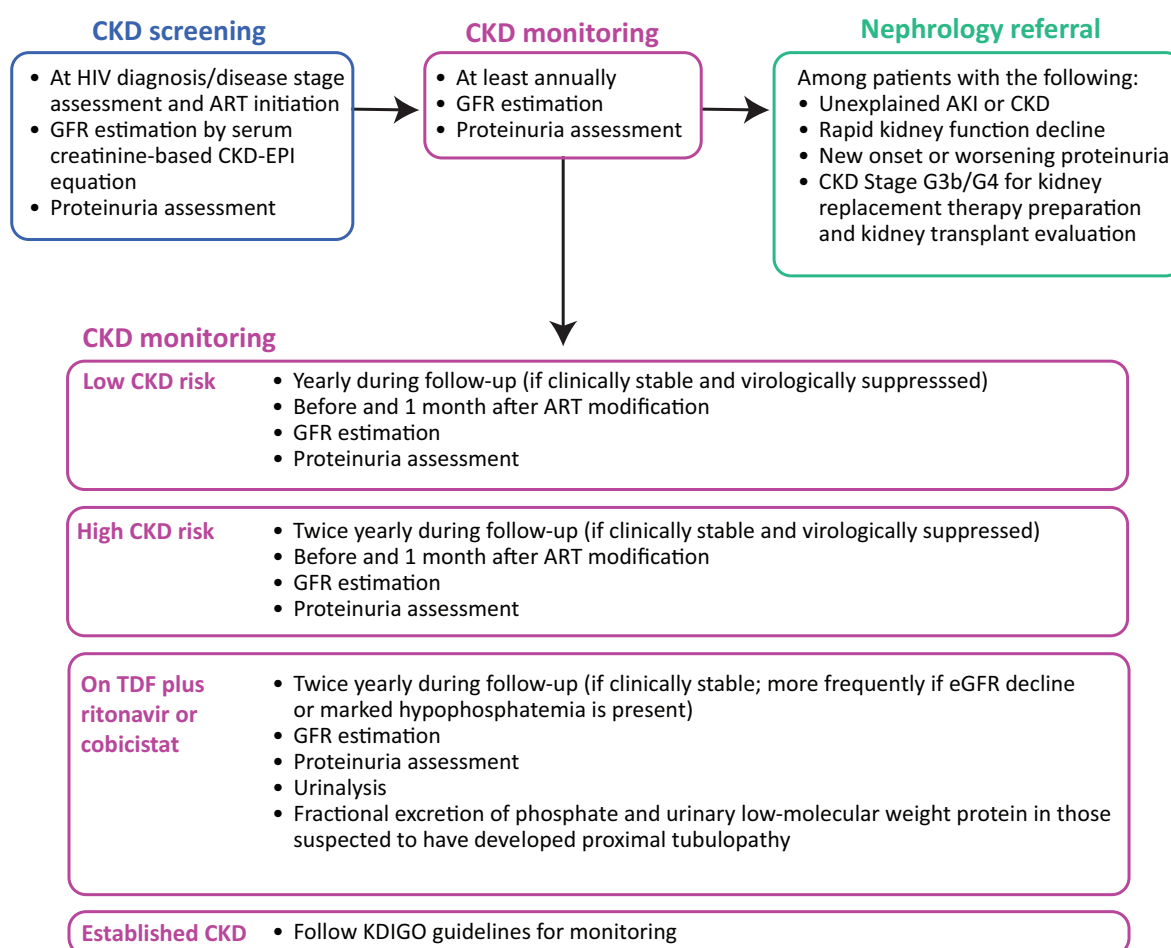
- Chronic kidney disease is defined as abnormal kidney function or structure that is present for at least three months
- Damage to the kidneys in PLHIV leading to CKD may broadly be due to:
  - i. HIV-associated nephropathy (HIVAN)
  - ii. HIV-associated immune complex kidney disease (HIVICK)
  - iii. Drug-induced tubular and interstitial damage
  - iv. Thrombotic microangiopathy (TMA)
- HIVAN:
  - o Due to direct HIV infection of renal tubular epithelial cells
  - o Prevalence has been steadily decreasing with the advent of effective antiretroviral therapy
  - o Classically presents as heavy proteinuria and reduced eGFR
- HIVICK:
  - o Due to deposition of immune complexes (antigen-antibody complexes) within the glomeruli, with resultant immune-mediated damage to the kidneys
- Drug-induced tubular and interstitial damage:
  - o Tenofovir Disoproxil Fumarate (TDF) can cause mitochondrial toxicity leading to proximal renal tubular damage that may present as Fanconi's syndrome (glycosuria in the presence of normal blood glucose, hyperphosphaturia, hypophosphatemia and proteinuria)
  - o Tenofovir Alafenamide (TAF) is associated with a 90% reduction in plasma tenofovir concentration resulting in less renal and bone adverse effects.
- Thrombotic microangiopathy (TMA):
  - o As a result of HIV-mediated endothelial cell injury with subsequent platelet activation, thrombosis and multiorgan dysfunction.

| Category                                                        | Cause / Examples                                                                                                                                                                                                           |
|-----------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>1. HIV-related glomerular disease</b>                        | <ul style="list-style-type: none"> <li>• HIV-associated nephropathy (HIVAN)</li> <li>• HIV-associated immune complex kidney disease (HIVICK)</li> </ul>                                                                    |
| <b>2. Antiretroviral therapy (ART)–associated kidney injury</b> | <ul style="list-style-type: none"> <li>• Tenofovir disoproxil fumarate (TDF)–induced nephrotoxicity</li> <li>• Crystal nephropathy (e.g., Indinavir)</li> <li>• Tubulointerstitial nephritis (e.g., Atazanavir)</li> </ul> |
| <b>3. Infiltrative kidney lesions</b>                           | <ul style="list-style-type: none"> <li>• Kaposi’s sarcoma</li> <li>• Lymphoma</li> </ul>                                                                                                                                   |
| <b>4. Opportunistic infections affecting kidney parenchyma</b>  | <ul style="list-style-type: none"> <li>• Viral: CMV, Parvovirus, Herpes simplex</li> <li>• Fungal: Cryptococcus, Candida</li> <li>• Mycobacterial infections</li> <li>• Bacterial pyelonephritis</li> </ul>                |
| <b>5. Urinary tract obstruction</b>                             | <ul style="list-style-type: none"> <li>• Intrinsic ureteral obstruction (e.g., fungus balls, stones)</li> <li>• Extrinsic ureteral obstruction (e.g., lymphadenopathy)</li> <li>• Bladder outlet obstruction</li> </ul>    |
| <b>6. Thrombotic microangiopathy (TMA)</b>                      | <ul style="list-style-type: none"> <li>• HIV-associated TMA</li> </ul>                                                                                                                                                     |

| <b>Causes of CKD in PLHIV</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ol style="list-style-type: none"> <li>1. HIV-associated nephropathy (HIVAN)</li> <li>2. HIV-associated immune complex kidney disease (HIVICK)</li> <li>3. Antiretroviral therapy-associated <ul style="list-style-type: none"> <li>• TDF-induced nephrotoxicity</li> <li>• Crystal nephropathy- Indinavir</li> <li>• Tubulointerstitial nephritis- Atazanavir</li> </ul> </li> <li>4. Infiltrative lesions of the kidney <ul style="list-style-type: none"> <li>• Kaposi’s sarcoma</li> <li>• Lymphoma</li> </ul> </li> <li>5. Opportunistic infections affecting kidney parenchyma <ul style="list-style-type: none"> <li>• Viral – CMV, Parvovirus, Herpes simplex</li> <li>• Fungal – Cryptococcus, Candida</li> <li>• Mycobacterial</li> <li>• Bacterial pyelonephritis</li> </ul> </li> <li>6. Urinary tract obstruction <ul style="list-style-type: none"> <li>• Intrinsic ureteral obstruction- fungus balls, stones</li> <li>• Extrinsic ureteral obstruction- lymphadenopathy</li> <li>• Bladder outlet obstruction</li> </ul> </li> <li>7. Thrombotic Microangiopathy</li> </ol> |

## B. Screening and early diagnosis of CKD among PLHIV

- Early diagnosis of CKD among PLHIV through effective screening mechanisms during their care at facility level is essential to reduce the number of PLHIV presenting with late stage kidney disease
- Screening for kidney disease should be done at initiation of ARVs and annually thereafter, using serum creatinine (with calculation of eGFR) and urinalysis (looking for proteinuria, haematuria)
- In patients who are at high risk of CKD e.g. with co-morbid diabetes mellitus and hypertension it is recommended to repeat the serum creatinine and urinalysis every six months (see Figure 25 below)
- Early referral to a nephrologist should be done in the following situations:
  - o If eGFR decreases by more than 25% from the baseline
  - o New onset proteinuria is noted on urinalysis
  - o eGFR less than 30ml/min/1.73m<sup>2</sup>



1 Adapted from: Swanepoel C, et al. Kidney disease in the setting of HIV infection: Conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) controversies conference. *Kidney International* 2018;93(3): 545–559.

\*Urinalysis should be performed in all HIV-positive individuals to detect worsening or new onset of proteinuria or hematuria.

\*\*More frequent monitoring is recommended in persons who are clinically unstable, severely immunocompromised, or viremic.

#Concomitant use of TDF plus ritonavir or the pharmacoenhancer cobicistat may increase the risk of TDF-associated nephrotoxicity.

*Figure 25: Recommendations for Kidney Disease Screening and Monitoring in PLHIV (KDIGO 2018)*

## **C. Management of CKD in PLHIV**

### **i. Antiretroviral therapy**

- Effective antiretroviral therapy has led to:
  - o Decline in the incidence of HIVAN
  - o Reduced progression of HIVAN to end stage kidney disease
  - o Reduction in incidence of HIV-associated thrombotic microangiopathy
- Essential to adjust doses of ARVs that are excreted predominantly via the kidneys once renal function begins to decline, as incorrect dosing is associated with higher mortality risk
- Avoid use of fixed dose combinations among PLHIV once eGFR is less than 60ml/min.

### **ii. Management of TDF-associated nephrotoxicity**

- Patients at higher risk for developing TDF-associated renal toxicity include those with:
  - o Pre-existing renal disease
  - o Hypertension
  - o Diabetes mellitus
  - o Severe wasting (weight below 60 kg in adults)
  - o Age > 45 years
  - o WHO stage 3 or 4
  - o CD4 < 200 cells/mm<sup>3</sup>
  - o High HIV viral load
  - o Concomitant nephrotoxic agents, e.g. acyclovir, NSAIDs and ritonavir
- TDF should generally be avoided if the eGFR is less than 50ml/min per 1.73m<sup>2</sup>
- In PLHIV who experience a more than 25% reduction in eGFR from baseline and an eGFR less than 50ml/min/1.73m<sup>2</sup> while on TDF, it should be discontinued and substituted with another 'renal-friendly' ARV such as Abacavir

### **iii. ACE inhibitors or Angiotensin Receptor Blockers (ARBs)**

- Use of ACE inhibitors e.g. Enalapril and ARBs such as Losartan and Telmisartan has been associated with reduced progression of chronic kidney disease to end stage kidney disease
- Treatment with one of these agents is recommended in patients with significant albuminuria (>30mg albumin/day in diabetic patients and >300mg/day in nondiabetic patients).

#### **iv. Control of blood pressure**

- Blood pressure target among patients with CKD is  $\leq 140/90$  mmHg
- The BP target is more stringent among CKD patients with moderately to severely increased albuminuria ( $\leq 130/80$  mmHg)
- Several classes of antihypertensive agents can be used to achieve target BP control, including ACE inhibitors/ARBs, calcium channel blockers, thiazide diuretics, beta adrenoceptor blockers, vasodilators such as hydralazine and centrally acting agents such as alpha methyldopa.

#### **v. Other considerations in management of CKD in PLHIV**

As in the non-HIV CKD population, the following issues must also be addressed among PLHIV with CKD:

- Address other cardiovascular risk factors (hyperlipidemia, weight loss, smoking cessation)
- Avoidance of nephrotoxins including NSAIDs
- Management of other complications of CKD including anaemia, chronic kidney disease mineral and bone disorders, hyperuricemia and metabolic acidosis.

#### **vi. Vaccination**

- PLHIV with CKD should get vaccinated against:
  - o Hepatitis B
  - o Influenza
  - o Pneumococcal
- Immune response to vaccination may be suboptimal due to the double immunosuppression from HIV and CKD

#### **vii. Appropriate drug dosing in PLHIV with CKD**

- Drugs that are predominantly excreted via the kidneys must be dose adjusted according to kidney function to avoid accumulation and resultant drug adverse effects
- Apart from Abacavir, other NRTIs will require dose adjustment in patients with chronic kidney disease
- No dose adjustment is required for NNRTIs, protease inhibitors and dolutegravir (see Table 23)

Table 23: Drug Dose Adjustment for ARVs in Adults with CKD

| ARV                                          | eGFR                                   |                             |                                     |                                 | Intermittent<br>Haemodialysis         | Peritoneal<br>dialysis |
|----------------------------------------------|----------------------------------------|-----------------------------|-------------------------------------|---------------------------------|---------------------------------------|------------------------|
|                                              | ≥50ml/min                              | 30-<br>49ml/min             | 10-29ml/min                         | <10ml/min                       |                                       |                        |
| <b>NRTIs</b>                                 |                                        |                             |                                     |                                 |                                       |                        |
| <b>Abacavir</b>                              | 300mg<br>q12h or<br>600mgq24h          | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>Emtricitabine</b>                         | 200mg q24h                             |                             | 200mg q72h                          | 200mg q96h                      | 200mg q24h                            | No data                |
| <b>Lamivudine</b>                            | 300mg<br>q24h                          |                             | 100mg q24h                          | 25-50mg q24h                    | 25-50mg q24h                          | 25-50mg<br>q24h        |
| <b>Tenofovir<br/>Disoproxil<br/>Fumarate</b> | 300mg<br>q24h                          | 300mg<br>q48h               | Not<br>recommended                  | Not<br>recommended              | 300mg q7d                             | No data                |
|                                              |                                        |                             | If no alternative:<br>300mg q72-96h | If no alternative:<br>300mg q7d |                                       |                        |
| <b>Tenofovir<br/>Alafenamide</b>             | 25mg q24h                              |                             |                                     | No data                         | 25mg q24h                             | No data                |
| <b>Zidovudine</b>                            | 300mg q12h                             |                             |                                     | 100mg q8h                       | 100mg q8h                             | No data                |
| <b>NNRTIs</b>                                |                                        |                             |                                     |                                 |                                       |                        |
| <b>Efavirenz</b>                             | 600mg<br>q24h                          | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>Nevirapine</b>                            | 200mg<br>q12h                          | No dose adjustment required |                                     |                                 | Additional<br>200mg after<br>dialysis | No dose<br>adjustment  |
| <b>Rilpivirine</b>                           | 25mg q24h                              | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>INSTIs</b>                                |                                        |                             |                                     |                                 |                                       |                        |
| <b>Dolutegravir</b>                          | 50mg q24h                              | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>Raltegravir</b>                           | 400mg<br>q12h<br>or 1200mg<br>q24h     | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>PIs</b>                                   |                                        |                             |                                     |                                 |                                       |                        |
| <b>ATV/r</b>                                 | 300/100mg<br>q24h                      | No dose adjustment required |                                     |                                 | Not<br>recommended                    | No dose<br>adjustment  |
| <b>DRV/r</b>                                 | 800/100mg<br>q24h<br>600/100mg<br>q12h | No dose adjustment required |                                     |                                 |                                       |                        |
| <b>LPV/r</b>                                 | 400/100mg<br>q12h                      | No dose adjustment required |                                     |                                 |                                       |                        |

\*\*Updated dosing guidance on Lamivudine: Can be dosed at 300mg every 24 hours even in patients with eGFR 30-49ml/min or lower. Lamivudine is well tolerated with a wide therapeutic window.

Source: Mounzer K, et al. To dose-adjust or not to dose-adjust: Lamivudine dose in kidney impairment. AIDS 2021; 35(8): 1201-1208.

*Table 24: Summary of Strategies to Reduce CKD Progression in PLHIV*

- Early diagnosis of CKD through annual screening using serum creatinine and urinalysis.
- Use of effective antiretroviral therapy to reduce risk of HIVAN
- Discontinue TDF if eGFR drops to less than 50mls/min/1.73m<sup>2</sup>
- Avoid nephrotoxins including NSAIDs, herbal medications
- Aggressive management of acute kidney injury depending on the cause
- Use of ACE inhibitors/ARBs in patients with proteinuria
- Control of co-morbid hypertension and diabetes mellitus to target levels
- Lifestyle changes- smoking cessation, weight loss, reduce alcohol intake
- Early referral to a nephrologist if eGFR declines by more than 25% from the baseline or new onset proteinuria noted

# Chapter 9: Kidney Disease and Pregnancy

## 9.1 Introduction

### Renal Changes During Pregnancy:

Certain changes occur in the renal and urinary system during pregnancy which are physiological (and therefore considered to be normal)

### Anatomic Changes

- Kidney size increases by 1-1.5 cm due to collecting system dilation, influenced by progesterone.
- Ureteral and pelvic dilation occurs, especially on the right side. This can last up to 12 weeks postpartum and should not be confused with hydronephrosis on imaging.

### Hemodynamic and Physiologic Changes

- Glomerular filtration rate (GFR) increases by 50% in the second trimester, causing hyperfiltration.
- Serum creatinine and BUN levels fall; any creatinine >75umol/L is abnormal.
- Renal plasma flow rises by 85% due to increased cardiac output and renal vasodilation.
- Blood pressure decreases from vasodilation. BP above >120mmHg or >80mmHg diastolic should raise concern in pregnancy.
- Sodium retention and blood volume increase, possibly causing benign oedema.
- Pregnancy increases glucose filtration, leading to renal glycosuria, while increased uric acid clearance lowers serum uric acid.

### Urinary Abnormalities

- Proteinuria up to 300 mg/day is normal, but higher levels suggest kidney disease or preeclampsia.
- Bacteriuria is common due to urinary stasis, with a risk of UTI or pyelonephritis.

## 9.2 Acute Kidney Injury in Pregnancy

### 9.1.2 Epidemiology

- **Incidence:** AKI in pregnancy is rare but increasing, rising from 2.29 to 4.52 per 10,000 deliveries (1998-2008). Higher rates in Black and Native American women.
- **Dialysis Requirement:** AKI requiring dialysis occurs in 1 per 10,000 pregnancies, with increased mortality risk.

## 9.2.2. Aetiology

| Early                                                                                                                                                                                                                                                                                                                                                    | Late                                                                                                                                                                          | Others                                                                                                                                                                                                                                                                                                                                                                                                               |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• Prerenal azotemia</li> <li>• Acute tubular necrosis</li> <li>• Renal cortical necrosis</li> <li>• Pyelonephritis</li> <li>• Thrombotic thrombocytopenic purpura</li> <li>• Preeclampsia (significant AKI risk factor)</li> <li>• Hemolytic-uremic syndrome</li> <li>• Acute fatty liver of pregnancy</li> </ul> | <ul style="list-style-type: none"> <li>• Preeclampsia (significant AKI risk factor)</li> <li>• Hemolytic-uremic syndrome</li> <li>• Acute fatty liver of pregnancy</li> </ul> | <ul style="list-style-type: none"> <li>• <b>Obstructive Uropathy:</b> Can occur due to gravid uterus, polyhydramnios, kidney stones, or fibroids.</li> <li>• <b>Nephrolithiasis:</b> Increased risk due to hypercalciuria from elevated vitamin D levels.</li> <li>• <b>Antiphospholipid Syndrome:</b> Requires screening for antibodies; treatment may include aspirin or heparin based on risk factors.</li> </ul> |

## 9.2.3 Management

### A. Acute Kidney Injury in Early Pregnancy:

#### I. Prerenal Azotemia

- Common Cause: Hyperemesis gravidarum (persistent vomiting, metabolic alkalosis).
- Treatment:
  - Antiemetics:
    - Pyridoxine (Vitamin B6) 10mg + Doxylamine 10mg (Nosic) 2 tablets stat (nocte) then 1 tablet TID to QID
    - Pyridoxine 12.5mg to 25mg TID to QID
    - Ondansetron 4mg TID to 8mg TID
    - Metoclopramide 10mg TID
    - Promethazine 12.5mg to 25mg TID to QID
  - If persists:
    - Consult obstetrician
    - Give IV rehydration: normal saline (avoid Ringer's Lactate or Hartmann's)
    - Give IV antiemetics: Ondansetron or Metoclopramide
    - Replace potassium as needed
- Other Cause: Hemorrhage from spontaneous abortion.

## ii. Acute Tubular Necrosis (ATN)

- First Trimester Cause: Hemorrhage (spontaneous abortion) or septic abortion (usually E. coli or Clostridium).
- Treatment: Fluid resuscitation, antibiotics if needed
- Severe Cases: Haemodialysis if indicated

## iii. Renal Cortical Necrosis (RCN)

- Rare but severe: More common in developing countries, often due to post-abortion sepsis.
- Symptoms: Hematuria, severe oliguria/anuria
- Diagnosis: CT scan shows radiolucent rim in cortex
- Management: requires haemodialysis
- Recovery: Slow and often incomplete, leading to end-stage renal disease

## iv. Acute Pyelonephritis

- Presentations: Most common in the second trimester. Lower urinary tract symptoms + Flank pain + systemic symptoms (fever and chills, nausea and vomiting)
- Diagnosis: Urine MCS, Blood culture and sensitivity
- Treatment: Hospitalization, IV antibiotics (e.g., ceftriaxone) and intravenous fluids.

## v. Thrombotic Thrombocytopenic Purpura (TTP)

- Presentation:
- More likely in the first trimester
- Presents with anemia (microangiopathic haemolytic anaemia), thrombocytopenia, and renal dysfunction

*Treatment:* Refer to nephrologist for plasmapheresis and further management

## B. Acute Kidney Injury in Late Pregnancy:

| Acute Tubular Necrosis (ATN)                                                                                                                                                                                                             | Acute Fatty Liver of Pregnancy (AFLP)                                                                                                                                                                                                                                                                                                                                                                |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• <i>Causes:</i> Preeclampsia, HELLP syndrome, uterine hemorrhage (abruptio placentae).</li><li>• <i>Treatment:</i> Manage underlying cause, fluid resuscitation, dialysis if necessary.</li></ul> | <ul style="list-style-type: none"><li>• <i>Rare disorder:</i> Occurs after week 34, with abdominal pain and jaundice.</li><li>• <i>Diagnosis:</i> Lab results (high bilirubin, mild AST/ALT elevation). Imaging (ultrasound, CT): evidence of fatty liver.</li><li>• <i>Complications:</i> Hypoglycemia, coagulopathy, AKI</li><li>• <i>Treatment:</i> Immediate delivery, supportive care</li></ul> |

## 9.3 Pregnancy in CKD

### 9.3.1 Implications of pregnancy in a woman with CKD

| A. Risks for the patient              | B. Risks for the fetus           |
|---------------------------------------|----------------------------------|
| Hypertension                          | Foetal loss: miscarriage or IUFD |
| Pre-eclampsia or eclampsia            | Foetal growth regression         |
| Progression of CKD                    | Premature delivery               |
| Kidney failure with need for dialysis |                                  |

### 9.3.2 Structure of care

- In view of these risks, pregnancy for a patient with CKD has to be planned, with involvement of a multidisciplinary team:
  - o Obstetrician
  - o Nephrologist/expert physician
  - o Expert midwife or midwifery team
- Role of the multidisciplinary team will be to offer advice and care for women with CKD who are pregnant or planning a pregnancy

### 9.3.3 Pre-Pregnancy care of CKD patient

| Clinical Area                             | Key Recommendations / Guidance                                                                                                                                                                                                                                                                                                                 |
|-------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Contraceptive Advice                      | <p>Offer safe and effective contraception to all reproductive-age women with CKD, especially those:</p> <ul style="list-style-type: none"> <li>• Receiving teratogenic medication</li> <li>• With active glomerulonephritis</li> <li>• Within one year of renal transplantation</li> <li>• Within one year of acute graft rejection</li> </ul> |
| Preferred Contraceptive Agents            | <ul style="list-style-type: none"> <li>• Progesterone-only pill</li> <li>• Progesterone subdermal implant</li> <li>• Progesterone intra-uterine system</li> </ul>                                                                                                                                                                              |
| Emergency Contraception                   | <ul style="list-style-type: none"> <li>• Progesterone-only emergency contraception</li> </ul>                                                                                                                                                                                                                                                  |
| Pre-Pregnancy Counselling                 | <p>Women with CKD considering pregnancy should receive counselling by a multidisciplinary team.</p>                                                                                                                                                                                                                                            |
| Risks to Discuss During Counselling       | <p>Advise that CKD increases the risk of:</p> <ul style="list-style-type: none"> <li>• Pre-eclampsia</li> <li>• Preterm birth</li> <li>• Fetal growth restriction</li> <li>• Neonatal unit (NNU) admission</li> <li>• Increased risk of caesarean delivery</li> </ul>                                                                          |
| Optimizing Maternal and Neonatal Outcomes | <ul style="list-style-type: none"> <li>• Optimize glycaemic control in women with diabetes mellitus</li> <li>• Minimize risk of exposure to teratogenic medications</li> <li>• Make a treatment plan in the event of hyperemesis</li> </ul>                                                                                                    |

### 9.3.4 Pregnancy care of CKD patient

| Domain         | Component                                 | Recommendation / Action                                                                                                                                                                                                                                                                                                                                                                                                                                                        |
|----------------|-------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Pregnancy Care | Assessment of renal function in pregnancy | <ul style="list-style-type: none"> <li>• Use serum creatinine</li> <li>• Estimated GFR (eGFR) is not valid for use in pregnancy</li> </ul>                                                                                                                                                                                                                                                                                                                                     |
|                | Quantification of proteinuria             | <ul style="list-style-type: none"> <li>• Formally quantify proteinuria in pregnancy by:</li> <li>• Urine protein: creatinine ratio (uPCR)</li> <li>• Urine albumin: creatinine ratio (uACR)</li> <li>• 24-hour urine collection for quantification of protein is not required</li> </ul>                                                                                                                                                                                       |
| Antenatal Care | General antenatal management              | <p>Pregnant women with CKD should:</p> <ul style="list-style-type: none"> <li>• receive routine antenatal care, in addition to specialist input</li> <li>• be referred for assessment by a consultant obstetrician</li> <li>• have access to usual trisomy screening</li> <li>• have scans to assess fetal growth and wellbeing in the third trimester</li> <li>• be screened for gestational diabetes, especially if on prednisolone and/or calcineurin inhibitors</li> </ul> |
|                | Pre-eclampsia prophylaxis                 | <ul style="list-style-type: none"> <li>• Low-dose aspirin (75–150 mg) in all women with CKD</li> </ul>                                                                                                                                                                                                                                                                                                                                                                         |
|                | Blood pressure (BP) management            | <ul style="list-style-type: none"> <li>• Target BP: <math>\leq 135/85</math> mmHg</li> <li>• Continue antihypertensive treatment unless:               <ol style="list-style-type: none"> <li>1. Systolic BP is consistently <math>&lt;110</math> mmHg</li> <li>2. Diastolic BP is consistently <math>&lt;70</math> mmHg</li> <li>3. There is symptomatic hypotension</li> </ol> </li> </ul>                                                                                   |
|                | Recommended antihypertensive agents       | <ul style="list-style-type: none"> <li>• Labetalol</li> <li>• Nifedipine</li> <li>• Methyldopa</li> </ul>                                                                                                                                                                                                                                                                                                                                                                      |
|                | Contraindicated medications               | <ul style="list-style-type: none"> <li>• ACE inhibitors</li> <li>• Angiotensin receptor antagonists</li> <li>• Diuretics</li> </ul>                                                                                                                                                                                                                                                                                                                                            |
|                | Anaemia management                        | <ul style="list-style-type: none"> <li>• Give parenteral iron if indicated</li> <li>• Give erythropoietin stimulating agents if indicated</li> </ul>                                                                                                                                                                                                                                                                                                                           |
|                | Bone health                               | <ul style="list-style-type: none"> <li>• Give vitamin D supplementation if vitamin D deficient</li> <li>• Discontinue calcimimetics</li> <li>• Discontinue non-calcium-based phosphate binders</li> </ul>                                                                                                                                                                                                                                                                      |

### 9.3.5 Management of UTI in pregnancy

- Can be asymptomatic bacteriuria or symptomatic UTI
- Both have adverse impact on maternal and foetal outcomes
- Screen for and treat UTI in pregnancy
- After a single UTI episode in pregnancy (including asymptomatic bacteriuria), give antibiotic prophylaxis during pregnancy if there is:
  - a. reflux nephropathy
  - b. congenital anomalies of the kidneys and urinary tract (CAKUT)
  - c. women with CKD taking immunosuppression
  - d. women with a history of recurrent UTI

### 9.3.6 Peripartum care

#### A. Inpatient monitoring:

- Take and document observations during any hospital admission: temperature, heart rate, blood pressure, respiratory rate, and oxygen saturation
- Calculate an early warning score and action appropriately
- Beware of increased risk of pulmonary oedema in women with CKD and pre-eclampsia

#### B. Timing of birth determined by:

- Obstetric indications
- Renal factors including:
  - o deteriorating renal function
  - o symptomatic hypoalbuminaemia
  - o pulmonary oedema
  - o refractory hypertension

### 9.3.7 Post-natal care

- Plan early postpartum renal follow up
- Prescribe medications that are compatible with breastfeeding
- No NSAIDs allowed
- Offer safe and effective contraception
- Provide updated pre-pregnancy counselling before future pregnancies

### 9.3.8 Dialysis in pregnancy

#### A. Women receiving maintenance dialysis before pregnancy:

##### i. Give pre-pregnancy counselling:

- Postponing pregnancy until transplantation (when feasible)
- Need for long frequent dialysis prior to and during pregnancy

##### ii. Dialysis dose:

- Give long, frequent haemodialysis either in-centre or at home to improve pregnancy outcomes
- Take into account residual renal function
- Aim for pre-dialysis urea  $\leq 12.5$  mmol/l

##### iii. If on peritoneal dialysis prior to pregnancy, convert to haemodialysis during pregnancy

#### B. When to initiate dialysis during pregnancy :

##### i. Maternal urea concentration $\geq 17$ -20 mmol/L

##### ii. Risks of preterm delivery outweigh those of dialysis initiation

##### iii. Other considerations:

- Gestation
- Renal function trajectory
- Fluid balance
- Biochemical parameters
- Blood pressure
- Uraemic symptoms

# Chapter 10: Dialysis

## 10.1 Introduction

Dialysis is a life-sustaining renal replacement therapy (RRT) used in patients with advanced acute kidney injury (AKI) or chronic kidney disease (CKD) when the kidneys are no longer able to adequately remove metabolic waste products, maintain fluid balance, or regulate electrolytes and acid–base status.

Dialysis may serve as:

- A bridge to kidney recovery in acute kidney injury,
- A long-term therapy in end-stage kidney disease (ESKD), or
- A supportive therapy prior to kidney transplantation.

The two principal forms of dialysis are haemodialysis (HD) and peritoneal dialysis (PD). Each modality has specific indications, infrastructure requirements, cost considerations, and patient-related factors that guide selection. dialysis units.

## 10.2 Acute Dialysis (Acute Kidney Injury)

### 10.2.1 Indications for initiating dialysis

Dialysis should be initiated in patients with severe kidney failure when conservative measures are insufficient to maintain metabolic and fluid stability.

Dialysis is generally recommended when:

- Estimated glomerular filtration rate (eGFR) is approximately 5–10 mL/min/1.73m<sup>2</sup>, or
- eGFR is <15 mL/min/1.73m<sup>2</sup> in the presence of one or more of the following:
  - o Symptoms or signs of uraemia (e.g. gastritis, pericarditis, encephalopathy, uremic frost, persistent nausea and vomiting)
  - o Diuretic-resistant fluid overload
  - o Poorly controlled hypertension
  - o Severe metabolic acidosis
  - o Hyperkalaemia refractory to medical therapy

The decision to initiate dialysis should be individualised and based on clinical judgment rather than laboratory values alone.

### 10.2.2 Modalities of renal replacement therapy in AKI

Several dialysis modalities may be used in the management of acute kidney injury, depending on patient condition, resource availability, and expertise.

These include:

- **Intermittent haemodialysis (IHD)**
- **Sustained low-efficiency dialysis (SLED / SLEDD)**
- **Continuous renal replacement therapy (CRRT)**
  - o Continuous venovenous haemofiltration (CVVH)
  - o Continuous venovenous haemodialysis (CVVHD)
  - o Continuous venovenous haemodiafiltration (CVVHDF)
- **Acute peritoneal dialysis (PD)**

Table 25: Overview of Renal Replacement Therapies

|                            | IHD          | SLED/EDD     | SIUF/SCUF     | C-HF/CVVHF   | C-HD/<br>CVVHD | C-HDF<br>CVVHDF | CCPD                    |
|----------------------------|--------------|--------------|---------------|--------------|----------------|-----------------|-------------------------|
| Duration hr                | 3-4          | 8-12         | 8- Continuous | Continuous   | Continuous     | Continuous      | Continuous              |
| Access                     | Blood access | Blood access | Blood access  | Blood access | Blood access   | Blood access    | Peritoneal access       |
| Solute removal             | Diffusion    | Diffusion    | Convection    | Convection   | Diffusion      | Both            | Diffusion               |
| Blood flow rate mL/min     | 250-400      | 100-200      | 50-100        | 100-300      | 100-200        | 100-300         | —                       |
| Dialysate flow rate mL/min | 500-800      | 100          | 0             | 0            | 9-34           | 9-34            | 2 L every 2 hr exchange |
| Replacement fluid L/day    | 0            | 0            | 0             | 24-96        | 0              | 24-48           | 0                       |
| Fluid removal L/day        | 0-4          | 0-4          | 0-5           | 0-4          | 0-4            | 0-4             | 0-4                     |
| Effluent L/day             | —            | —            | 0-5           | 24-100       | 12-52          | 36-100          | 24-28                   |
| Effluent saturation %      | 15-40        | 60-70        | 100           | 85-100       | 100            | 85-100          | 60-70                   |
| Urea clearance mL/min      | 180-240      | 75-90        | 3.5           | 14-69        | 8-36           | 21-69           | 10-14                   |

Data from Daugirdas JT, Blake PG, Ing TS: Handbook of Dialysis, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2007.

IHD, Intermittent hemodialysis; SLED, slow sustained extended dialysis; EDD, extended daily dialysis; SIUF, slow intermittent ultrafiltration; SCUF, slow continuous ultrafiltration; C-HF, continuous hemofiltration; CVVHF, continuous venovenous hemofiltration; C-HD, continuous hemodialysis; CVVHD, continuous venovenous hemodialysis; C-HDF, continuous hemodiafiltration; CVVHDF, continuous venovenous hemodiafiltration; CCPD, continuous cyclical peritoneal dialysis.

### 10.2.3 Selection of modality

#### Choice of Renal Replacement Therapy is Dependent on:

- Availability of:
  - o HD machine (can do both intermittent HD and SLEDD)
  - o CRRT machine (requires a special machine)
- Availability of experienced personnel
- Availability of dialysis catheters:
  - o HD: temporary catheters required
  - o PD catheter:
    - Temporary PD catheter can be inserted bedside or in theatre
    - Can be modified from urethral catheter or NG tube
- Cost consideration:
  - o CRRT is approximately 10 times more expensive than other HD techniques
- Patient condition:
  - o Haemodynamically unstable patients may tolerate CRRT better than intermittent HD
  - o SLEDD allows gentle dialysis while providing for opportunities in between HD sessions when investigations and procedures can be done

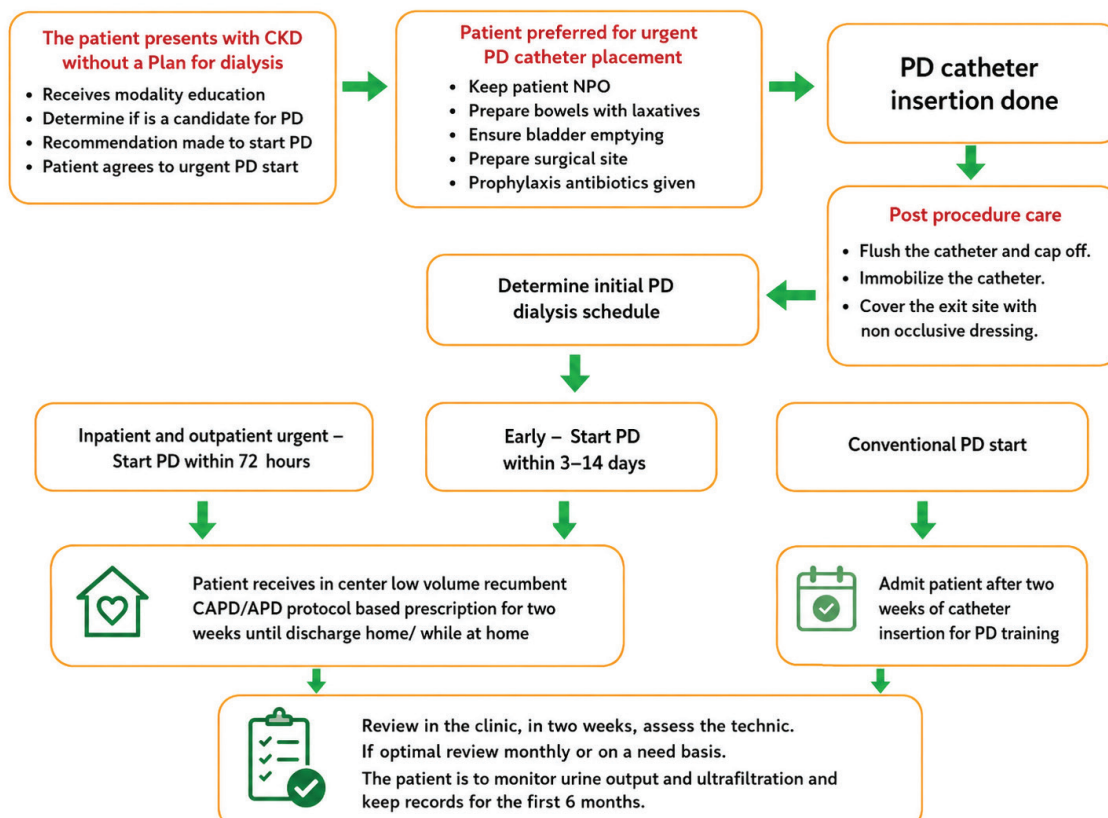


Figure 26: Choice of Renal Replacement Therapies

## 10.3 Acute Haemodialysis

Acute haemodialysis is performed in patients with acute kidney injury who meet indications for renal replacement therapy and require rapid correction of metabolic, electrolyte, or fluid derangements.

### 10.3.1 Pre-dialysis assessment

Before initiating acute haemodialysis:

- Confirm indication for RRT
- Get informed consent:
  - o For temporary HD catheter insertion
  - o For HD procedure
- Get sample for pre-HD serology:
  - o **Hepatitis B:** needs isolation of patient (separate room) and machine, and needs dedicated nurse
  - o **Hepatitis C:** may not need isolation (follow institution's policy)
  - o **HIV:** does not need isolation

### 10.3.2 Vascular access for acute haemodialysis

Temporary central venous catheters are required for acute HD.

#### A. Catheter Selection

- Straight catheters are preferred over J-shaped catheters due to better blood flow rates and longer durability.
- For adults: select internal diameter French size 12–14.
- Catheter length depends on insertion site:
  - o Right internal jugular: 12–15 cm
  - o Left internal jugular: 15–20 cm
  - o Left femoral vein: 20–24 cm
  - o Avoid right femoral vein when possible, to preserve vasculature for future kidney transplantation.
  - o Avoid subclavian vein catheterization due to risk of central venous stenosis.

#### B. Preferred Insertion Sites (in order)

1. Right internal jugular vein
2. Left internal jugular vein
3. Femoral veins
4. Subclavian veins (least preferred)

### 10.3.3 Catheter insertion procedure

- Ultrasound guidance is strongly recommended.
- Continuous telemetry monitoring is helpful.
- Strict aseptic technique must be observed:

- o Procedure may be performed bedside (isolated), in HDU/ICU, or in theatre.
- o All personnel must wear face masks.
- o The operator must:
  - Perform hand hygiene
  - Wear sterile gown, gloves, head cover, and mask
  - Prepare skin with antiseptic solution (chlorhexidine-alcohol preferred to iodine)
  - Use sterile drapes
  - Use sterile-covered ultrasound probe
- After insertion:
  - o Confirm adequate flow from arterial and venous ports
  - o Lock catheter with heparin
  - o Obtain post-procedure chest X-ray for jugular or subclavian placements

### 10.3.4 Acute haemodialysis prescription

- Consult a nephrologist
- Together with the nephrologist, consider:
  - o Goals of treatment
  - o Haemodynamic condition of patient
  - o Fluid status of patient
  - o Fluid needs of patient (e.g. need for blood transfusion)
  - o Electrolytes of patient: serum potassium and sodium
  - o Bleeding risk of patient: to adjust anticoagulant treatment
  - o Possibility of heparin-induced thrombocytopenia
- Elements of HD prescription:
  - In consultation with a nephrologist decide on dialysis modality and prescription as per chart above, with specific emphasis on:
    - o Blood flow rate
    - o Dialysate flow rate
    - o Dialyzer (membrane) size
    - o Duration on dialysis

If haemodynamically stable Can do either intermittent HD or SLEDD

If haemodynamically unstable Do SLEDD (readily available, cheaper) or CRRT

Table 26: Criteria for Stopping RRT in AKI

| Criteria                                 | Details                       |
|------------------------------------------|-------------------------------|
| eGFR                                     | ≥15 mL/min/1.73m <sup>2</sup> |
| Urine output improves (without diuretic) | >500 mL/24h                   |
| Urine output improves (with diuretic)    | >1000 mL/24h                  |

## 10.4 Acute Peritoneal Dialysis

Acute peritoneal dialysis (PD) is an alternative modality of renal replacement therapy in acute kidney injury, particularly in settings where haemodialysis or CRRT is unavailable, contraindicated, or not tolerated. It may also be preferred in haemodynamically unstable patients or in resource-limited settings.

### 10.4.1 Indications for acute peritoneal dialysis

Acute PD may be considered when:

- Haemodialysis or CRRT is not available
- Vascular access is difficult or contraindicated
- The patient is haemodynamically unstable
- There is a need for gradual solute and fluid removal
- In selected paediatric patients

### 10.4.2 Acute pd prescription (adapted from ISPD guidelines)

Acute PD dosing should be guided by the International Society for Peritoneal Dialysis (ISPD) recommendations.

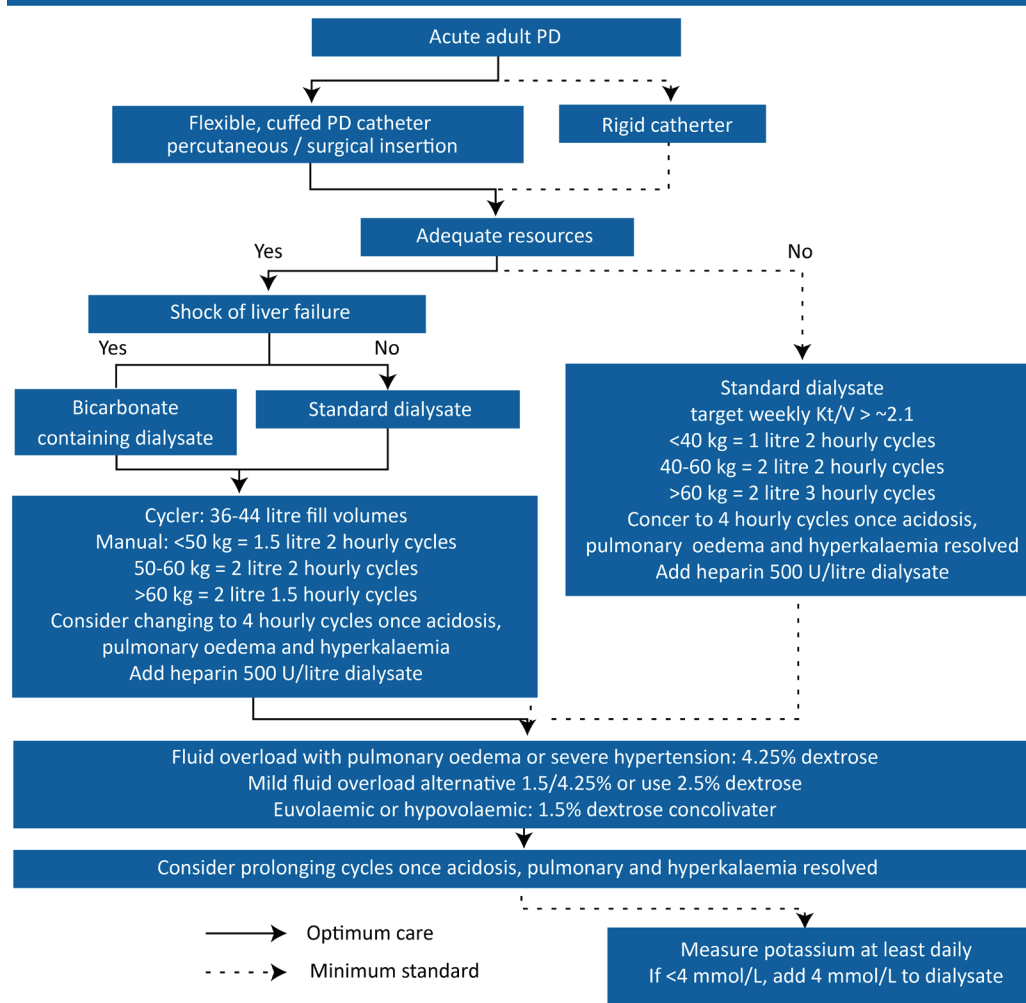
Key elements of prescription include:

- Dialysate fill volume
- Dwell time
- Number of exchanges per day
- Total daily dialysate volume
- Glucose concentration selection based on fluid status

The dose of dialysis should aim to achieve adequate solute clearance and fluid removal while minimizing

## Prescription of acute PD in adult patients

Suggested dosing algorithm



Acute PD dosing guidelines adapted from ISPD guidelines REF Cullis B, Al-Hwiesh A, Kilonzo K, McCulloch M, Niang A, Nourse P, Parapiboon W, Ponce D, Finkelstein FO. ISPD guidelines for peritoneal dialysis in acute kidney injury: 2020 update (adults). *Perit Dial Int.* 2021 Jan;41(1):15-31. doi: 10.1177/0896860820970834. Epub 2020 Dec 3. PMID: 33267747.

Figure 27: Acute PD Dosing Guidelines (Adapted from ISPD 2020)

### 10.4.3 Use of improvised fluids and catheters

In resource-constrained settings, improvised PD solutions and temporary catheters may be used when standard PD fluids are unavailable.

Temporary PD catheters:

- May be inserted bedside or in theatre
- Can be modified from urethral catheters or nasogastric tubes (where appropriate and sterile technique maintained)

When preparing improvised PD fluids:

- Verify the concentration of IV solutions before mixing, as these may vary by country
- Always maintain strict sterile technique

- Minimize the number of components added to reduce infection risk
- Avoid mixing bicarbonate and calcium-containing solutions due to precipitation

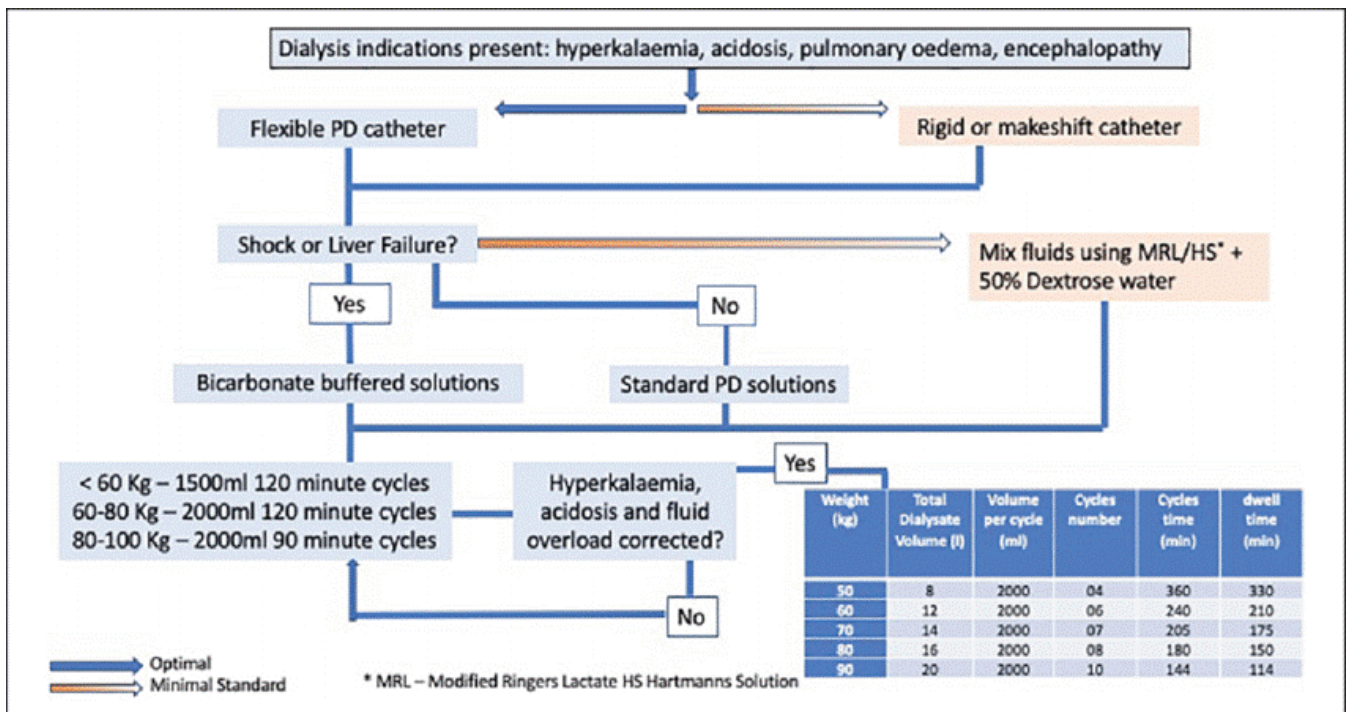


Figure 28: Preparation of Improved PD Fluids

### 10.4.4 Monitoring during acute PD

Patients on acute PD should be monitored for:

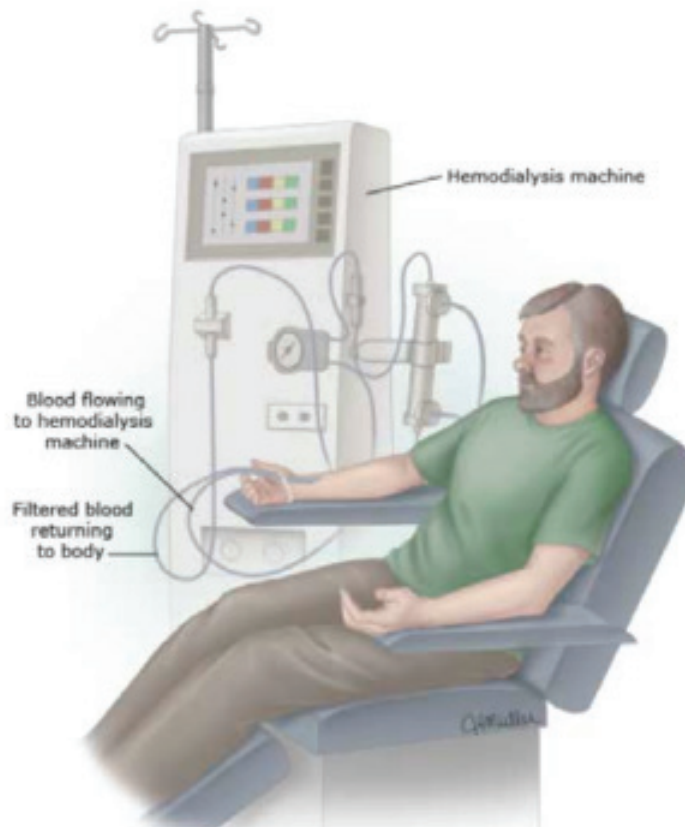
- Ultrafiltration volume
- Electrolytes
- Acid-base status
- Signs of peritonitis
- Catheter function

The decision to continue or discontinue PD should be guided by clinical response and recovery of renal function.

## 10.5 Maintenance (Chronic) Haemodialysis

### 10.5.1 Maintenance (chronic) haemodialysis

- Haemodialysis is the treatment for kidney failure in which a dialyzer (artificial kidney) is used to filter and remove waste products and excess fluid from the blood when the kidneys are unable to do so adequately.
- Blood flows from the body through a vascular access into the dialysis machine, where it passes through the dialyzer. After filtration, the cleaned blood is returned to the body.
- Vascular access may be through an arteriovenous fistula (AVF), arteriovenous graft (AVG), or a central venous catheter.
- Haemodialysis for chronic kidney disease is typically performed three times per week, with each session lasting approximately 4–5 hours, although schedules may be individualised.
- Dialysis must be conducted in government-accredited dialysis centers.



*This image shows a person getting haemodialysis. Blood flows from the body to the haemodialysis machine, where it is filtered. Then, the blood is returned to the body. This can be done through an “access” in the person’s arm, as shown here. It can also be done through a “central venous catheter,” which usually goes into the neck.*

## 10.5.2 Dialysis prescription

Dialysis is a medical prescription and must be individualised for each patient. It should be reviewed regularly by the attending nephrologist.

### A. General Principles

- A dialysis-trained nurse or technologist must perform procedures according to the nephrologist's prescription.
- Dialysis prescription should be adjusted based on:
  - o Blood pressure control
  - o Fluid balance
  - o Residual renal function
  - o Dialysis-related symptoms
  - o Age and weight
  - o Vascular access efficiency
  - o Comorbid conditions (cardiac, pulmonary, neurological, hepatic)
- Medication timing and dose adjustments should also be reviewed in relation to dialysis sessions.

### B. Elements of a Dialysis Prescription

The prescription should specify:

- Mode of dialysis (conventional, convective, or hybrid techniques)
- Frequency per week
- Duration of each session
- Dialyzer type:
  - o Efficiency (low or high)
  - o Permeability (low-flux or high-flux)
  - o Size (1.0–2.1 m<sup>2</sup>)
- Vascular access type
- Blood pump flow rate
- Dialysate flow rate
- Target dry weight
- Ultrafiltration target volume and rate
- Anticoagulation type and dose
- ESA/EPO and iron therapy

Additional parameters may include:

- Dialysate composition (sodium, potassium, calcium, bicarbonate, glucose)
- Dialysate temperature
- Sodium modelling
- Ultrafiltration modelling
- Prescribed dialysis dose (Kt/V)
- Management of haemodynamic instability
- Management of dialysis-related symptoms

### C. Dose of Dialysis and Adequacy

Adequate dialysis is essential for symptom control, metabolic stability, and improved survival.

#### i. Measurement of dose

Dialysis dose may be assessed using:

- o Urea kinetic modelling (Kt/V)
- o Online Kt/V
- o Urea reduction ratio (URR)

The dialysis team should routinely monitor delivered dose.

#### ii. Recommended targets

- o Three sessions per week
- o Total 12–15 hours per week
- o Mid-week Kt/V  $\geq 1.2$  per session
- OR
- o URR  $\geq 65\%$  per session
- Twice-weekly schedules are generally insufficient unless there is significant residual renal function.
- Clinical signs and symptoms must also be considered when assessing adequacy.

## 10.5.3 Haemodialysis membranes

- Dialyzer membranes that minimize complement and leukocyte activation should be used.
- Synthetic polymer hollow-fibre dialyzers are recommended.

## 10.5.4 Anticoagulation during haemodialysis

During HD, blood contact with extracorporeal surfaces increases thrombogenicity.

#### i. Standard Anticoagulation

Unfractionated heparin is recommended:

- Initial bolus: 50 units/kg (typically 1000–5000 units)
- Followed by:
  - o Continuous infusion 1000–1500 units/hour
- OR
- o Intermittent boluses during dialysis

#### ii. Options for Patients at Risk of Bleeding

- Heparin-free dialysis
- Low-dose heparin
- Regional citrate anticoagulation
- Regional anticoagulation with protamine reversal
- Heparin-coated dialyzers

## 10.5.5 Vascular access for haemodialysis

Reliable vascular access is essential for effective and safe haemodialysis. The choice of access influences adequacy of dialysis, infection risk, and long-term patient outcomes.

### Hierarchy of Vascular Access (Preferred Order):

A. Arteriovenous fistula (AVF)

B. Arteriovenous graft (AVG)

C. Central venous catheter (CVC)

Permanent access (AVF or AVG) should be prioritized whenever possible.

### A. Arteriovenous Fistula (AVF) and Arteriovenous Graft (AVG)

| Category                               | Key Recommendations/Details                                                                                                                                                                                                                                                                                                                                                                                 |
|----------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>First Choice of Access</b>          | <p>AVF is the preferred vascular access due to:</p> <ul style="list-style-type: none"> <li>• Superior long-term patency</li> <li>• Improved blood flow over time</li> <li>• Lower rates of infection</li> <li>• Lower incidence of stenosis and thrombosis</li> <li>• Reduced risk of vascular steal syndrome compared to catheters</li> </ul>                                                              |
| <b>Timing of Access Creation</b>       | <ul style="list-style-type: none"> <li>• Access should be created when eGFR falls below 20 mL/min/1.73m<sup>2</sup> to allow adequate maturation.</li> <li>• AVF typically requires 6–8 weeks to mature before cannulation.</li> </ul>                                                                                                                                                                      |
| <b>Pre-operative Evaluation</b>        | <ul style="list-style-type: none"> <li>• Vascular mapping using physical examination and Doppler ultrasound is recommended.</li> <li>• Prefer placement in the non-dominant arm.</li> <li>• Placement sequence: wrist (radiocephalic) → elbow (brachiocephalic).</li> </ul> <p>If AVF is not feasible, an AVG using synthetic material may be established.</p>                                              |
| <b>Use of AVF</b>                      | <ul style="list-style-type: none"> <li>• Do not cannulate before 6–8 weeks.</li> <li>• Initial cannulation should be performed by experienced personnel.</li> <li>• Begin with 17-gauge needles and increase size as fistula matures.</li> </ul>                                                                                                                                                            |
| <b>Rule of 6 (Maturation Criteria)</b> | <ul style="list-style-type: none"> <li>• Flow &gt; 600 mL/min</li> <li>• Diameter &gt; 6 mm</li> <li>• Depth &lt; 6 mm below skin</li> <li>• Length &gt; 6 cm for cannulation</li> </ul> <p>Maturation period &gt; 6 weeks</p>                                                                                                                                                                              |
| <b>Care of AVF/AVG</b>                 | <ul style="list-style-type: none"> <li>• Maintain good hygiene.</li> <li>• Monitor regularly by physical examination.</li> <li>• Educate patients to recognize: <ul style="list-style-type: none"> <li>o Signs of infection</li> <li>o Change in thrill</li> </ul> </li> <li>• Avoid heavy lifting or compression of the access arm.</li> <li>• Use strict aseptic technique during cannulation.</li> </ul> |
| <b>Surveillance for Stenosis</b>       | <ul style="list-style-type: none"> <li>• Monitor static dialysis venous pressure</li> <li>• Perform duplex ultrasound when indicated</li> <li>• Assess for recirculation</li> </ul>                                                                                                                                                                                                                         |

## B. Central Venous Catheters (CVCs)

Central venous catheters are temporary access options and should not be used long-term unless AVF/AVG creation is not possible.

### Indications

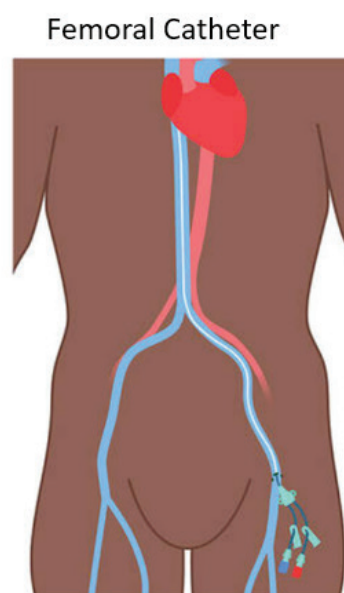
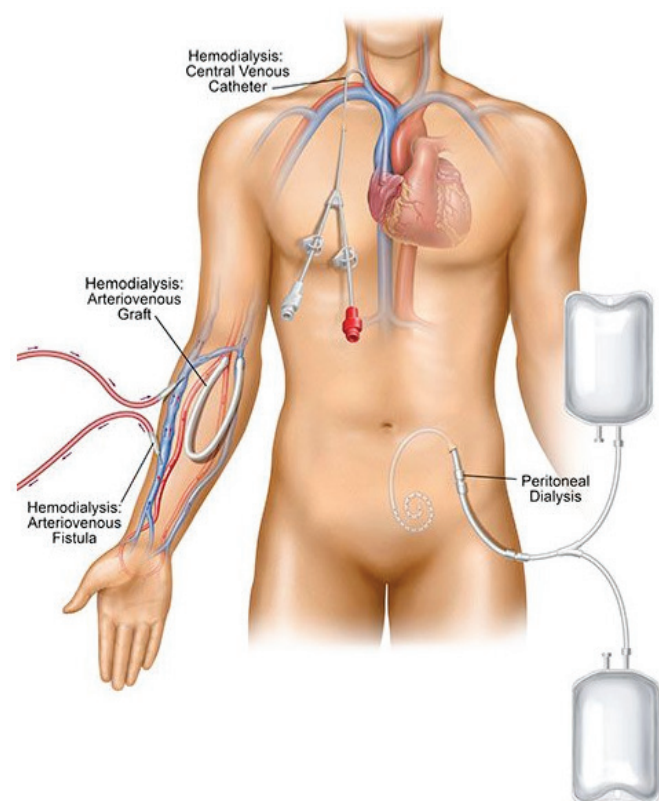
- Bridge while AVF matures
- Emergency dialysis
- Patients unsuitable for permanent access

### Preferred sites (in order)

- Right internal jugular (12–15 cm)
- Left internal jugular (15–20 cm)
- Left femoral vein (20–24 cm)

### Avoid

- Subclavian vein (risk of stenosis)
- Right femoral vein (preserve for transplantation)



Ref: <https://www.azuravascularcare.com>

Figure 29: Vascular Access Options in Haemodialysis

## 10.5.6 Choice of haemodialysis catheters

Catheter selection should consider patient condition, BMI, need for mobility, and procedural expertise.

Table 27: Types of Haemodialysis Catheters and their Complications

| Access                        | Choice                                                                                                                                                                                                                                                                                                                                              | Complications                                                                                                                                                                                                                                                     |
|-------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Right internal jugular</b> | <ul style="list-style-type: none"> <li>Critically ill and bed-bound with body mass index &gt;28</li> <li>aortic aneurysm repair</li> <li>Ambulatory patient/mobility required for rehabilitation</li> </ul>                                                                                                                                         | <ul style="list-style-type: none"> <li>Arterial puncture</li> <li>Pneumothorax</li> <li>Hemothorax</li> <li>Arrhythmias</li> <li>Air embolism</li> <li>Perforation of cardiac chambers</li> <li>Pericardial tamponade</li> <li>Central venous stenosis</li> </ul> |
| <b>Femoral</b>                | <ul style="list-style-type: none"> <li>Critically ill and bed-bound with body mass index &lt;24</li> <li>Tracheostomy present or planned in near-term</li> <li>Need for long-term haemodialysis access present, highly likely or planned</li> <li>Emergency dialysis required plus inexperienced operator and/or no access to ultrasound</li> </ul> | <ul style="list-style-type: none"> <li>Retroperitoneal haemorrhage</li> <li>Arterial puncture</li> <li>Air embolism</li> </ul>                                                                                                                                    |
| <b>Left internal jugular</b>  | <ul style="list-style-type: none"> <li>Contraindications to right internal jugular and femoral sites</li> </ul>                                                                                                                                                                                                                                     | <ul style="list-style-type: none"> <li>Arterial puncture</li> <li>Pneumothorax</li> <li>Hemothorax</li> <li>Arrhythmias</li> <li>Air embolism</li> <li>Perforation of cardiac chambers</li> <li>Pericardial tamponade</li> <li>Central venous stenosis</li> </ul> |
| <b>Subclavian</b>             | <ul style="list-style-type: none"> <li>Contraindications to internal jugular</li> <li>Right side to be used preferentially</li> </ul>                                                                                                                                                                                                               | <ul style="list-style-type: none"> <li>Arterial puncture</li> <li>Pneumothorax</li> <li>Hemothorax</li> <li>Arrhythmias</li> <li>Air embolism</li> <li>Perforation of cardiac chambers</li> <li>Pericardial tamponade</li> <li>Central venous stenosis</li> </ul> |

## 10.5.7 Infection prevention and vascular access care

Infection is a major cause of morbidity and mortality in haemodialysis patients, particularly among those using central venous catheters. Strict infection prevention practices must be implemented in all dialysis units.

### A. General Infection Prevention Measures

- Dialysis units should implement structured infection prevention programs that include:
- Monthly surveillance for bloodstream infections (BSIs) and other dialysis-related infections
- Routine hand hygiene audits, with results shared with staff
- Regular observation of catheter and vascular access care practices
- Competency-based training and re-evaluation of staff every 6–12 months
- Standardized patient education on:
  - o Hand hygiene
  - o Access care
  - o Recognizing signs of infection
  - o Risks associated with catheter use

Efforts should be made to reduce catheter use by facilitating timely AVF/AVG creation.

### B. Skin Antisepsis and Catheter Hub Care

- Use alcohol-based chlorhexidine (>0.5%) as the first-line antiseptic for:
  - o Central line insertion
  - o Dressing changes
- Povidone-iodine (preferably with alcohol) or 70% alcohol may be used in patients with chlorhexidine intolerance.

Catheter hub disinfection must be performed:

- Every time the catheter is accessed
- After cap removal
- Before connection and after disconnection
- Antimicrobial ointment (e.g., bacitracin, polymyxin B, povidone-iodine) may be applied at catheter exit sites during dressing changes.

### C. Haemodialysis Catheter Connection Procedure

To reduce infection risk:

- Perform hand hygiene and wear clean gloves.
- Clamp catheter before removing caps.
- Disinfect catheter hubs using appropriate antiseptic.
- Scrub hub threads and end thoroughly with friction.
- Allow antiseptic to dry before connection.
- Avoid leaving catheter hubs open longer than necessary.

#### D. Haemodialysis Catheter Disconnection Procedure

- Perform hand hygiene and wear clean gloves.
- Clamp the catheter before disconnecting.
- Disinfect the hub prior to applying the new cap.
- Use sterile caps.
- Ensure the catheter remains clamped after disconnection.
- Remove gloves and perform hand hygiene.

Strict aseptic handling of catheter hubs is mandatory.

#### E. Catheter-Related Infections

- Types
- Exit-site infection
- Tunnel infection
- Catheter-related bloodstream infection (CRBSI)

Management depends on clinical presentation and the organism involved.

Table 28: Central Venous Haemodialysis Catheter Infections and their Management

| Type of infection      | Signs and symptoms                                                                                            | Treatment                                                                                                                                                                                                                                                                                                                                                                                                     |
|------------------------|---------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Tunnel track infection | Fever, chills, hemodynamic instability, CVC dysfunction, hypothermia, nausea and vomiting and general malaise | If drainage is present culture before initiation of antibiotic<br><br>Empiric antibiotic to cover gram-positive organisms for 7-14 days                                                                                                                                                                                                                                                                       |
| Exit site infection    |                                                                                                               | Treatment is according to culture from the exit site drainage<br><br>Empiric antibiotic to target gram-positive and negative bacteria for 10-14 days                                                                                                                                                                                                                                                          |
| Bacteraemia (CRBSI)    |                                                                                                               | CRBSI should have blood cultures obtained from the CVC and peripheral source (dialysis circuit ± peripheral veins)<br><br>Broad-spectrum antibiotics that treat both Gram-positive and Gram-negative organisms should be initiated immediately<br><br>Due to the high prevalence of methicillin-resistant <i>S aureus</i> , empiric therapy should include coverage for methicillin-resistant <i>S aureus</i> |

Adapted from KDOQI guidelines (2019)

## Central Venous Stenosis

Empiric therapy should cover:

- Gram-positive organisms (including MRSA)
- Gram-negative organisms

CVC removal may be required in cases of:

- Persistent bacteraemia (48–72 hours)
- Hemodynamic instability
- Metastatic infection
- Tunnel infection
- Infection due to *Staphylococcus aureus*, *Pseudomonas*, fungi, or mycobacteria

## F. Central venous stenosis may occur following catheter use

Clinical features may include:

- Swelling of face or upper limb
- Prominent collateral veins
- Dysfunction of vascular access

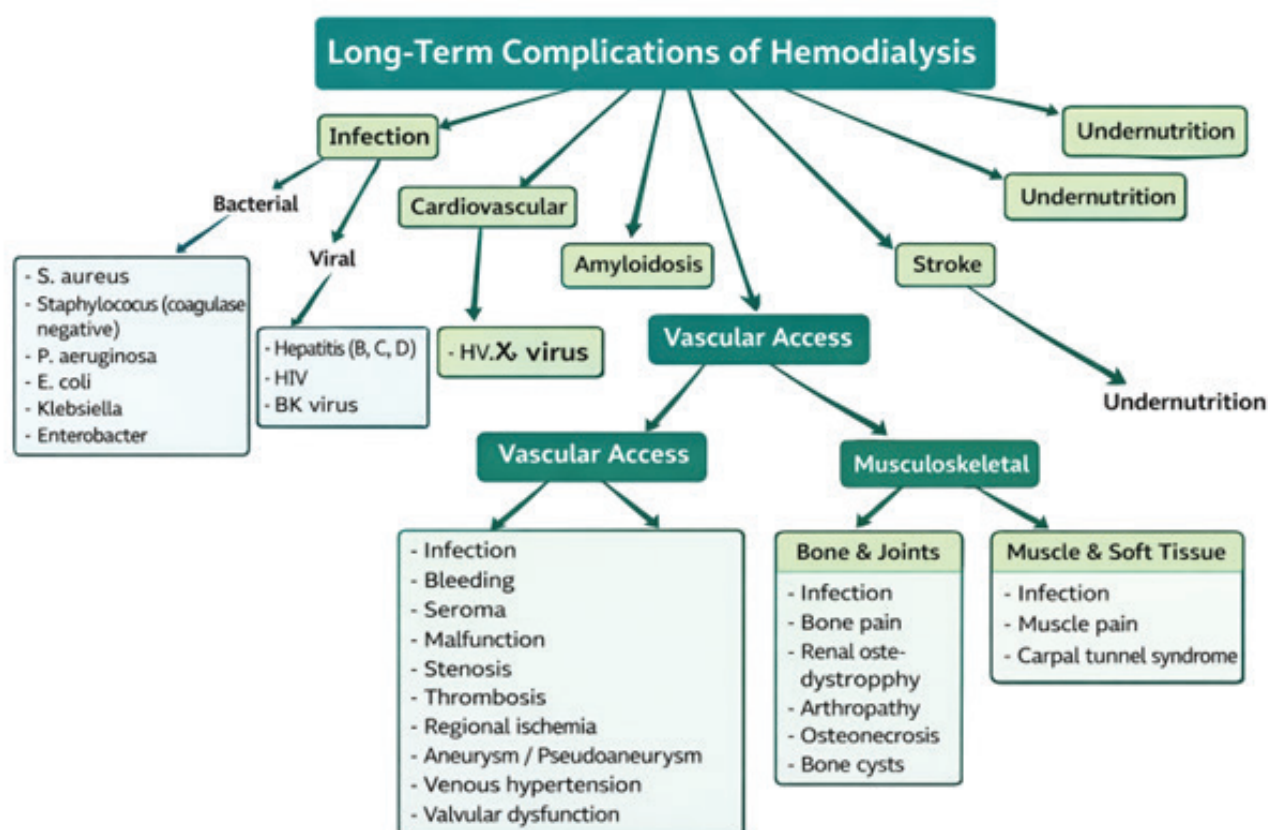
Table 29: Clinical Features of Central Venous Stenosis

| Timing of Occurrence     | Signs and Symptoms/ Comments                                                                                                                                         |
|--------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Early signs and symptoms |                                                                                                                                                                      |
| Swelling                 | Typically asymmetric, affecting the hands and arms                                                                                                                   |
| Pain                     | Pain that can be attributed to central venous stenosis/occlusion, such as aching and heaviness of an extremity, when other causes have been excluded                 |
| Cutaneous findings       | Venous collaterals; skin discoloration (red, purple, or blue discoloration)                                                                                          |
| Late signs and symptoms  |                                                                                                                                                                      |
| Swelling                 | More widespread, affecting the arms, head, neck, or trunk (including breasts).<br>Swelling may be unilateral, bilateral, or involve only the head, face, and neck    |
| Pain                     | Persistent pain attributed to central venous stenosis/occlusion (e.g., persistent aching, chest or extremity heaviness)                                              |
| Cutaneous findings       | Venous collaterals; skin discoloration (red, purple, or blue discoloration; chronic pigmentation changes); lymphatic blistering or weeping; stasis ulcers; phlebitis |
| Respiratory compromise   | Hoarse voice and/or respiratory distress from laryngeal oedema, pleural effusion, or chest (or breast) swelling that can cause restrictive pulmonary compromise      |
| Neurologic symptoms      | Visual or auditory disturbances, exophthalmos, cognitive disabilities, headaches, or seizures when other causes have been excluded                                   |

## 10.5.8 Complications of haemodialysis

Complications of haemodialysis may be classified as acute (occurring during or shortly after dialysis sessions) or chronic (long-term consequences of dialysis and kidney failure).

| Acute complications                                                                                                                                                                                                                                                                                                                                | Chronic complications                                                                                                                                                                                                                                             |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> <li>• Hypotension</li> <li>• Hypertension</li> <li>• Muscle cramps</li> <li>• Dialysis disequilibrium syndrome</li> <li>• Hypoglycaemia</li> <li>• Bleeding</li> <li>• Acute febrile reactions</li> <li>• Dialysis-related allergic reactions</li> <li>• Convulsions</li> <li>• Sudden cardiac death</li> </ul> | <ul style="list-style-type: none"> <li>• Vascular access failure</li> <li>• Central venous stenosis</li> <li>• Anaemia</li> <li>• Chronic inflammation</li> <li>• Malnutrition</li> <li>• Mineral and bone disorders</li> <li>• Cardiovascular disease</li> </ul> |



Adapted from Habas, et al

Management requires multidisciplinary follow-up and regular reassessment.

Figure 30: Long-Term Complications of Haemodialysis

### 10.5.9 Laboratory monitoring in haemodialysis patients

Regular laboratory monitoring is essential to guide dialysis adequacy, anaemia management, and metabolic control.

| Monthly tests:                                                                                                                                                                                     | 3 monthly tests                                                                                                                                  | 6 monthly tests                                                                                                                               |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Complete blood count</li><li>• Sodium, Potassium, Urea, Creatinine</li><li>• Phosphorous, Calcium, Alkaline Phosphatase</li><li>• Albumin levels</li></ul> | <p>Serology:</p> <ul style="list-style-type: none"><li>• HIV</li><li>• Hepatitis B surface antigen (HBsAg)</li><li>• Hepatitis C virus</li></ul> | <ul style="list-style-type: none"><li>• Parathyroid hormone (PTH), Vitamin D</li><li>• Serum ferritin, serum transferrin saturation</li></ul> |

### 10.5.10 Vaccination in CKD and Haemodialysis Patients

Vaccination should follow national immunization guidelines and may include:

- Hepatitis B
- Influenza
- Pneumococcal vaccination

Table 30: Vaccination Schedule for Stage 1–5 CKD Patients

| Vaccine                                              | Schedule / Other Notes                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           |
|------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Influenza                                            | <ul style="list-style-type: none"> <li>Annual recombinant or inactivated vaccine.</li> <li>Live attenuated vaccine is contraindicated.</li> <li>Intramuscular administration annually.</li> </ul>                                                                                                                                                                                                                                                                                                                                |
| Hepatitis B                                          | <ul style="list-style-type: none"> <li>Four-dose schedule (0, 1, 2, and 6 months), 20 µg intramuscular recombinant vaccine.</li> <li>Monitor 1–2 months after vaccination series to assess response, then every 6–12 months thereafter.</li> <li>When titers fall below protective levels (anti-HBs &lt; 10 IU/mL), administer a booster dose.</li> </ul>                                                                                                                                                                        |
| Pneumococcal vaccines PCV13 & PPSV23 (Age 19–64)     | <ul style="list-style-type: none"> <li>If never vaccinated with PCV13 or PPSV23: administer 1 dose of PPSV23 immediately, 1 dose of PCV13 at least 1 year after PPSV23, and a second dose of PPSV23 1 year after PCV13 (first PPSV23 must be ≥ 5 years after the first dose of PPSV23).</li> </ul>                                                                                                                                                                                                                               |
|                                                      | <ul style="list-style-type: none"> <li>If previously vaccinated with 1 dose PPSV23 ≥ 1 year ago: give 1 dose of PCV13 now, then 1 dose of PPSV23 ≥ 8 weeks after PCV13 (must be ≥ 5 years after the first PPSV23).</li> <li>If previously vaccinated with 2 doses PPSV23 and last dose was ≥ 1 year ago: give 1 dose of PCV13 now.</li> <li>If previously vaccinated with 1 dose PCV13 ≥ 8 weeks ago and 1 dose PPSV23: administer 1 dose PPSV23 ≥ 5 years after first PPSV23 dose and 1 dose PPSV23 ≥ 5 years later.</li> </ul> |
| Pneumococcal vaccines PCV13 & PPSV23 (Age ≥ 65)      | <ul style="list-style-type: none"> <li>If never vaccinated with PCV13: administer 1 dose PCV13 now, then 1 dose PPSV23 ≥ 8 weeks after PCV13 (must be ≥ 5 years after the first PPSV23).</li> <li>If previously vaccinated with ≥ 1 dose PCV13 ≥ 8 weeks ago: administer 1 dose PPSV23 now.</li> </ul>                                                                                                                                                                                                                           |
| Yellow fever                                         | <ul style="list-style-type: none"> <li>0.5 mL given subcutaneously.</li> <li>NB: Live vaccines are contraindicated in transplant patients</li> </ul>                                                                                                                                                                                                                                                                                                                                                                             |
| Varicella virus vaccine (Recombinant zoster vaccine) | <ul style="list-style-type: none"> <li>Two doses administered intramuscularly. Second dose after 2–6 months.</li> <li>NB: If expedited vaccination intended (e.g., prior to immunosuppression), second dose can be given after 1–2 months.</li> </ul>                                                                                                                                                                                                                                                                            |

## 10.6 Peritoneal Dialysis (Maintenance)

### 10.6.1 Introduction to peritoneal dialysis

- Peritoneal dialysis (PD) is a renal replacement therapy in which the peritoneal membrane serves as a natural semi-permeable membrane to remove waste products and excess fluid.
- Dialysate is infused into the peritoneal cavity through a peritoneal dialysis catheter, allowing solute and fluid exchange to occur across the peritoneal membrane before drainage.
- PD is primarily a home-based therapy and offers greater flexibility and patient autonomy compared to in-centre haemodialysis.

PD may be delivered as:

- Continuous Ambulatory Peritoneal Dialysis (CAPD)
- Automated Peritoneal Dialysis (APD)

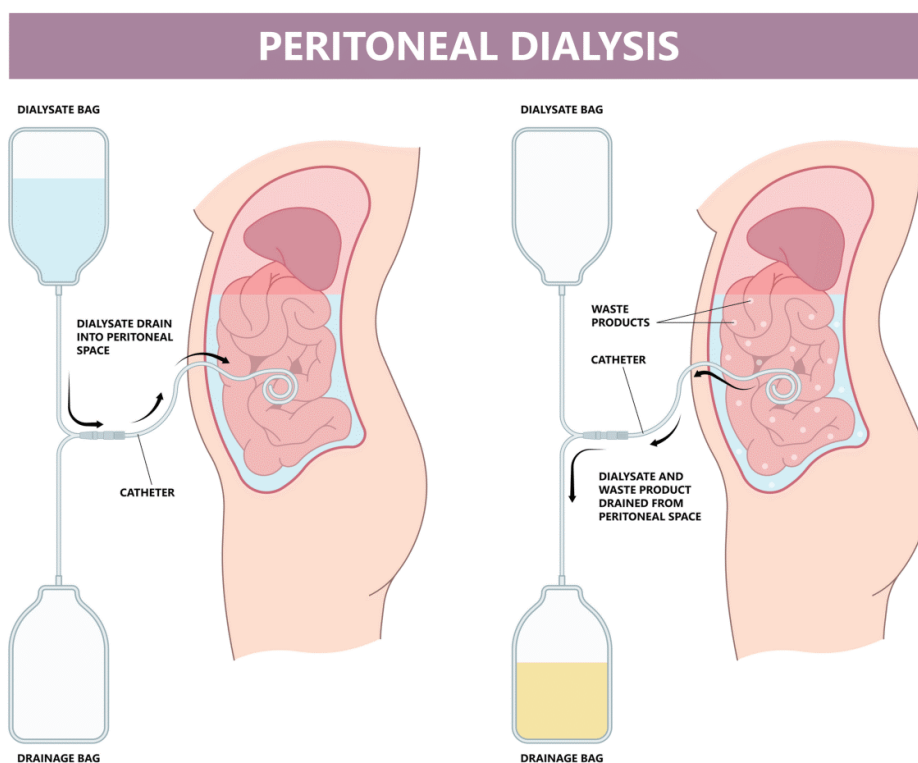


Figure 31: Overview of Peritoneal Dialysis

### 10.6.2 Patient selection for peritoneal dialysis

PD is suitable for patients with:

- Adequate cognitive and physical ability (or caregiver support)
- Intact peritoneal cavity
- No active intra-abdominal infection
- Acceptable home environment for sterile exchanges
- Motivation and capacity to adhere to aseptic technique

Relative contraindications may include:

- Extensive abdominal adhesions
- Active inflammatory bowel disease
- Severe malnutrition
- Recurrent abdominal infections

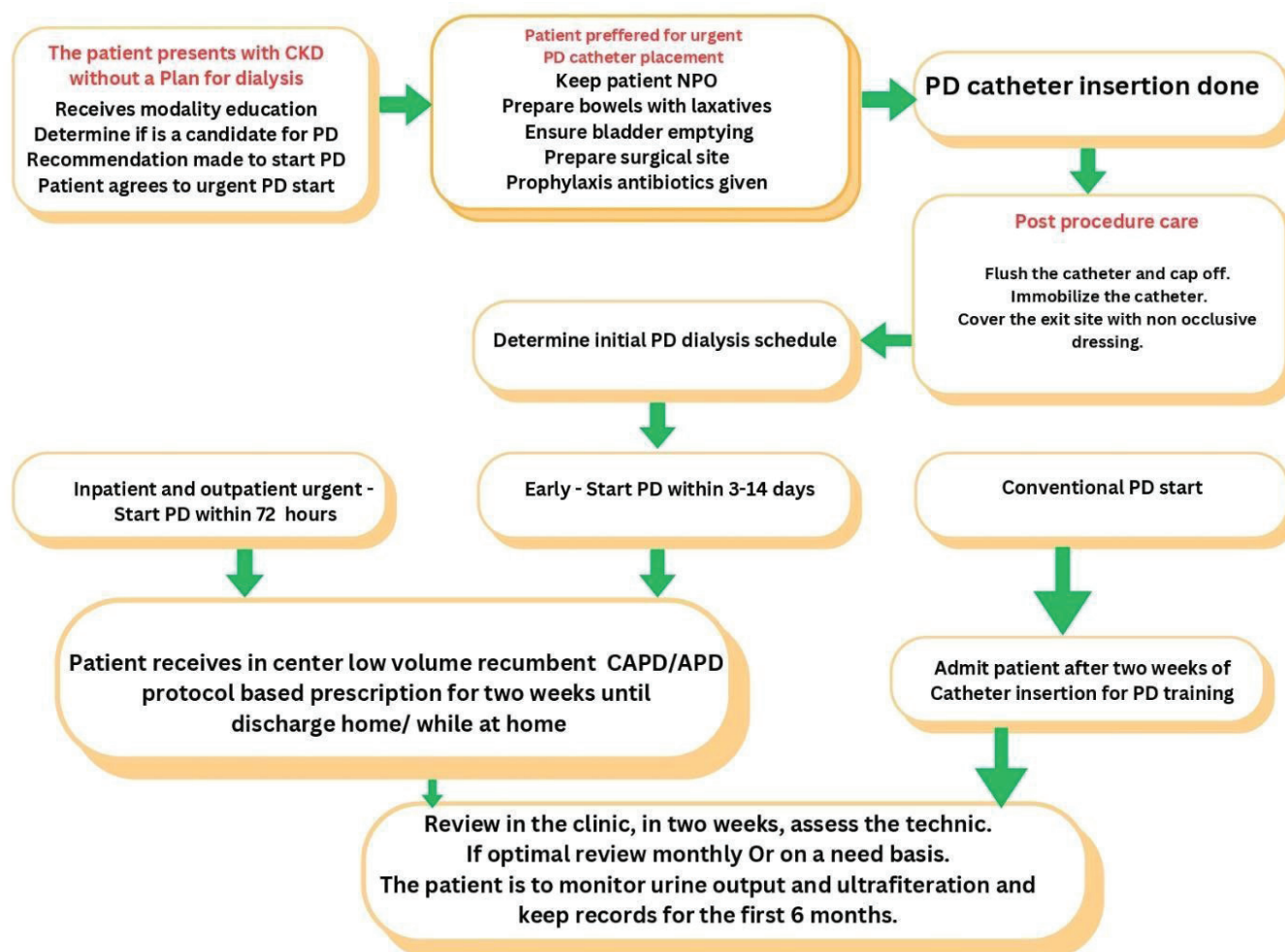


Figure 32: Peritoneal dialysis flowchart

### 10.6.3 Peritoneal dialysis catheter and access

A functioning PD catheter is essential for successful therapy.

#### A. Access Team

Catheter insertion should be performed by trained personnel (surgeon, nephrologist, or interventional radiologist) with appropriate expertise.

#### B. Timing of Insertion

Catheter should ideally be inserted at least 2 weeks prior to initiation of PD to allow healing and reduce leakage risk.

#### C. Pre-Procedure Preparation

- Prophylactic antibiotics should be administered prior to insertion.
- Strict aseptic technique must be followed.

#### D. Post-Insertion Care

- Maintain sterile dressing.
- Avoid early heavy physical activity.
- Monitor for leakage, bleeding, or signs of infection.

### 10.6.4 Peritoneal dialysis prescription

PD prescription should be individualised and reviewed regularly.

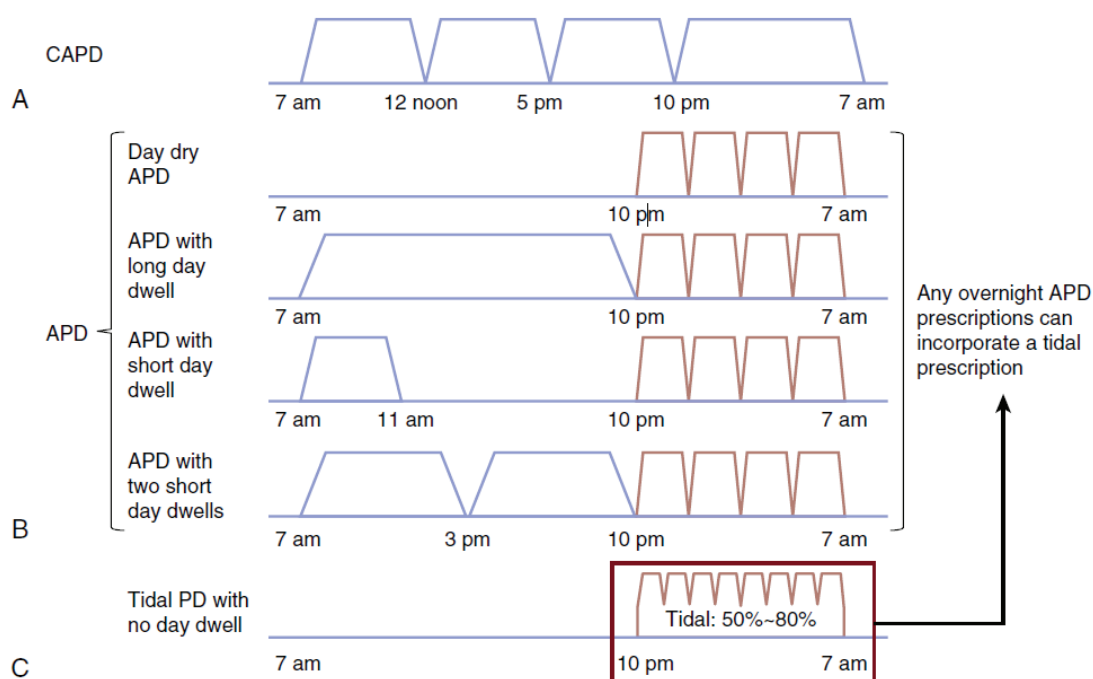
#### A. Components of PD Prescription

- Fill volume
- Dwell time
- Number of exchanges per day
- Dialysate glucose concentration
- Total daily dialysate volume
- Use of automated or manual technique

#### B. Adequacy Targets

- Weekly Kt/V  $\geq 1.7$
- Creatinine clearance  $\geq 50$  L/week/1.73m<sup>2</sup>

Residual renal function should be preserved whenever possible.



Ref: Primer of Kidney Disease

Figure 33: Types of Peritoneal Dialysis Prescriptions

| Typical Incremental Peritoneal Dialysis Prescriptions                                                  |                              |
|--------------------------------------------------------------------------------------------------------|------------------------------|
| CAPD                                                                                                   | APD                          |
| 3 × 2 L daily                                                                                          | APD with no day dwell        |
| 2 × 2 L daily (single or both icodextrin)                                                              | APD five nights a week       |
| 1 × 2 L icodextrin long dwell daily                                                                    | APD three nights a week      |
| 4 × 1.5 L daily                                                                                        | APD with 1.5 L dwell volumes |
| CAPD 4–6 days a week                                                                                   | APD for 6 h each night       |
| PD: peritoneal dialysis; APD, automated peritoneal; CAPD, continuous ambulatory peritoneal dialysis.   |                              |
| From Blake PG, Dong J, Davies SJ. Incremental peritoneal dialysis. Perit Dial Int. 2020;40(3):320–326. |                              |

Figure 34: Incremental prescriptions in peritoneal dialysis

## 10.6.5 PD fluids

PD fluids may contain:

- Glucose (various concentrations)
- Icodextrin
- Amino acid-based solutions

Selection depends on fluid status, nutritional needs, and metabolic profile.

Table 31: Types of Peritoneal Dialysis Fluids

|                           | Dianeal Standard<br>Glucose PD<br>Solutions                                                                                                                                                     | Physioneal                                            | Icodextrin<br>or<br>Extraneal<br>or 7.5%<br>Solution | Amino<br>Acid or<br>Nutrineal<br>or 1.1%<br>Solution | StaySafe Standard<br>Glucose PD Solutions                                                                                                                        | StaySafe Balance                                                                                                                                                 | Bica Vera   |
|---------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------|------------------------------------------------------|------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------|
| Sodium (mmol/L)           | 132                                                                                                                                                                                             | 132                                                   | 133                                                  | 132                                                  | 134                                                                                                                                                              | 134                                                                                                                                                              | 134         |
| Calcium (mmol/L)          |                                                                                                                                                                                                 |                                                       |                                                      |                                                      |                                                                                                                                                                  |                                                                                                                                                                  |             |
| Low                       | 1.25 (PD-2)                                                                                                                                                                                     | 1.25                                                  | —                                                    | 1.25                                                 | 1.25                                                                                                                                                             | 1.25                                                                                                                                                             | —           |
| Standard                  | 1.75 (PD-4)                                                                                                                                                                                     | 1.75                                                  | 1.75                                                 | —                                                    | 1.75                                                                                                                                                             | 1.75                                                                                                                                                             | 1.75        |
| Ultralow                  | 1.00 (PD-1)                                                                                                                                                                                     | —                                                     | —                                                    | —                                                    | —                                                                                                                                                                | —                                                                                                                                                                | —           |
| Magnesium<br>(mmol/L)     | 0.25                                                                                                                                                                                            | 0.25                                                  | 0.25                                                 | 0.25                                                 | 0.5                                                                                                                                                              | 0.5                                                                                                                                                              | 0.5         |
| Chloride (mmol/L)         | 96                                                                                                                                                                                              | 95 (Physioneal-40)<br>101 (Physioneal-35)             | 96                                                   | 105                                                  | 103.5                                                                                                                                                            | 100.5 for 1.25 mmol Ca<br>101.5 for 1.75 mmol Ca                                                                                                                 | 104.5       |
| Lactate (mmol/L)          | 40                                                                                                                                                                                              | 15 (Physioneal-40)<br>10 (Physioneal-35)              | 40                                                   | 40                                                   | 35                                                                                                                                                               | 2                                                                                                                                                                | 0           |
| Bicarbonate<br>(mmol/L)   | —                                                                                                                                                                                               | 25                                                    | 0                                                    | 0                                                    | 0                                                                                                                                                                | 35                                                                                                                                                               | 35          |
| Glucose (mmol/L)          | 1.36%, 76<br>2.27%, 126<br>3.86%, 214                                                                                                                                                           | 1.36%, 75.5<br>2.27%, 126<br>3.86%, 214               | 0                                                    | 0                                                    | 1.5%, 83.2<br>2.3%, 126.1<br>4.25%, 235.8                                                                                                                        | 1.5%, 83.2<br>2.3%, 126.1<br>4.25%, 235.8                                                                                                                        | 1.5%, 83.25 |
| Icodextrin (g/L)          | 0                                                                                                                                                                                               | 0                                                     | 75                                                   | 0                                                    | 0                                                                                                                                                                | 0                                                                                                                                                                | 0           |
| Amino acids<br>(mmol/L)   | 0                                                                                                                                                                                               | 0                                                     | 0                                                    | 87*                                                  | 0                                                                                                                                                                | 0                                                                                                                                                                | 0           |
| Overall pH                | 5.5                                                                                                                                                                                             | 7.4                                                   | 5.2                                                  | 6.6                                                  | 7                                                                                                                                                                | 7                                                                                                                                                                | 7.4         |
| Approximate<br>osmolality | PD-2<br>1.36%, 346 mOsm<br>2.27%, 396 mOsm<br>3.86%, 485 mOsm<br>PD-4<br>1.36%, 345 mOsm<br>2.27%, 395 mOsm<br>3.86%, 483 mOsm<br>PD-1<br>1.36%, 344 mOsm<br>2.27%, 394 mOsm<br>3.86%, 484 mOsm | 1.36%, 344 mOsm<br>2.27%, 395 mOsm<br>3.86%, 483 mOsm | 284 mOsm                                             | 365 mOsm                                             | 1.25 mmol/L Ca solution<br>1.5%, 356 mOsm<br>2.3%, 399 mOsm<br>4.25%, 509 mOsm<br>1.75 mmol/L Ca solution<br>1.5%, 358 mOsm<br>2.3%, 401 mOsm<br>4.25%, 511 mOsm | 1.25 mmol/L Ca solution<br>1.5%, 356 mOsm<br>2.3%, 399 mOsm<br>4.25%, 509 mOsm<br>1.75 mmol/L Ca solution<br>1.5%, 358 mOsm<br>2.3%, 401 mOsm<br>4.25%, 511 mOsm | 358 mOsm    |

\*The 1.1% solutions consist of a combination of amino acids, including histidine, valine, isoleucine, alanine, leucine, arginine, lysine, glycine, methionine, proline, phenylalanine, serine, threonine, tyrosine, and tryptophan.  
(From Himmelfarb J, Izzler TA: Chronic Kidney Disease, Dialysis, and Transplantation, A Companion Guide to Brenner and Rector's the Kidney, ed 4, Elsevier, 2019.)

## 10.6.6 Monitoring in Peritoneal Dialysis

Patients on PD should be monitored for:

- Ultrafiltration volume
- Weight trends
- Blood pressure
- Electrolytes
- Residual renal function
- Signs of peritonitis
- Exit-site condition

Peritoneal Equilibration Test (PET) may be used to classify membrane transport status.

## 10.6.7 PD-Related Infections

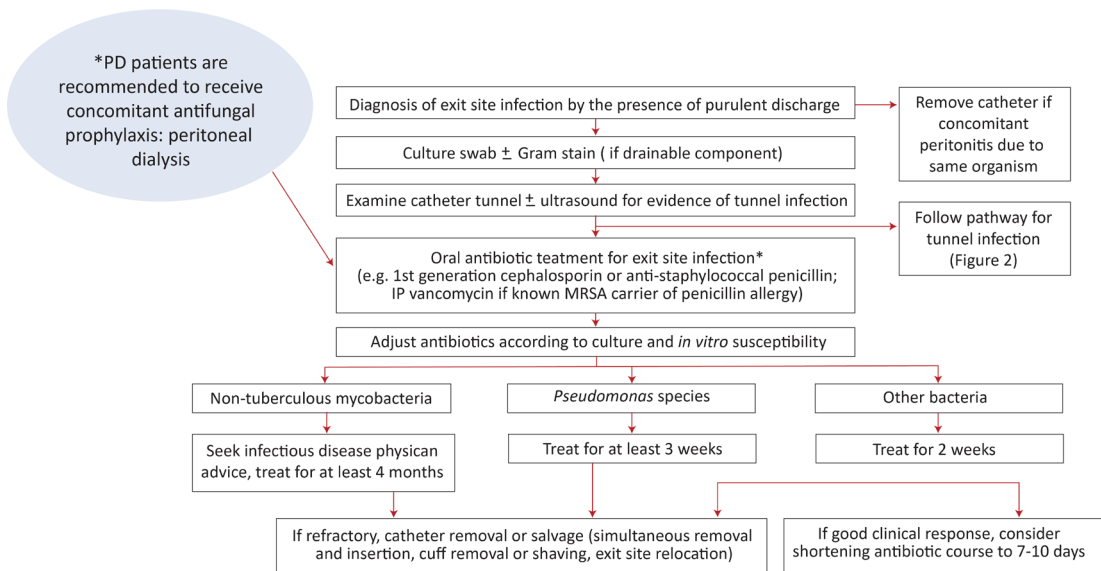
Infection remains the most significant complication of PD.

### A. Exit-Site and Tunnel Infections

Clinical features:

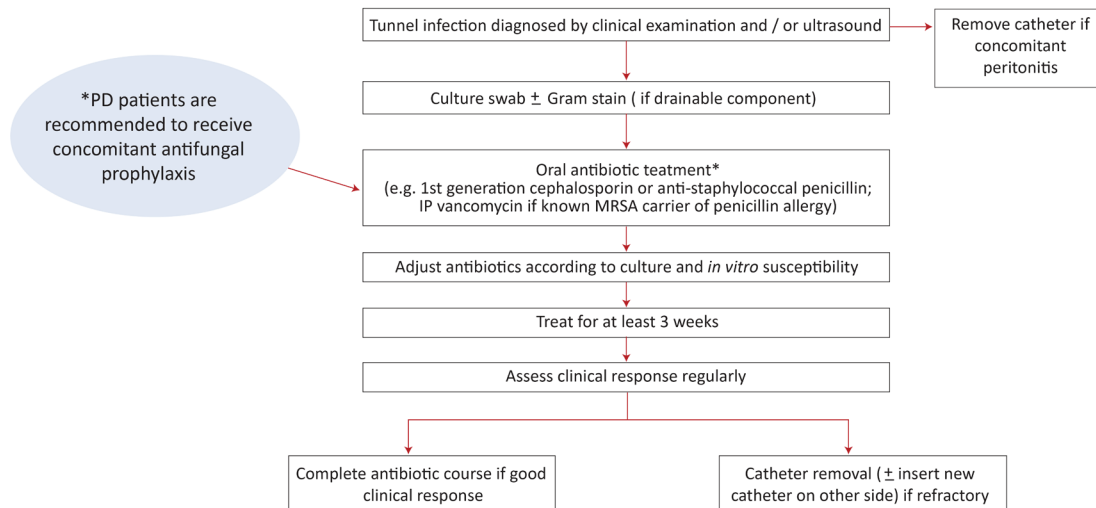
- Redness
- Pain
- Discharge

Management includes appropriate antibiotic therapy based on culture results.



Adopted from ISPD guidelines 2022

Figure 35: Management of PD exit site infections



Adopted from ISPD guidelines 2022

Figure 36: Management of PD catheter tunnel infections

## B. Peritonitis

Diagnosis requires at least two of the following:

- Abdominal pain and/or cloudy effluent
- Dialysate white cell count >100 cells/mL (with >50% polymorphonuclear cells)
- Positive dialysate culture

Treatment:

- Initiate broad-spectrum intraperitoneal antibiotics promptly
- Modify based on culture and sensitivity
- Consider catheter removal in refractory cases

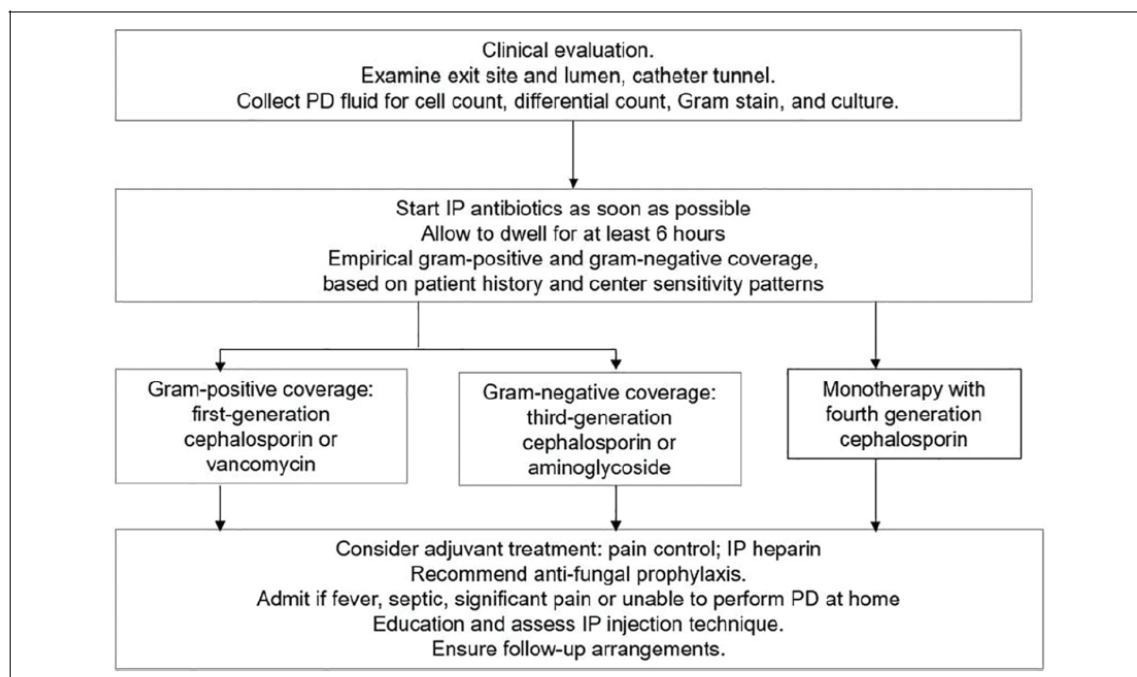


Figure 37: Management Algorithm for PD Peritonitis

Table 32: Summary of the Complications of Peritoneal Dialysis

| Early                                                                | Late                                                                                      |
|----------------------------------------------------------------------|-------------------------------------------------------------------------------------------|
| Surgical wound hemorrhage or catheter exit-site hemorrhage           | Problems with the outflow of the dialysate                                                |
| Bloody fluid from the catheter                                       | Hernias                                                                                   |
| Intestinal perforation, urinary bladder perforation                  | Leakage of dialysate to the scrotum                                                       |
| Dialysate leakage through the wound or exit site of the catheter     | Exit-site infections<br>Peritonitis<br>Encapsulating peritoneal sclerosis                 |
| Catheter related perioperative (perforation of viscus or hemorrhage) | Obstruction to flow<br>Leakage (exit site or concealed)<br>Pain (on infusion or drainage) |
| Related to increased intra-abdominal pressure                        | hernia<br>pleural leak (hydrothorax)<br>back pain                                         |
| Metabolic                                                            | Hyperglycemia, Hypertriglyceridemia, Hyperinsulinemia and Hyperleptinemia                 |
| Others                                                               | hemoperitoneum<br>encapsulating peritoneal sclerosis                                      |

Adopted from McCormick et al., 2018, and Sarabia-Pérez et al., 2023.

## Prevention

- Routine training and retraining in aseptic technique
- Exit-site care education
- Audit of peritonitis rates
- Use of flush-before-fill systems

## 10.6.8 Nutrition in Peritoneal Dialysis

Nutritional requirements include:

- Protein intake approximately 1.0 g/kg/day
- Energy intake approximately 30–35 kcal/kg/day
- Adjustment for glucose absorbed from dialysate

Nutritional status should be assessed regularly.

## 10.6.9 Indications for Switching from PD to Haemodialysis

Transition from PD to HD may be required in cases of:

- Recurrent or refractory peritonitis
- Ultrafiltration failure
- Inadequate solute clearance
- Mechanical catheter failure
- Severe malnutrition
- Patient preference

# Chapter 11: Electrolyte and Acid-Base Disorders

## 11.1 Introduction

The kidneys play a central role in maintaining electrolyte and acid–base homeostasis. They regulate the concentrations of sodium, potassium, calcium, magnesium, and phosphate in the bloodstream and are responsible for excreting excess hydrogen ions while conserving bicarbonate.

Electrolytes are essential for maintaining fluid balance, neuromuscular function, cardiac conduction, and cellular metabolism. In kidney disease, impaired glomerular filtration and tubular function predispose patients to electrolyte and acid–base disturbances. These abnormalities may worsen clinical outcomes and require prompt recognition and appropriate management.

Importantly, electrolyte and acid–base disorders are not limited to patients with chronic kidney disease (CKD) and may occur in individuals with normal renal function.

## 11.2 Common Electrolyte Derangements

The most common electrolyte derangements associated with kidney disease include:

- Hyperkalemia: Elevated potassium levels
- Hypokalemia: Decreased potassium levels
- Hypernatremia: Elevated sodium levels
- Hyponatraemia: Low sodium levels

### 11.2.1 Causes of electrolyte derangements

The causes of these electrolyte derangements in kidney disease can vary widely, but they often include:

- Decreased renal clearance: Impaired excretion of electrolytes due to reduced glomerular filtration rate (GFR).
- Dietary factors: High intake of potassium or phosphate-rich foods, particularly in patients on certain medications or with dietary restrictions.
- Acid-base imbalances: Conditions such as metabolic acidosis can influence electrolyte levels, particularly calcium and potassium.
- Medications: Some diuretics, ACE inhibitors, and other drugs can contribute to electrolyte imbalances.
- Comorbid conditions: Diabetes, heart failure, and other chronic illnesses can complicate electrolyte regulation.

## 11.3 Sodium Disorders

- Sodium ( $\text{Na}^+$ ) is the principal extracellular cation and a major determinant of extracellular fluid (ECF) volume.
- Serum sodium concentration reflects the ratio of total body sodium to total body water:
- $\text{Serum Na}^+ = \frac{\text{extracellular fluid (ECF) sodium}}{\text{extracellular fluid (ECF) water}}$
- Therefore, sodium disorders usually reflect disturbances in water balance rather than absolute sodium deficiency or excess.

### 11.3.1 Hyponatraemia

#### A. Definition

Serum sodium  $[\text{Na}] < 135 \text{ mmol/L}$

Hyponatraemia is typically a disorder of excess water relative to sodium.

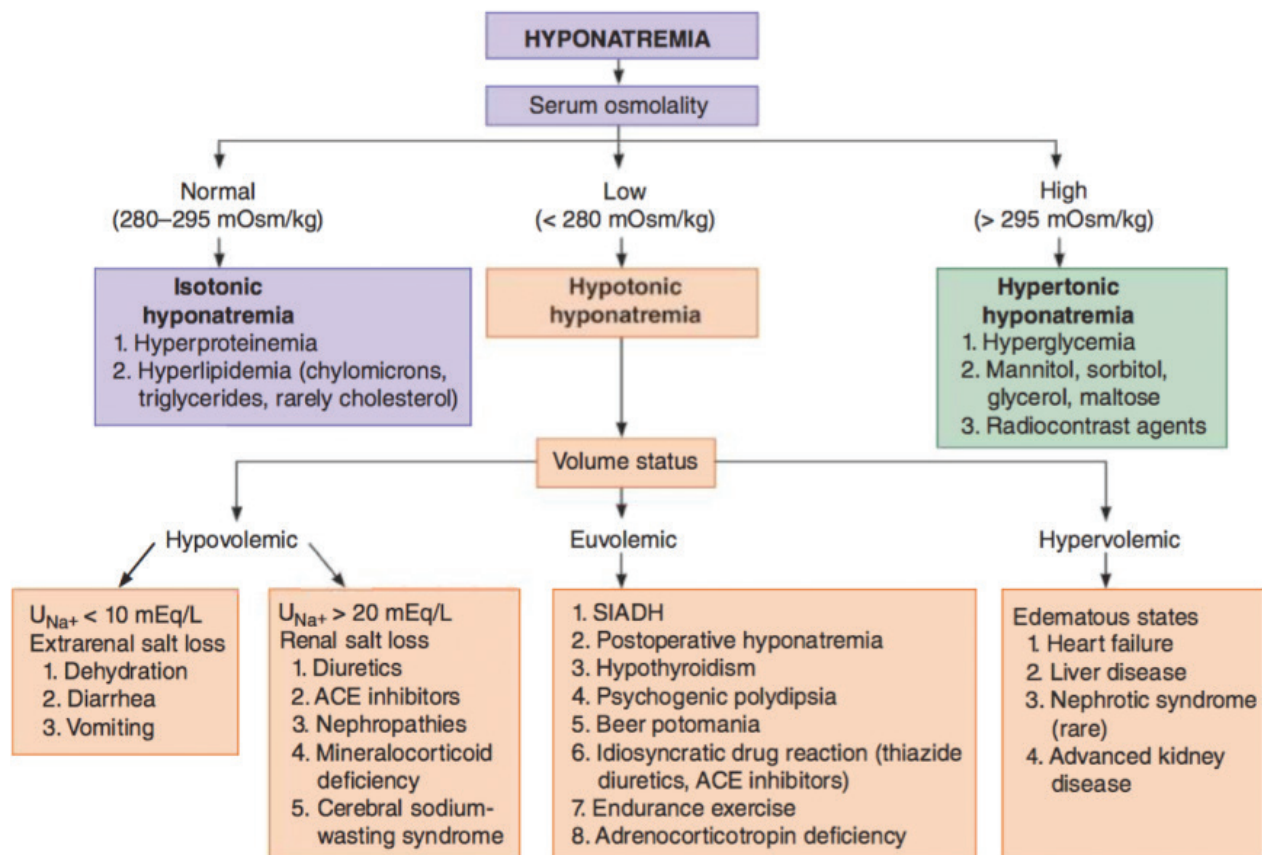
#### B. Evaluation of hyponatraemia

Evaluation should include:

- Assessment of severity based on serum sodium level
- Assessment of symptoms (headache, confusion, seizures, coma)
- Determination of onset:
  - o Acute:  $<48$  hours
  - o Chronic:  $>48$  hours
- Clinical assessment of volume status:
  - o Hypovolaemic
  - o Euvolaemic
  - o Hypervolaemic
- Measurement of serum and urine sodium
- Measurement of serum and urine osmolality

Table 33: Severity of Hyponatraemia

| Serum Sodium (mmol/L) | Severity               |
|-----------------------|------------------------|
| $<120$                | Severe hyponatraemia   |
| 120–130               | Moderate hyponatraemia |
| 131–135               | Mild hyponatraemia     |



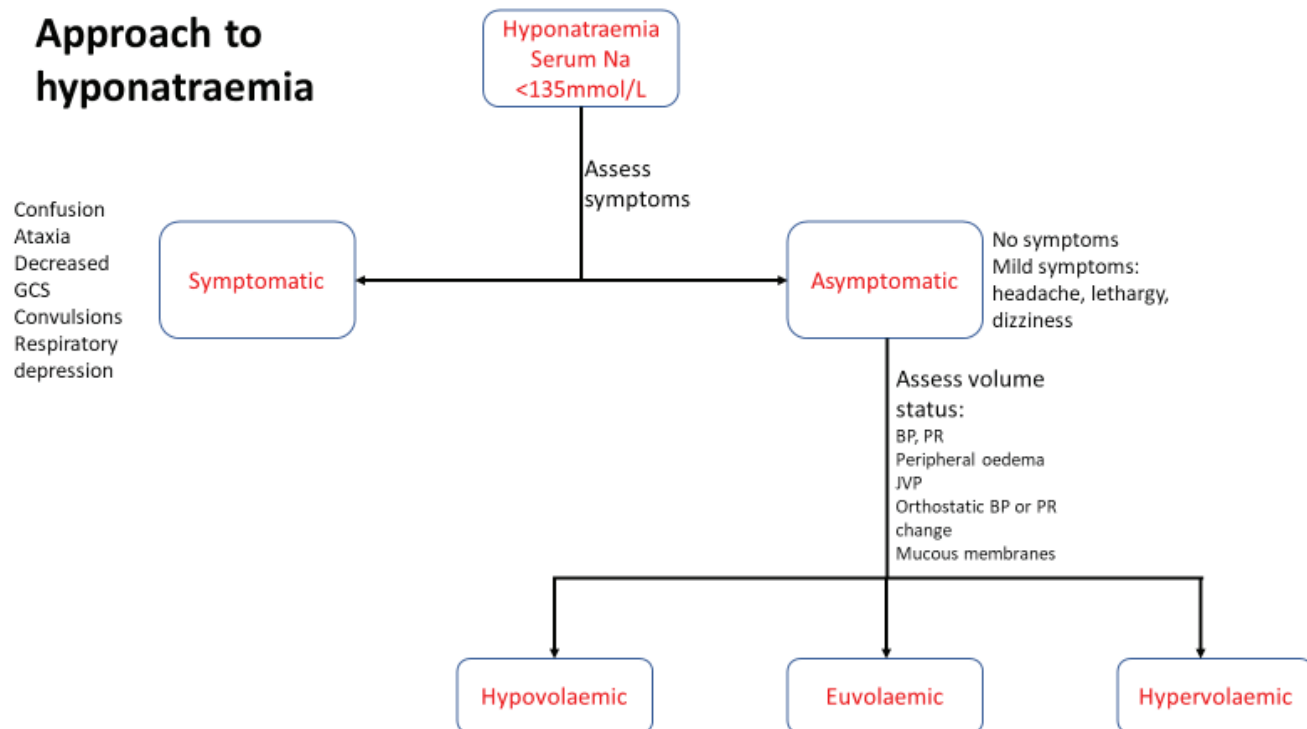
Approach to Hyponatraemia, (from Papadakis M.A., McPhee S.J., Rabow M.W. Eds. Maxine A. Current Medical Diagnosis & Treatment 2017 New York, NY: McGraw-Hill)

Figure 38: Approach to Hyponatraemia

### C. Diagnosis and Assessment:

- Diagnostic steps:
  - o Determine the severity by serum [Na] level (Table 33) and symptoms (e.g. headache, confusion, convulsions and coma)
  - o Determine the rate of onset (acute < 48 hours vs chronic >48 hrs)
  - o Determine volume status by clinical examination (hypovolemia, euvolemia or hypervolemia) (Figure 38)
- Principles:
  - o Severe symptomatic hyponatraemia requires urgent treatment regardless of cause, preferably in an ICU setup.
  - o Calculate sodium deficit to determine volume of sodium replacement:
  - o Na deficit = total body water (weight (kg) X 0.5 in females and 0.6 in males) X (desired Na – current Na)
  - o Normal saline = 0.9% saline = isotonic saline: contains 154mEq Na per litre
  - o Hypertonic saline = 3% saline: contains 513mEq Na per litre (or about 50mEq Na per 100mL bottle)

## D. Clinical approach to hyponatraemia



Approach to Hyponatraemia, (from Papadakis M.A., McPhee S.J., Rabow M.W. Eds. Maxine A. Current Medical Diagnosis & Treatment 2017 New York, NY: McGraw-Hill)

Figure 39: Approach to Hyponatraemia

## E. Management of Symptomatic Hyponatraemia

- Usually acute (<24h) and severe (serum Na <120mEq/L)
- Must be corrected promptly because it can lead to cerebral oedema, irreversible neurologic damage, respiratory arrest, brainstem herniation, and death

### Treatment: Hypertonic saline (3%)

- Continuous infusion 0.5–2 mL/kg/hour until symptoms resolve or
- 100–150 mL IV over 20 minutes, may repeat if needed

Monitor serum sodium every 2 hours.

### Correction Targets:

- Increase 1–2 mmol/L per hour initially
- 4–6 mmol/L rise sufficient to relieve symptoms
- Maximum 6–12 mmol/L in first 24 hours
- Maximum 18 mmol/L in first 48 hours

Excessively rapid correction may cause osmotic demyelination syndrome.

Once symptoms resolve, transition to isotonic saline guided by calculated sodium deficit and target sodium.

## **F. Classification-Based Management of Hyponatraemia**

### **i. Hypovolaemic Hyponatraemia**

Normal (0.9%) saline intravenously

- Sodium chloride tablets
  - o 1g NaCl tablet = 394mg Na = 17mEq Na
  - o Dose 1-2 tablets TID
- Table salt
  - o 1 teaspoon NaCl = 5.69gNaCl = approximately 100mEqNa
  - o Dose: Half to 1 teaspoon OD
- Manage aetiology

### **ii. Hypervolaemic Hyponatraemia**

- Restrict free water (to near zero) and total fluids (to less than 1.0L/24h)
- Loop diuretics (to encourage loss of more water than sodium)
- Block ADH with vasopressin receptor type 2 (V2) inhibitors (vaptans): e.g. Tolvaptan (not recommended in liver disease) 15-30mg OD
- Manage underlying cause

### **iii. Euvolaemic Hyponatraemia**

- Fluid restriction to 500 mL less than the daily urinary volume
- Salt and protein intake should not be restricted
- V2-receptor antagonist: e.g. Tolvaptan 15-30mg OD
- Treat hypothyroidism or Cushing's syndrome

## **11.3.2 Hypernatraemia**

### **A. Definition**

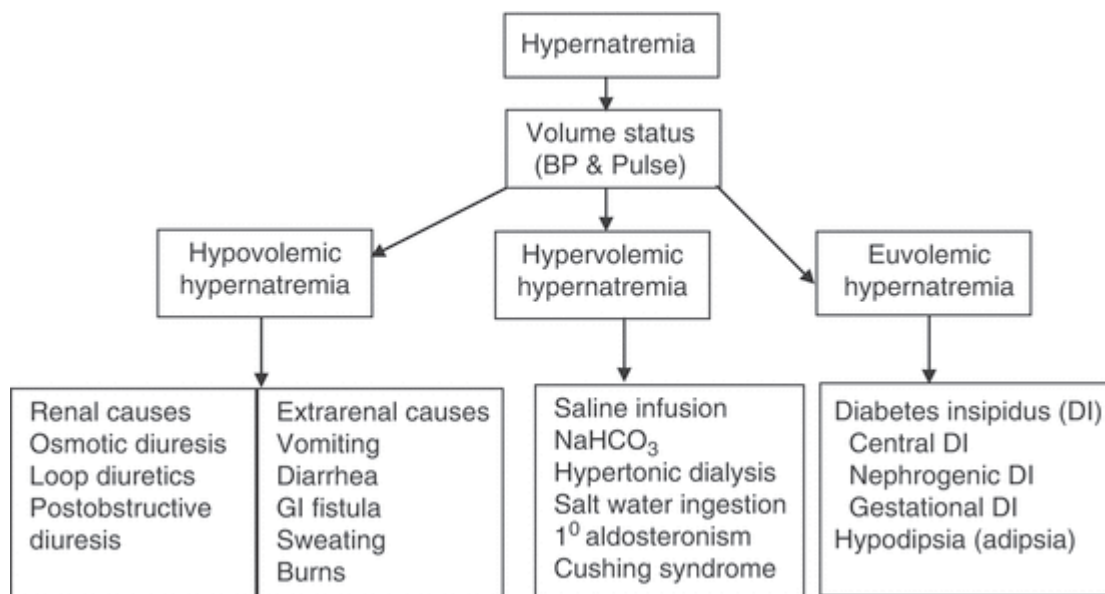
Serum Na concentration [Na] > 145 mmol/L.

Hypernatraemia usually reflects water deficit rather than sodium excess.

### **B. Causes**

- Reduced water intake (impaired thirst, limited access)
- Pure water loss (diabetes insipidus)
- Hypotonic fluid loss (diarrhoea, renal losses)
- Rarely sodium gain (iatrogenic)

### C. Classification of hypernatremia



Reddi, A.S. (2018). Disorders of Water Balance: Hypernatremia. In: Fluid, Electrolyte and Acid-Base Disorders. Springer, Cham. [https://doi.org/10.1007/978-3-319-60167-0\\_13](https://doi.org/10.1007/978-3-319-60167-0_13)

Table 40: Classification of hypernatremia

### D. Evaluation of hypernatremia

- Determine volume status
- Assess access to water
- Measure urine output and osmolality
- Consider diabetes insipidus if urine output >3 L/day and urine osmolality <100 mOsm/kg

### Assessment and Diagnosis of Hypernatremia

- Asymptomatic or mild symptoms in adults: irritability, nausea, weakness, altered mental status, or coma
- Severe symptoms if rapid [Na] increase or serum [Na].160mEq/L or in infants: tachypnea, muscle weakness, restlessness, a high-pitched cry, insomnia, lethargy, and coma

### E. Classification-Based Management of Hypernatremia

#### I. Hypovolaemic hypernatremia:

- Water replacement:
  - o Calculate water deficit =
$$\frac{\text{total body water (body weight X 0.5 in females or 0.6 in males)}}{\text{(current [Na]-target [Na])/140}}$$
- To correct fluid loss, the replacement fluid has to be more hypotonic than the fluid being lost (e.g. urine, diarrhoea, sweat)
  - o Administration routes:
    - Enteral water preferred: orally or nasogastric or nasojejunal tube

- If enteral not possible or adequate: IVI:
  - 5% Dextrose ([Na] = 0mEq/L)
  - Other options (if replacement fluid is hypotonic to fluid being lost):
    - o Half-saline ([Na] = 77mEq/L)
    - o Ringer's lactate ([Na] = 130mEq/L)
- Monitor serum [Na] every 6h and adjust fluid replacement rate as appropriate
- Target [Na] increase:
  - o If acute or severe symptoms: [Na] decrease by 1mEq/L every hour
  - o If chronic or minimal symptoms: [Na] decrease by 0.5mEq/L every hour (not >10mEq/L over 24h)
  - o Faster increase in Na can cause cerebral oedema
- Target final [Na]: 140mEq/L
- Treat underlying cause

#### ii. Euvolaemic hypernatremia:

- Treat underlying cause
- Diabetes insipidus: refer to nephrologist

#### iii. Hypervolemic hypernatremia:

Treat underlying cause

## 11.4 Potassium Disorders

- Potassium is the most abundant cation:  $\approx 4000\text{mmol}$  in body
- However, only 2% in extracellular fluid (60mmol); 98% in intracellular fluid
- Normal serum potassium concentration ([K]) 3.5-5.0mEq/L
- Potassium disorders interfere with the cell membrane potential, interfering with neural and muscular function

### 11.4.1 Hypokalaemia

#### A. Definition:

Serum [K] <3.5mEq/L

## B. Aetiology of Hypokalemia

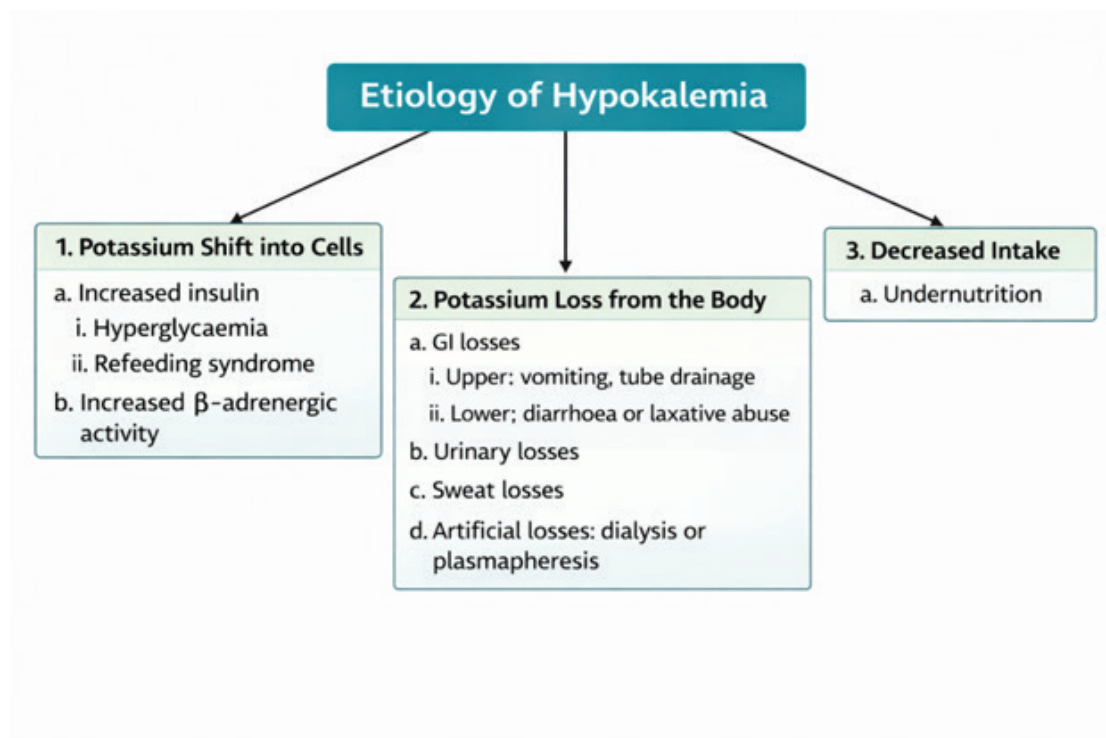


Figure 41: Aetiology of Hypokalaemia

## C. Clinical Features

Often asymptomatic, but can present with:

- Muscular: weakness from skeletal muscle hyperpolarization
- Cardiovascular: arrhythmias
- Renal: impaired concentration, metabolic alkalosis, tubulointerstitial fibrosis
- Hormonal: impaired insulin release, insulin resistance
- ECG changes:
  - o Appearance of U wave
  - o QT interval looks prolonged
  - o Flattened T wave
  - o Widened QRS
  - o Supraventricular and ventricular ectopic rhythms

## D. Clinical Approach to Hypokalaemia

### Step 1. Is there an emergency?

Evaluate for danger signs and symptoms and treat urgently:

- Muscle weakness: can progress to respiratory failure
- ECG changes: progressive flattening of T waves with appearance of U waves ST segment depression, arrhythmias

Step 2. Is it acute (less than 48h) or chronic?

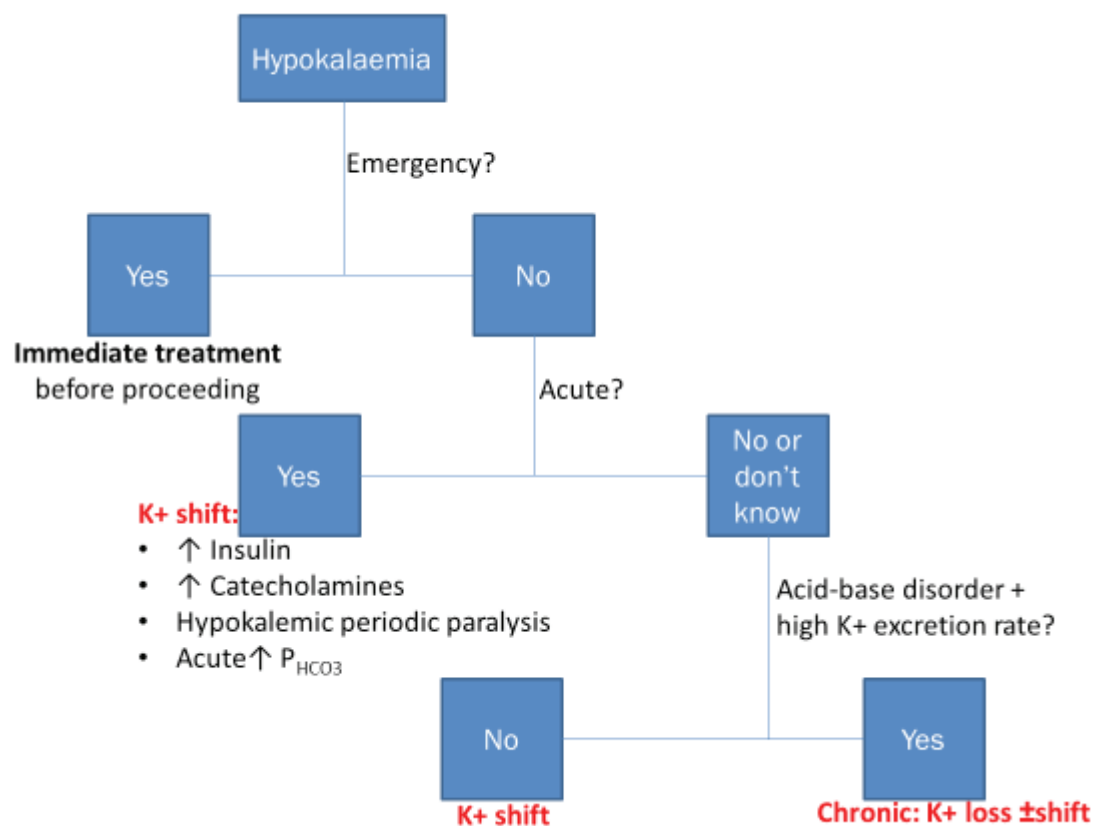
Acute = shift

Chronic is often from losses:

- Evaluate for common causes of hypokalaemia:
  - o GI losses: vomiting, diarrhoea
  - o Urine losses: diuretic use, especially thiazide diuretics

Step 3. If no common cause found, do a spot urine [K] and urine creatinine (Cr) concentration and calculate the urine K/Cr ratio)

- Urine K/Cr ratio  $<1.5\text{mEq/mmol}$  ( $13\text{mEq/g}$ ):
  - o = appropriate tubular reabsorption of potassium
  - o therefore, extrarenal losses (or transcellular shift)
- Urine K/Cr ratio  $>1.5\text{mEq/mmol}$  ( $13\text{mEq/g}$ ):
  - o = inappropriate tubular reabsorption of potassium
  - o therefore, renal losses
  - o Refer to nephrologist to complete evaluation



K+: serum potassium level;  $\text{PHCO}_3$ : plasma bicarbonate concentration

Figure 42: Clinical Approach to Hypokalemia

## E. Management of Hypokalaemia

### 1. Severe symptoms or serum [K] <3.0mEq/L or ECG changes:

- ECG continuous monitoring
- Estimate potassium deficit: Every 0.3mEq/L decrease in serum [K] = 100mEq reduction in total body potassium
- Intravenous replacement needed
  - o Risk of arrhythmias with too-rapid infusion
  - o Preferably administer via central venous line:
    - 80-120mEq potassium chloride (KCl) in 500mL normal saline over 4h
    - Avoid giving KCl in dextrose-containing fluids – can worsen hypokalaemia by causing intracellular shift
  - o If peripheral line:
    - Maximum concentration of IV fluid potassium: 40mEq/L
    - Maximum rate of IV fluid potassium infusion: 40mEq/h
    - Use 40mEq KCl in 1L normal saline over 4-6 hour
- Monitor serum K every 6h

### 2. Mild or no symptoms:

Enteral replacement:

- Preferred route
- Potassium citrate syrup (approx. 1 mmol/mL)
  - o Give 20-30ml mixed with fruit juice (to mask bitter taste) 6-8 hourly
  - o Safe and effective
- Potassium chloride liquid (from IV vial; 2mmol/mL)
  - o Give 20-30mL mixed with fruit juice 6-8 hourly
- Potassium chloride tablets:
  - o Slow K is slow-release
  - o Too slow: not to be used in emergency or urgent cases
  - o Causes more gastric corrosion as local K concentration can be high
- Monitor serum [K] daily

Identify underlying cause and manage appropriately

If persistent or recurring hypokalaemia: check serum magnesium concentration and replace as appropriate

### 3. Hypokalaemia in diabetic ketoacidosis (DKA):

Consult with physician or diabetologist and follow DKA guidelines

## 11.4.2 Hyperkalaemia

### A. Definition

Serum [K] >5.0mEq/L

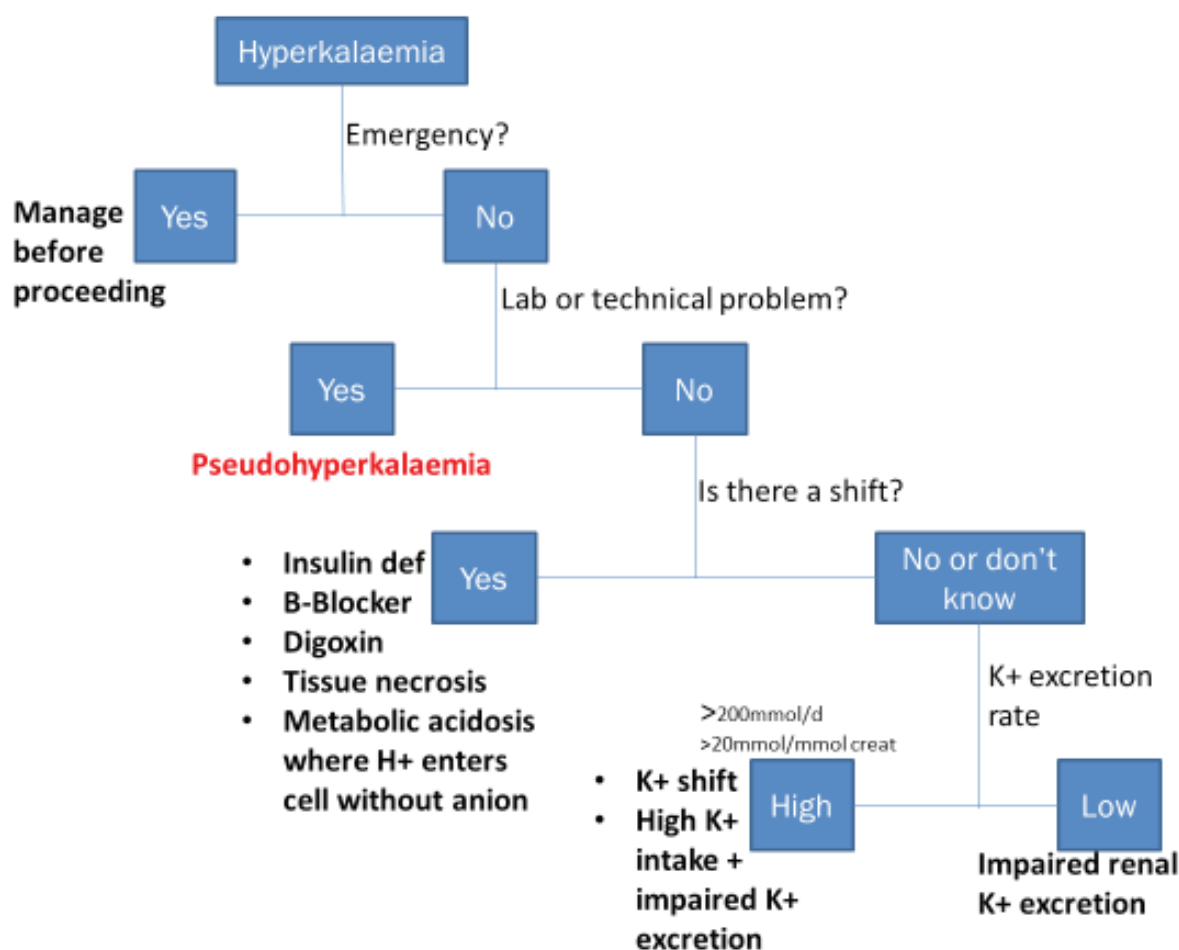
### B. Aetiology:

- Hyperkalaemia rarely occurs from excess intake unless there is a concomitant renal excretion problem
- Pseudohyperkalaemia is common: from haemolysis of the sample

### C. Classification

- Mild: 5.5–5.9 mEq/L
- Moderate: 6.0–6.4 mEq/L
- Severe:  $\geq 6.5$  mEq/L or ECG changes

### D. Clinical approach to Hyperkalaemia



H+: serum hydrogen ions; K+: serum potassium level

Figure 43: Clinical Approach to Hypokalemia

## E. Evaluation and Management

### Step 1: Is there an emergency?

- Assess for presence of:
  - o Muscle weakness
  - o ECG changes:
    - Peaked narrow-based T-waves
    - Widened QRS duration
    - Prolonged PR interval
    - Broad P waves, disappearing P waves
    - Sine wave pattern
  - o Severe hyperkalaemia

If emergency present:

- Protect the heart:
  - o Intravenous calcium:
    - 10% Calcium Gluconate 30ml slow intravenous infusion over 10 minutes or
      - 10% Calcium Chloride 10mL slow intravenous infusion over 10 minutes (preferred during resuscitation)
    - Do NOT give IV push: can cause cardiac arrest
- Immediately lower serum [K]:
  - o Shift potassium into cells:
    - Soluble insulin 10 units plus 50% dextrose 50mL IV push
      - Monitor blood sugar hourly during the first 3 hours due to risk of hypoglycaemia
      - If RBS before insulin is less than 7.0mmol/L, after the insulin-dextrose push give 10% Dextrose to run at 50mL/h for 5h
    - $\beta$ 2-agonist: Salbutamol 10 to 20 mg by nebulisation over 10 mins
      - Avoid in patients at risk of arrhythmias
      - Not to be used as sole therapy for hyperkalaemia

### Step 2: Remove potassium from the body

- For all severity classes of hyperkalaemia
- For both acute and chronic hyperkalaemia
- Routes of removal:

| Via urine:                                                                                                                                                                                                                                                                                                                                                                                                                                                                         | Via GIT:                                                                                                                                                                                                                                                                                                                                                                                                                      | Via Haemodialysis:                                                                                                                                                                                                     |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Diuretic:</b> <ul style="list-style-type: none"> <li>Furosemide 20-40mg IV or 40-80mg PO BID or TID (depending on volume status of patient)</li> </ul> <b>Sodium bicarbonate:</b> <ul style="list-style-type: none"> <li>Intravenously 300mg IV: use only during resuscitation if hyperkalaemia is thought to be the cause of cardiac arrest</li> <li>Orally 600mg to 1200mg BID for chronic hyperkalaemia in CKD (especially if serum HCO<sub>3</sub> &lt;22mmol/L)</li> </ul> | <b>Cation exchange resins:</b> <ul style="list-style-type: none"> <li>Sodium Polystyrene Sulfonate (SPS) 15g BID/TID</li> <li>Calcium Polystyrene Sulfonate 15gBID/TID preferred if patient is fluid overloaded</li> </ul> <b>Newer resins preferred where available:</b> <ul style="list-style-type: none"> <li>Patiromer 8.4g PO OD</li> <li>Sodium Zirconium Cyclosilicate (SZC) 10g PO TID for 48h then 10g OD</li> </ul> | <ul style="list-style-type: none"> <li>Preferred method for CKD stage 5 patients already on maintenance HD</li> <li>Also used if medical therapy fails</li> <li>Concomitant medical therapy should continue</li> </ul> |

### Step 3: Address aetiology

- Remove sources of K<sup>+</sup> load:
  - Dietary: fruits, vegetables, and juices
  - Assess for blood cell breakdown: GI blood, haematoma, haemolysis
- Remove causes of impaired renal K<sup>+</sup> elimination:
  - Potassium-sparing diuretics: spironolactone, amiloride
  - Angiotensin blockers: angiotensin converting enzyme inhibitors (e.g. enalapril), angiotensin-1 receptor blockers (e.g. losartan), angiotensin-receptor-neprilysin inhibitors (ARNI) (e.g. sacubitril-valsartan)
  - NSAIDs
  - Trimethoprim (e.g. in Septrin)

### **F. Monitoring of Hyperkalaemia**

- Acute or severe hyperkalaemia
  - Repeat serum K every 4h in first 24h then daily in first 1 week until controlled and stable
- Chronic hyperkalaemia:
  - At risk of hyperkalaemia e.g. diabetes, CKD: serum K every 4 months
  - Angiotensin blockade or mineralocorticoid receptor antagonist:
    - Before initiation: measure serum K: if hyperkalaemia present caution with angiotensin blocker, avoid mineralocorticoid receptor antagonist
    - After initiation: serum K after 4 weeks
- After episode of hyperkalaemia:
  - Mild hyperkalaemia: repeat serum K within 1 week
  - Moderate hyperkalaemia: repeat serum K within 24h
  - Severe hyperkalaemia: repeat serum K every 4h in first 24h then daily in first 1 week until controlled and stable.

## 11.5 Acid–Base Disorders

### 11.5.1 Definitions

- Normal ranges:
  - o Arterial pH 7.35-7.45 ( $7.40 \pm 0.05$ )
  - o Arterial pCO<sub>2</sub> 35-40mmHg ( $40 \pm 5$ )
  - o Arterial HCO<sub>3</sub> 22-26mmol/L ( $24 \pm 2$ )
- Acidaemia: arterial pH < 7.35.
- Acidosis: process of getting acidaemia
  - o Can be:
- Metabolic (gain in H<sup>+</sup> or HCO<sub>3</sub><sup>-</sup> loss) or Respiratory (gain in CO<sub>2</sub>)
- Compensated (fully (no acidaemia present) or partially (acidaemia present but less than expected)) or Uncompensated (expected acidaemia present)
- Alkalemia: arterial pH > 7.45
- Alkalosis: process of getting alkalaemia
  - o Can be:
- Metabolic (loss in H<sup>+</sup> or HCO<sub>3</sub><sup>-</sup> gain) or Respiratory (loss in CO<sub>2</sub>)
- Compensated {fully (no alkalaemia present) or partially (alkalaemia present but less than expected)} or Uncompensated (expected alkalaemia present)

### 11.5.2. Approach to acid-base disorder

#### Step 1: Examine the pH, HCO<sub>3</sub><sup>-</sup>, and the P0<sub>2</sub>

- Low pH or low-normal pH:
  - o Acidosis is going on
  - o Check expected cause of acidosis:
- Low HCO<sub>3</sub><sup>-</sup>: metabolic acidosis
- High pCO<sub>2</sub>: respiratory acidosis
- High pH or high-normal pH:
  - o Alkalosis is going on
  - o Check expected cause of alkalosis:
    - High HCO<sub>3</sub><sup>-</sup>: metabolic alkalosis
    - Low pCO<sub>2</sub>: respiratory alkalosis

#### Step 2: Determine the compensatory response:

- Is it fully compensated or partially compensated?
- Consult a physician or intensivist, or a nephrologist to guide

### 11.5.3 Metabolic Acidosis

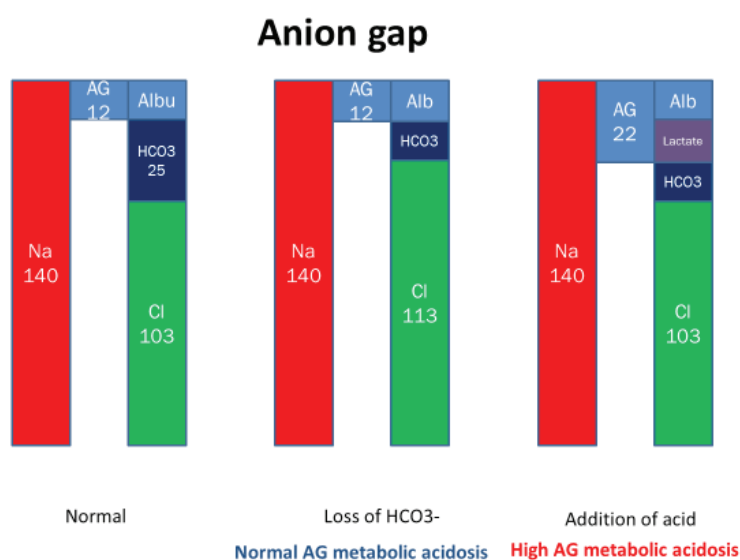
**A. Definition:** Serum HCO<sub>3</sub> concentration <22mmol/L (mEq/L)

## B. Aetiology of Metabolic Acidosis

| Endogenous Source                                                                                                               | Exogenous Sources                                                          | Renal Causes                                                                                                                           | Gastrointestinal causes                                                                          | Others                                                                        |
|---------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------|
| Ketoacids (Diabetes, starvation, alcoholic)<br><br>Lactic acidosis (tissue hypoperfusion, drug, liver disease)<br>Renal Failure | Metabolism of toxic alcohol(ethylene glycol, methanol, and salicylic acid) | <ul style="list-style-type: none"> <li>Renal Tubular acidosis</li> <li>Renal Failure</li> <li>Carbonic anhydrase inhibitors</li> </ul> | <ul style="list-style-type: none"> <li>Severe Diarrhoea</li> <li>Ureterosigmoidoscopy</li> </ul> | <ul style="list-style-type: none"> <li>Ammonium chloride ingestion</li> </ul> |

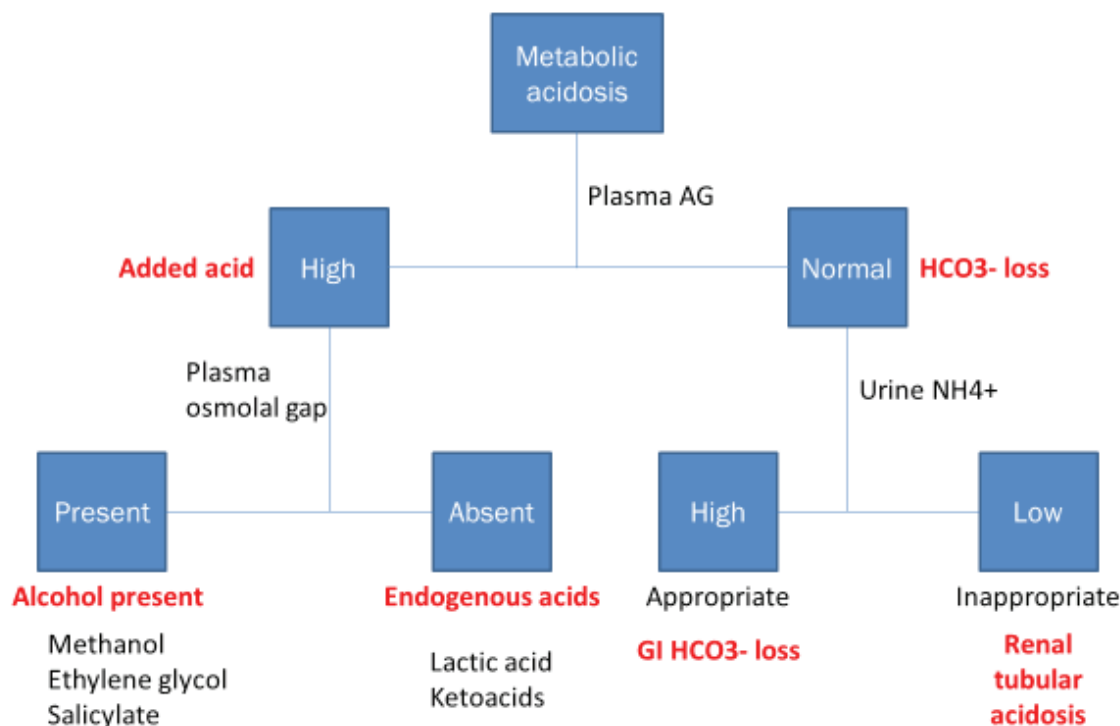
## C. Approach to Metabolic Acidosis

- Principle of electroneutrality: Serum negative ions (anions, e.g.  $\text{Cl}^-$ ,  $\text{HCO}_3^-$ ) must balance serum positive ions (cations, e.g.  $\text{Na}^+$ ,  $\text{K}^+$ )
- When  $\text{HCO}_3^-$  concentration decreases in metabolic acidosis, an extra anion must compensate for this decrease: this is the anion gap
- Anion gap calculation:
  - Checks for presence of extra anion that is not chloride ( $\text{Cl}^-$ )
  - $\text{AG} = [\text{Na}^+] - ([\text{HCO}_3^-] + [\text{Cl}^-])$
  - Normal plasma AG =  $12 \pm 2 \text{ mEq/L}$ : caused mainly by serum albumin



AG: anion gap; Alb: albumin; Cl: chloride;  $\text{HCO}_3$ : bicarbonate; Na: sodium

Figure 44: Concept of Anion Gap



AG: anion gap; HCO<sub>3</sub><sup>-</sup>: bicarbonate ions; NH<sub>4</sub>: ammonium Step 1: Calculate the serum anion gap

Figure 45: Clinical Approach to Metabolic Acidosis

Step 1: Calculate the serum anion gap

- Normal anion gap metabolic acidosis (NAGMA):
  - o Anion Gap < 12
  - o Indicates loss of HCO<sub>3</sub><sup>-</sup>:
    - GI loss: diarrhoea
    - Renal loss: renal tubular acidosis
  - o If no overt diarrhoea, consult nephrologist for further evaluation
- High anion gap metabolic acidosis (HAGMA):
  - o Anion gap > 12 (usually >20)
  - o Indicates acid (H<sup>+</sup> donor) has been added to serum: acid gain
  - o H<sup>+</sup> donor comes as H-Anion which dissociates to H<sup>+</sup> (causing acidosis) and Anion (causing anion gap)
  - o Acid gain can be:
    - Endogenous:
      - Lactic acidosis:
        - o Type 1 from hypoperfusion: e.g. hypovolaemia, shock
        - o Type 2: from impaired mitochondrial respiration: e.g. metformin
      - Ketoacidosis:
        - o Diabetic ketoacidosis
        - o Alcohol ketoacidosis
        - o Fasting ketoacidosis

- o Pregnancy ketoacidosis
- Exogenous:
  - Usually from metabolism of an alcohol:
    - o Methanol to formic acid
    - o Ethylene glycol to glycolic acid
    - o Salicylic acid
    - o Detect presence of alcohol by calculating serum osmolol gap:
 

Serum osmolol gap = measured serum osmolality (measured directly by lab) – calculated serum osmolality (2 X {serum Na + K} + serum urea + blood glucose)
- o Normal osmolol gap = -10 to +10mOsm/kg
- o Critical osmolol gap = 15mOsm/kg: a medical emergency

#### D. Management of Metabolic Acidosis

Severe acidosis: pH < 7.1 or pH < 7.2 with renal failure or hemodynamically unstable

- o Give IV sodium bicarbonate (NaHCO<sub>3</sub>):
    - 150-300mEq over 1h and repeat if needed
  - o Monitor arterial blood gas every 4h
  - o Check serum calcium and correct if low: NaHCO<sub>3</sub> can precipitate seizures in hypocalcaemic patients
- Identify and treat aetiology:
- o Improve perfusion: volume bolus, vasopressors, blood transfusion
  - o Treat ketoacidosis: insulin infusion in diabetic ketoacidosis
  - o Manage diarrhoea
  - o Manage intoxications by alcohols
  - Dialysis (usually haemodialysis) if acidosis persisting or severe or if an alcohol suspected

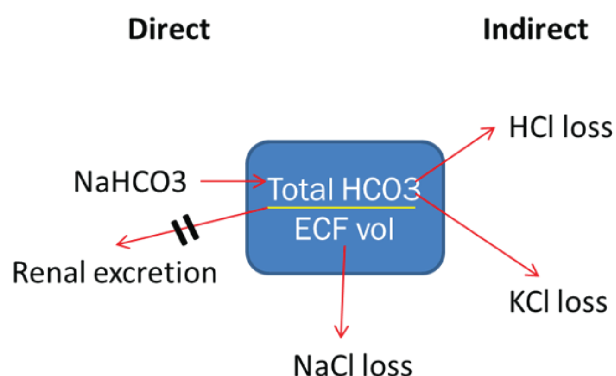
## 11.5.4 Metabolic Alkalosis

**A. Definition:** Serum HCO<sub>3</sub><sup>-</sup> > 26mmol/L

#### B. Aetiology of Metabolic Alkalosis

- Common causes of metabolic alkalosis:
  - o Prolonged vomiting and nasogastric tube suctioning
  - o Diuretic use
  - o Primary aldosteronism

- Metabolic alkalosis due to volume loss (contraction alkalosis) ( $\downarrow$ ECF volume):
  - o Urine loss:
    - Diuretics
    - Diuretic-like states: Bartter syndrome, Gitelman syndrome
  - o Upper GI loss:
    - Vomiting
    - NG tube drainage
- Metabolic alkalosis due to alkali gain ( $\uparrow$ HCO<sub>3</sub>):
  - o Normal renal function (only in association with K<sup>+</sup> depletion or low NaCl intake):
    - Alkali intake
      - Oral NaHCO<sub>3</sub>
      - IV NaHCO<sub>3</sub> to treat metabolic acidosis
      - Large quantities of citrate during:
  - o Transfusion of >8 units of stored blood
  - o Use of citrate as anticoagulant in HD
  - o Transfusion of FFP as replacement fluid during plasmapheresis
  - o Renal failure:
    - Milk-alkali syndrome
    - Aluminium hydroxide with K<sup>+</sup>-exchange resin
    - Extensive use of crack cocaine in dialysis patients

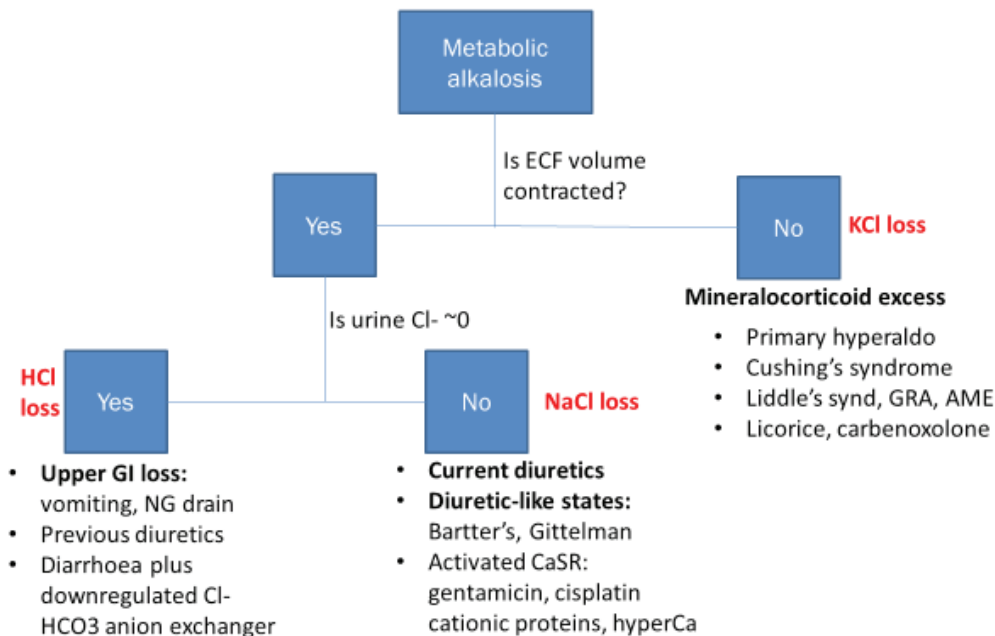


ECF: extracellular fluid; HCl: hydrochloric acid; HCO<sub>3</sub>: bicarbonate; KCl: potassium chloride; NaCl: sodium chloride; NaHCO<sub>3</sub>: sodium bicarbonate

Figure 46: Aetiology of Metabolic Alkalosis

### C. Approach to metabolic alkalosis

- $[\text{HCO}_3] = \text{total HCO}_3 / \text{ECF water volume}$
- Hence, increased serum HCO<sub>3</sub> may be due to:
  - o Increased total HCO<sub>3</sub>
  - o Decreased ECF volume
- Once elevated serum HCO<sub>3</sub> occurs, renal tubular excretion of HCO<sub>3</sub> increases and eliminates the excess HCO<sub>3</sub>. Hence, maintenance of metabolic alkalosis requires a concomitant decrease in renal HCO<sub>3</sub> excretion (= increase in renal H<sup>+</sup> excretion)



AME: apparent mineralocorticoid excess syndrome; CaSR: calcium-sensing receptor; Cl<sup>-</sup>: chloride ions; ECF: extracellular fluid; GRA: glucocorticoid-remediable aldosteronism; HCl: hydrochloric acid; HCO<sub>3</sub><sup>-</sup>: bicarbonate; hyperaldo: hyperaldosteronism; hyperCa: hypercalcaemia; KCl: potassium chloride; NaCl: sodium chloride; NG: nasogastric; synd: syndrome;

Figure 47: Approach to Metabolic Alkalosis

#### D. Management of Metabolic Alkalosis

- Identify and treat aetiology
- Hypovolaemic (chloride-sensitive) metabolic alkalosis:
  - o Provide chloride:
    - Intravenous normal saline (0.9% NaCl) is critical
    - Rate: start at 100mL/h and titrate to achieve 50mL/h more than urine losses and insensible losses
    - Target urine Cl >25mEq/L
  - o Provide potassium:
    - Treatment of concomitant hypokalaemia is also critical in resolving the metabolic alkalosis
    - Give potassium chloride infusion to target serum K<sup>+</sup> >4.5mEq/L
    - Avoid potassium citrate: citrate will worsen the metabolic alkalosis
- Euvolaemic/hypervolaemic (chloride-resistant) metabolic alkalosis:
  - o Replace potassium as above
  - o Acetazolamide to promote HCO<sub>3</sub><sup>-</sup> excretion (increase potassium supplementation to avoid hypokalaemia)
  - o Haemodialysis for severe metabolic alkalosis (pH.7.50)
  - o Consult nephrologist

# Chapter 12: Nutrition in Kidney Disease

## 12.1 Introduction

Nutrition plays a critical role in the prevention and management of kidney disease across all stages of care, from early chronic kidney disease (CKD) to dialysis and kidney transplantation. Appropriate nutritional management supports preservation of kidney function, prevention of complications, improvement of treatment outcomes, and enhancement of patients' quality of life. This chapter provides a standardized approach to nutrition care for patients with kidney disease, guided by the Nutrition Care Process (NCP), which includes nutrition assessment, diagnosis, intervention, monitoring, and evaluation, to ensure comprehensive, patient-centered, and evidence-based nutritional management.

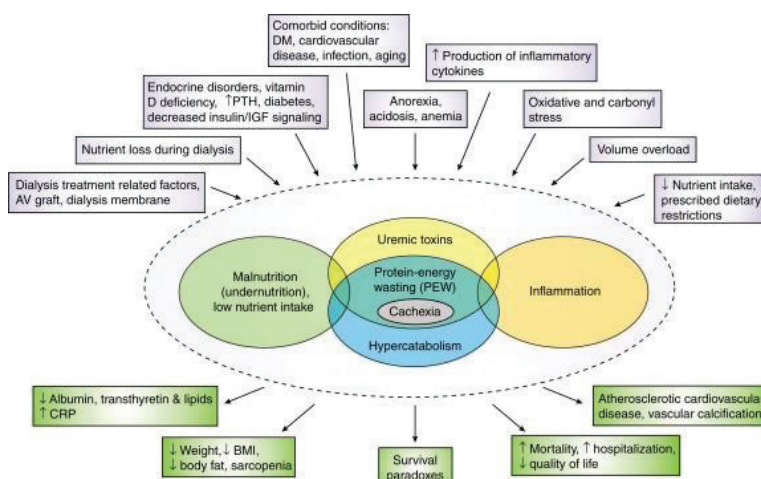
## 12.2 Goals of Medical Nutrition Therapy in CKD

- Maintain normal body mass and composition
- Prevent or retard development of bone disease.
- To assess, prevent and treat protein energy wasting.
- Minimize comorbidities associated with CKD
- Slow progression of CKD
- Achieve good long-term outcomes in mortality and morbidity
- Maintain normal growth and development in children

## 12.3 Aetiology of Malnutrition in CKD

### 12.3.1 Pathophysiological interactions underlying protein–energy wasting in ckd

Figure 48 depicts the interconnected mechanisms underlying protein–energy wasting (PEW) in chronic kidney disease, where inflammation, uremic toxins, and hypercatabolism interact with malnutrition and reduced nutrient intake. Contributing factors such as comorbidities, endocrine disturbances, dialysis-related losses, and oxidative stress collectively lead to muscle loss, hypoalbuminemia, increased cardiovascular risk, and higher morbidity and mortality.



Note: (Adapted from Fouque, Kalantar-Zadeh, Kopple, Cano, Chauveau, Cuppari, ... & Wanner, 2008)

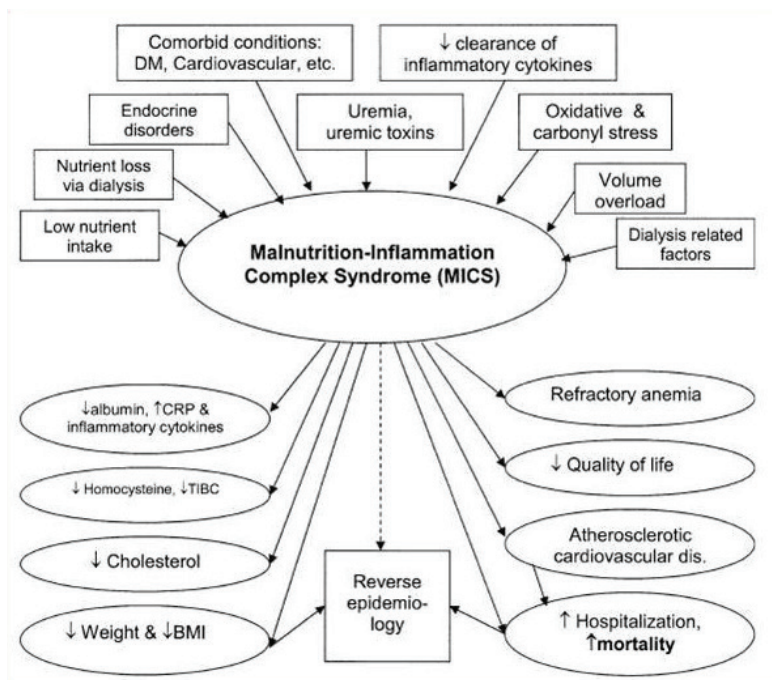
Figure 48: The Causes and Manifestations of the Protein–Energy Wasting Syndrome in Kidney Disease

### 12.3.2 Causes of wasting and protein–energy malnutrition (PEM) in dialysis patients

1. **Inadequate nutrient intake anorexia which is caused by**
  - Uraemic toxicity
  - Impaired gastric emptying
  - Inflammation with/without comorbid conditions
  - Emotional and/or psychological disorders
2. **Dietary restrictions**
  - Prescribed restrictions: Low-potassium, low-phosphate regimens
  - Social constraints: Poverty, inadequate dietary support
  - Physical incapacity: Inability to acquire or prepare food or to eat
3. **Nutrient losses during dialysis**
  - Loss through the haemodialysis membrane into the haemodialysate
  - Adherence to the haemodialysis membrane or tubing
  - Loss into the peritoneal dialysate
4. **Hypercatabolism which is caused by**
  - Comorbid illnesses
  - Cardiovascular diseases
  - Diabetic complications
  - Infection and/or sepsis
  - Other comorbid conditions
5. **Hypercatabolism which is associated with dialysis treatment**
  - Negative protein balance
  - Negative energy balance
6. **Endocrine disorders of Uraemia**
  - Resistance to insulin
  - Resistance to growth hormones and/or IGF-1
  - Increased serum levels of or sensitivity to glucagon
  - Hyperparathyroidism
  - Other endocrine disorders
7. **Acidaemia with metabolic acidosis**
  - Concurrent nutrient loss with frequent blood losses

### 12.3.3 Relationship between PEM and inflammation

- Chronic kidney disease is associated with persistent inflammation due to uremia, oxidative stress, comorbidities, endocrine disturbances, and dialysis-related factors.
- Reduced clearance of inflammatory cytokines sustains a chronic inflammatory state that drives Protein–Energy Malnutrition (PEM).
- Proinflammatory cytokines (e.g., TNF- $\alpha$ ) promote catabolism, increase protein degradation, suppress protein synthesis, and induce anorexia, leading to muscle wasting and weight loss.
- Inflammation contributes to biochemical changes such as hypoalbuminemia and hypocholesterolemia—markers of poor nutritional status and strong predictors of mortality in dialysis patients.
- The interplay between malnutrition and inflammation (MICS) is strongly associated with refractory anemia, cardiovascular disease, reduced quality of life, increased hospitalization, and higher mortality (reverse epidemiology in CKD). Ref to figure 49



Adapted from Anand, Chandrasekaran, & Alam, 2013

Figure 49: Malnutrition Inflammation Complex Syndrome

## 12.4 Nutrition Assessment

### 12.4.1 Nutritional Assessment Tools for Patients with Kidney Disease

- Nutrition assessment for renal patients is important for it aids in making diagnosis and intervene successfully, this includes:
  - Anthropometry
  - Biochemical parameters
  - Subjective Global Assessment (SGA)
  - Dietary Assessment
- Biochemical monitoring is critical to guide individualised care and prevent complications.

## 12.4.2 Screening for undernutrition

### A. Core Nutritional Screening Parameters in Dialysis

As part of routine screening for undernutrition in patients undergoing dialysis, the following minimum parameters should be assessed to enable early identification and timely nutritional intervention, thereby improving clinical outcomes and reducing mortality:

- Actual Body Weight (ABW): concern if ABW < 85% of Ideal Body Weight (IBW)
- Reduction in oedema free body weight: concern if reduction ≥5% in 3 months or ≥10% in 6 months
- BMI: concern if <23kg/m<sup>2</sup>
- Subjective Global Assessment (SGA): concern if B/C on 3-point scale or 1-5 on 7-point scale

### B. Frequency of Screening for Undernutrition in CKD

- At least monthly in MHD and PD patients
- At least every 3 months in patients with CKD 4-5 or post-transplantation
- At least every 6 months in patients with CKD 1-3

|                                                                                                                                                                                                                             |                                                                                                                                                                                                                           |                                                                                                                                                           |                                                                                                                                    |                                                                                                                                                                                                                                                               |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Dietary intake<br>Appetite<br>Weight for age<br>Weight for length<br>Height for age<br>Height velocity<br>Head circumference for age<br>Weight gain per day<br>Biochemistry:<br>*S. albumin                                 | Dietary intake<br>Appetite<br>Anthropometry:<br>Weight for age<br>Weight for height<br>Height for age<br>Height velocity<br>Head circumference for age<br>BMI for height age in > 2 years<br>Biochemistry:<br>*S. albumin | Dietary intake<br>Appetite<br>Anthropometry:<br>Weight for age<br>BMI for height age<br>Height for age<br>Height velocity<br>Biochemistry:<br>*S. albumin | Dietary intake<br>Appetite<br>Anthropometry:<br>Weight for age<br>BMI for height age<br>Height for age<br>Height velocity<br>nPCR* | Dietary intake<br>Body weight, BMI<br>Skinfold thickness<br>^Waist circumference<br>Body composition:<br>*BIA/ DXA<br>Complementary tools:<br>nPCR / <sup>#</sup> S. albumin<br>Muscle strength<br>Resting energy expenditure<br>^Composite tools:<br>SGA/MIS |
|                                                                                                                                          |                                                                                                                                        |                                                                        |                                                 |                                                                                                                                                                           |
| Infants                                                                                                                                                                                                                     | Toddlers                                                                                                                                                                                                                  | Children                                                                                                                                                  | Adolescents                                                                                                                        | Adults                                                                                                                                                                                                                                                        |
| CKD2-5D                                                                                                                                                                                                                     | CKD 2-5D                                                                                                                                                                                                                  | CKD 2-5D                                                                                                                                                  | CKD2-5D                                                                                                                            | CKD1-5D                                                                                                                                                                                                                                                       |
| Note: Frequency of monitoring varies with age and stage of CKD in children and varies with stage of CKD in adults.<br># After excluding non- nutritional causes for hypoalbuminemia<br>*Maintenance hemodialysis<br>^ CKD5D |                                                                                                                                                                                                                           |                                                                                                                                                           |                                                                                                                                    |                                                                                                                                                                                                                                                               |

Ref: Mak, R. H., Iyengar, A., & Wang, A. Y. (2023). Nutrition Management for Chronic Kidney Disease: Differences and Special Needs for Children and Adults. *Seminars in nephrology*, 43(4), 151441. <https://doi.org/10.1016/j.semnephrol.2023.151441>

Figure 50: Recommended Assessment Tools for Nutrition Status in CKD Patients across the Life Cycle

Table 34: Summary of Types of Objective Nutrition Assessment for Adults

| No. | Guideline Topic               | CKD Stage                | Guideline Statement                                                                                                                                                                                                           |
|-----|-------------------------------|--------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1.  | Bioimpedance                  | MHD                      | Suggest using BIA, preferable multi-frequency BIA to assess body composition if available; perform > 30 min after end of HD session                                                                                           |
|     |                               | CKD, PD                  | Insufficient evidence to suggest use of BIA                                                                                                                                                                                   |
| 2.  | DXA*                          | 1-5D,<br>Post-Transplant | Reasonable to use DXA when feasible (Gold standard for measuring body composition)                                                                                                                                            |
| 3.  | BW/BMI                        | Same as above            | Reasonable to consider assessing body composition in combination with BW/BMI at 1st visit and periodically over time                                                                                                          |
| 4.  | Frequency of BW/<br>BMI       | Same as above            | For clinically stable measure/monitor changes at least: <ul style="list-style-type: none"> <li>Monthly (MHD, PD)</li> <li>Every 3 months (CKD 4-5, Post-Transplant)</li> <li>Every 6 months (CKD 1-3)</li> </ul>              |
| 5.  | Body Weight                   | Same as above            | Reasonable to use clinical judgement to determine method for measuring BW (actual, WT history, serial WT measures, adjustments for oedema, ascites, and polycystic kidney disease) due to absence of standard reference norms |
| 6.  | BMI as predictor of mortality | PD                       | Suggest underweight status based on BMI be used to predict higher mortality                                                                                                                                                   |

\*DXA....

Note: Adapted from McCann, 2021, National Kidney Foundation.

Table 35: Evaluation of Growth and Nutritional Status in Children with CKD

| Area of Guidance                 | Key Recommendations                                                                                            | Details / Parameters to Monitor                                                                                                                                                                                                                                                                                                                                                                                                                                                    |
|----------------------------------|----------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Routine Evaluation</b>        | Children with CKD stages 2–5 and 5D should undergo regular monitoring of nutritional status and growth.        | <ul style="list-style-type: none"> <li>Assessments should be scheduled periodically across all CKD stages.</li> </ul>                                                                                                                                                                                                                                                                                                                                                              |
| <b>Parameters for Assessment</b> | Growth and nutrition should be evaluated using a combination of dietary, anthropometric, and clinical markers. | <ul style="list-style-type: none"> <li>Dietary intake (3-day food record or three 24-h recalls)</li> <li>Height/length-for-age percentile or SDS</li> <li>Height/length velocity-for-age percentile or SDS</li> <li>Estimated dry weight &amp; weight-for-age percentile or SDS</li> <li>BMI-for-height-age percentile or SDS</li> <li>Head circumference-for-age percentile or SDS (<math>\leq 3</math> yrs)</li> <li>nPCR (in hemodialyzed adolescents with stage 5D)</li> </ul> |
| <b>Frequency of Monitoring</b>   | The frequency of assessments should depend on age, CKD stage, and clinical status.                             | <ul style="list-style-type: none"> <li>At least twice as often as in healthy children of the same age.</li> <li>More frequent checks needed for: infants, polyuric children, those with growth delay, low/decreasing BMI, comorbidities, or recent acute illness/changes in diet.</li> </ul>                                                                                                                                                                                       |

Adapted from KDOQI Work Group. (2009). KDOQI clinical practice guideline for nutrition in children with chronic kidney disease: 2008 update. American Journal of Kidney Diseases, 53(3, Suppl. 2), S11–S104. <https://doi.org/10.1053/j.ajkd.2008.11.017>

## 12.5 Evaluation of Nutrition in CKD

A thorough evaluation of patients with chronic kidney disease (CKD) calls for a structured multidimensional approach that integrates several key components of assessment. This includes clinical examination, biochemical evaluation, and dietary assessment to monitor kidney function, detect complications early, guide therapeutic decisions, and optimize overall outcomes. The table below XXX outlines the key domains, components, and essential parameters/tools used in routine CKD evaluation across different levels of care.

Table 36: Clinical and Biochemical Evaluation of CKD

| Component                                     | Parameters / Tools                                                                                                                                                                                                                                        |
|-----------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>A. General Examination</b>                 |                                                                                                                                                                                                                                                           |
| <b>i. Clinical assessment</b>                 | <ul style="list-style-type: none"> <li>• Weight</li> <li>• BMI</li> <li>• Blood Pressure</li> </ul>                                                                                                                                                       |
| <b>B. Biochemical Evaluation</b>              |                                                                                                                                                                                                                                                           |
| <b>i. Kidney Function Tests</b>               | <ul style="list-style-type: none"> <li>• Serum Creatinine</li> <li>• Cystatin C</li> <li>• Estimated GFR (eGFR)</li> <li>• Blood Urea Nitrogen (BUN)</li> <li>• Urine Albumin-Creatinine Ratio (uACR) or Urine Protein-Creatinine Ratio (uPCR)</li> </ul> |
| <b>ii. Mineral &amp; Acid–Base Balance</b>    | <ul style="list-style-type: none"> <li>• Potassium</li> <li>• Sodium</li> <li>• Bicarbonate / PCO<sub>2</sub></li> </ul>                                                                                                                                  |
| <b>iii. Nutrition Biochemical Markers</b>     | <ul style="list-style-type: none"> <li>• Serum Albumin</li> <li>• nPNA (Normalized Protein Nitrogen Appearance)</li> </ul>                                                                                                                                |
| <b>iv. Anaemia in CKD</b>                     | <ul style="list-style-type: none"> <li>• Hemoglobin &amp; Hematocrit</li> <li>• Ferritin &amp; Transferrin Saturation</li> <li>• Mean Corpuscular Volume</li> </ul>                                                                                       |
| <b>v. Mineral and Bone Disorder (CKD-MBD)</b> | <ul style="list-style-type: none"> <li>• Parathyroid Hormone (PTH)</li> <li>• Calcium &amp; Phosphorus</li> <li>• Vitamin D</li> </ul>                                                                                                                    |
| <b>vi. Cardiovascular Health</b>              | <ul style="list-style-type: none"> <li>• Cholesterol Panel</li> <li>• Hemoglobin A1C</li> <li>• Blood Glucose</li> </ul>                                                                                                                                  |

## 12.6 Dietary Assessment

The following may be used to determine the dietary intake of patients with CKD:

- 3-day food records
- 24-hour food recalls
- Food frequency questionnaires
- Normalized protein catabolic rate (nPCR)
- Normalized protein nitrogen appearance (nPNA)

## 12.7 Nutritional Diagnosis and Nutrition Assessment Scoring Tools

Protein–energy wasting (PEW) is the state of decreased body stores of protein and energy fuels (that is, body protein and fat masses).

Tools used in identifying Protein Energy Wasting risk in patients on haemodialysis are:

1. 7-point SGA
2. Protein Energy Wasting (PEW) score
3. NutrE Using ISRNM Diagnostic Criteria for PEW
4. The ISRNM Diagnostic Criteria for Protein–Energy Wasting (PEW) in CKD
5. Malnutrition Inflammation Score
6. Geriatric Nutritional Risk Index (GNRI)

### 12.7.1 The 7-point SGA

Has subjective ratings of 6 components to reflect nutritional status:

1. Weight change
2. Dietary intake
3. Gastrointestinal symptoms
4. Functional capacity
5. Disease state/comorbidities
6. Physical examination

*Table 37: Overall SGA Rating*

| SGA Rating                         | Score       | Category           |
|------------------------------------|-------------|--------------------|
| Well-nourished                     | 6 or 7      | Not at risk of PEW |
| Moderate or suspected malnourished | 5 or 4 or 3 | At risk of PEW     |
| Severely malnourished              | 2 or 1      | At risk of PEW     |

Table 38: Overall SGA Scoring

| SGA Rating       | Score  |
|------------------|--------|
| Well Nourished   | 4      |
| Slightly Wasted  | 3      |
| Moderate Wasting | 2      |
| Severe Wasting   | 0 or 1 |

## 12.7.2 The protein energy wasting score (PEW Score)

The 4 Components for the PEW Score are in Table 39:

Table 39: Components of the PEW Score

| Component                                                     | Value                     |
|---------------------------------------------------------------|---------------------------|
| Serum albumin level                                           | 3.8 g/dL                  |
| Body mass index (BMI)                                         | 23 kg/m <sup>2</sup>      |
| Pre-dialysis serum Creatinine normalized by body surface area | 380 mmol/L/m <sup>2</sup> |
| Normalized protein nitrogen appearance                        | 0.8 g/kg/day              |

## 12.7.3 NutrE using ISRNM diagnostic criteria for PEW

- NutrE is a common clinical practice tool to determine the PEW risk of a patient
- ISRNM diagnostic criteria in NutrE facilitates confirmation of the PEW risk in stage 5 CKD patients on MHD

Table 40: Parameters for the NutrE Evaluation

| No. | Nutrition Assessment Domain in Nutrition        | Parameters in NutrE                                                                                                                                                                    |
|-----|-------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1.  | Food/Nutrition-related History                  | Dietary intake data (on both dialysis and non-dialysis days)                                                                                                                           |
| 2.  | Anthropometric measurements and Vital Signs     | Height, Weight, Dry weight, Average Interdialytic Weight gain, Blood Pressure                                                                                                          |
| 3.  | Biochemical Data, Medical Tests, and Procedures | Serum Albumin, Blood Urea Nitrogen, Normalized Protein Catabolic Rate, C-reactive Protein, White Blood Cells, Hemoglobin, Intact Parathyroid Hormone, Hemoglobin A1C, Serum Creatinine |
| 4.  | Nutrition-focused physical findings             | Nutrition-Specific Quality of Life (NSQOL) Questionnaire results, Subjective Global Assessment (SGA) Subcomponents (Functional Capacity, Physical Examination)                         |
| 5.  | Client History                                  | Demographic Characteristics (Age, Gender, Race, Ethnicity and Chronic Kidney Disease Etiology)                                                                                         |

- Evaluations of each nutrition assessment domain and diagnostic criterion guides in clinical decisions to assign the patient as either “not at PEW risk” or “at PEW risk.”

## 12.7.4 The ISRNM diagnostic criteria for protein–energy wasting (PEW) in CKD

| Category               | Threshold                                                                                                                                                                                                                                                                                                       |
|------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Biochemical indicators | <ul style="list-style-type: none"> <li>- Serum Albumin &lt; 3.8 g/dL*</li> <li>- Serum Prealbumin (transthyretin) &lt; 30 mg/dL*</li> <li>- Serum Cholesterol &lt; 100 mg/dL*</li> </ul>                                                                                                                        |
| Body mass              | <ul style="list-style-type: none"> <li>- BMI &lt; 23 kg/m<sup>2</sup></li> <li>- Unintentional weight loss: 5% over 3 months or 10% over 6 months†</li> <li>- Total body fat percentage &lt; 10%†</li> </ul>                                                                                                    |
| Muscle mass            | <ul style="list-style-type: none"> <li>- Muscle wasting: reduced muscle mass (≥ 5% over 3 months or ≥ 10% over 6 months)</li> <li>- Reduced mid-arm muscle circumference area‡ (≥10% reduction compared to the 15th percentile of reference population)</li> <li>- Appearance of reduced creatinine§</li> </ul> |
| Dietary intake         | <ul style="list-style-type: none"> <li>- Unintentional low DPI: &lt;0.8 g/kg/day for ≥2 months in Haemodialysis patients; &lt;0.6 g/kg/day for CKD stage 2–5 patients</li> <li>- Unintentional low DEI: &lt;25 kcal/kg/day for ≥2 months<sup>k</sup></li> </ul>                                                 |

## 12.7.5 Malnutrition inflammation score

- The Malnutrition–Inflammation Score (MIS) consists of 10 elements that capture different dimensions of the malnutrition–inflammation complex
- Seven of these overlap with the Subjective Global Assessment (SGA) and include:
  - o weight change
  - o dietary intake
  - o gastrointestinal symptoms
  - o functional status
  - o comorbidities
  - o fat stores
  - o muscle wasting
- The remaining three are unique to the MIS:
  - o body mass index
  - o serum albumin
  - o total iron-binding capacity
- Each item is graded on a four-point scale, from 0 (normal) to 3 (severely impaired)
- When summed, the total score ranges from 0, indicating normal status, to 30, representing severe malnutrition and inflammation.

Table 41: Components of the Malnutrition–Inflammation Score

| MIS Components                                                                                             | Score                                                |                                                                             |                                                                        |                                                     |
|------------------------------------------------------------------------------------------------------------|------------------------------------------------------|-----------------------------------------------------------------------------|------------------------------------------------------------------------|-----------------------------------------------------|
|                                                                                                            | 0                                                    | 1                                                                           | 2                                                                      | 3                                                   |
| <b>(A) Medical History:</b>                                                                                |                                                      |                                                                             |                                                                        |                                                     |
| 1. Change in end dialysis dry weight (overall change in the past 3–6 months)                               | <0.5 Kg                                              | 0.5–1.0 Kg                                                                  | ≥1 Kg but <5%                                                          | ≥ 5%                                                |
| 2. Dietary intake                                                                                          | Good appetite, no deterioration of dietary intake    | Sub-optimal solid dietary intake                                            | Moderate overall decrease to full liquid diet                          | Hypo-caloric liquid to starvation                   |
| 3. Gastrointestinal symptoms                                                                               | No symptoms with good appetite                       | Mild symptoms, poor appetite or nauseated occasionally                      | Occasional vomiting or moderate GI symptoms                            | Frequent diarrhea or vomiting or severe anorexia    |
| 4. Functional capacity (nutritionally related functional impairment)                                       | Normal to improved functional capacity, feeling fine | Occasional difficulty with baseline ambulation, or feeling tired frequently | Difficulty with otherwise independent activities (e.g. going bathroom) | Bed/chair ridden, or little to no physical activity |
| 5. Co-morbidity *                                                                                          | No comorbidity                                       | Mild comorbidity (excluding MCC)                                            | Moderate comorbidity (including one MCC)                               | Any severe multiple comorbidity (≥2 MCC)            |
| <b>(B) Physical exam:</b>                                                                                  |                                                      |                                                                             |                                                                        |                                                     |
| 6. Decreased fat stores or loss of subcutaneous fat (below eyes, triceps, biceps, triceps, chest)          | No change                                            | mild                                                                        | moderate                                                               | sever                                               |
| 7. Signs of muscle wasting (temple, clavicle, scapula, ribs, quadriceps, knee, interosseous)               | No change                                            | mild                                                                        | moderate                                                               | sever                                               |
| <b>(C) Body size</b>                                                                                       |                                                      |                                                                             |                                                                        |                                                     |
| 8. Body mass index (kg/m <sup>2</sup> )                                                                    | ≥ 20                                                 | 18–19.9                                                                     | 16–17.99                                                               | <16                                                 |
| <b>(D) Laboratory parameters</b>                                                                           |                                                      |                                                                             |                                                                        |                                                     |
| 9. Serum albumin (g/L)                                                                                     | ≥4                                                   | 3.5–3.9                                                                     | 3.–3.4                                                                 | <3.0                                                |
| 10. Serum total iron binding capacity (mg/dL)                                                              | ≥ 250                                                | 200–249                                                                     | 150–199                                                                | <150                                                |
| Total MIS = Sum of the above 10 components, ranging from 0 (no malnutrition) to 30 (severely malnourished) |                                                      |                                                                             |                                                                        |                                                     |

Adapted from Rambod, Bross, Zitterkoph, Benner, Pithia, Colman, Kovesdy, Kopple, & Kalantar-Zadeh, 2008

## 12.7.6 Geriatric nutritional risk index (GNRI)

- Nutritional status should be done at the first hospital visit
- $GNRI = 14.89 \times \text{serum albumin (g/dL)} + 41.7 \times (\text{current body weight} \div \text{ideal body weight})$   
where  
the weight ratio capped at 1  
ideal body weight is derived from height, assuming a BMI of 22 kg/m<sup>2</sup>.
- Patients should then be classified into 4 risk categories:
  - no risk (GNRI > 98)
  - low risk (92–98)
  - moderate risk (82–91)
  - high risk (< 82)

## 12.8 Nutritional intervention

### 12.8.1 Nutrition consultation

- Optimum nutrition status for CKD patients is essential as it is linked with reduced disease progression therefore improving or maintaining kidney function and also preventing mortality.
- It is essential that all patients with CKD above stage 3 should have nutrition consultation during each hospital appointment to improve quality of care.

### 12.8.2 Protein and energy intake in adults with CKD

| Topic                                                | Recommendation / Summary                                                                                                                                                                                                                                            |
|------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Energy Intake                                        | <ul style="list-style-type: none"><li>25–35 kcal/kg ideal body weight/day for CKD stages 1–5D and post-transplant.</li><li>Adjust based on age, sex, physical activity, body composition, weight goals, CKD stage, inflammation, and co-existing illness.</li></ul> |
| Protein Restriction (CKD 3–5, Non-Dialysis)          | <ul style="list-style-type: none"><li>Low-protein diet: 0.55–0.60 g/kg/day</li><li>Very low-protein diet: 0.28–0.43 g/kg/day + keto acid analogs (to meet 0.55–0.60 g/kg/day).</li><li>Purpose: slow CKD progression, lower mortality, improve QoL.</li></ul>       |
| Protein Intake (Haemodialysis & Peritoneal Dialysis) | <ul style="list-style-type: none"><li>Stable adults on MHD or PD: 1.0–1.2 g/kg/day to maintain nutritional stability.</li></ul>                                                                                                                                     |
| Protein Intake (CKD with Diabetes)                   | <ul style="list-style-type: none"><li>Non-dialysis CKD 3–5: 0.8–0.9 g/kg/day (balance nutrition + glycemic control).</li><li>Dialysis + diabetes: 1.0–1.2 g/kg/day.</li><li>If glycemic instability present, consider higher protein intake.</li></ul>              |
| Dietary Patterns – Mediterranean Diet                | <ul style="list-style-type: none"><li>May improve lipid profile in CKD 1–5 (non-dialysis) and post-transplant patients.</li></ul>                                                                                                                                   |
| Dietary Patterns – Fruits & Vegetables               | <ul style="list-style-type: none"><li>In CKD stages 1–4: higher intake may reduce weight, blood pressure, and net acid production (NEAP).</li></ul>                                                                                                                 |

### 12.8.3 Protein and energy intake in dialysis

Table 42: Summary of Comparison of HD and PD Nutrients

| Nutrient             | Haemodialysis (HD)        | Peritoneal Dialysis (PD)           |
|----------------------|---------------------------|------------------------------------|
| Energy (kcal/kg/day) | 30–35                     | 30–35 (+glucose from dialysate)    |
| Protein (g/kg/day)   | 1.0–1.2                   | 1.2–1.3                            |
| Potassium (mg/day)   | ≤ 2000 (individualised)   | Variable, monitor losses           |
| Phosphorus (mg/day)  | 800–1000                  | 800–1000                           |
| Sodium (mg/day)      | 2000–2300                 | 2000–2300                          |
| Fluid                | 300–500 mL + urine output | Variable, based on ultrafiltration |

Table 43: Caloric Contribution from Dextrose in Peritoneal Dialysis Solutions

| % Dextrose | g Dextrose/L | kcal/L (g × 3.4) | Available CAPD kcal Absorbed | Available CCPD kcal Absorbed |
|------------|--------------|------------------|------------------------------|------------------------------|
| 1.5%       | 15           | 51               | 31–36 kcal/L                 | 20–26 kcal/L                 |
| 2.5%       | 25           | 85               | 51–60 kcal/L                 | 34–43 kcal/L                 |
| 4.25%      | 42.5         | 145              | 87–102 kcal/L                | 58–73 kcal/L                 |

## 12.8.4 Nutritional requirements in acute kidney injury

| Nutrient Category                      | Recommended Requirements / Considerations                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |
|----------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Energy</b>                          | 20–30 kcal/kg/day (not exceeding 25 kcal/kg/day in critically ill patients). Use habitual or ideal body weight. Indirect calorimetry preferred for accuracy. Avoid overfeeding due to risk of hyperglycemia, hypertriglyceridemia, and delayed renal recovery.                                                                                                                                                                                                                                                                                                                    |
| <b>Proteins</b>                        | <ul style="list-style-type: none"> <li>AKI without KRT: 0.8–1 g/kg/day</li> <li>AKI on Intermittent Haemodialysis (IHD): 1–1.3 g/kg/day</li> <li>AKI on Continuous/Prolonged KRT (CKRT/PIKRT): 1.5–1.8 g/kg/day (+0.2 g/kg/day for dialysis-related amino acid loss).</li> </ul> <p>Combination of essential and non-essential amino acids recommended. Very high protein (&gt;2.2 g/kg/day) may worsen outcomes.</p>                                                                                                                                                             |
| <b>Carbohydrates</b>                   | 3–5 g/kg/day (maximum 7 g/kg/day). Provide ≥150 g/day to support glucose-dependent tissues (brain, RBCs, renal medulla). Maintain glucose target 110–180 mg/dL. Avoid exceeding 5 mg/kg/min infusion. Monitor for insulin resistance and hyperglycemia.                                                                                                                                                                                                                                                                                                                           |
| <b>Lipids</b>                          | 0.8–1 g/kg/day (30–35% of non-protein energy). In PN: administer via 20% emulsions over 18–24h. Stop if triglycerides >400 mg/dL. Medium-chain triglycerides (MCT/LCT) blends may help reduce triglycerides but no clear clinical advantage. No lipid loss occurs via KRT.                                                                                                                                                                                                                                                                                                        |
| <b>Micronutrients (Trace Elements)</b> | Losses occur during CKRT, especially selenium, zinc, chromium, and copper. Supplementation may be necessary. Zinc balance often positive due to PN and replacement fluids. Selenium frequently lost → negative balance.                                                                                                                                                                                                                                                                                                                                                           |
| <b>Vitamins</b>                        | Water-soluble vitamins (C, thiamine, folate) lost in CKRT. Losses: Vitamin C up to 100 mg/day, folate 600 nmol/day, thiamine >1.5x daily intake. Supplementation needed: Vitamin C 50–100 mg/day (up to 200 mg in CKRT). Avoid excess due to oxalosis risk. Fat-soluble vitamin supplementation usually not required.                                                                                                                                                                                                                                                             |
| <b>Electrolytes</b>                    | <ul style="list-style-type: none"> <li>Sodium &amp; Potassium: Common abnormalities (hypo/hyponatremia, hyperkalemia); corrected with dialysis fluid composition.</li> <li>Phosphate: High risk of hypophosphatemia in CKRT → IV phosphate supplementation or phosphate-containing dialysis fluids recommended.</li> <li>Magnesium: Hypomagnesemia frequent during CKRT with citrate anticoagulation; requires MgSO<sub>4</sub> infusion (2–4 g/day).</li> <li>Calcium: Imbalances depend on dialysis fluid composition and citrate use. Careful monitoring essential.</li> </ul> |

Adapted from Di Mario, Sabatino & Fiaccadori, 2024

## 12.8.5 Nutritional supplementation in adults with CKD

| Supplement Type                                     | Recommendation / Summary                                                                                                                                                                                                                                                                                                                                                     |
|-----------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Oral Protein–Energy Supplements</b>              | Adults with CKD stages 3–5D or post-transplant patients who have, or are at risk of, protein-energy wasting should be considered for at least a 3-month trial of oral nutritional supplements if dietary counseling alone does not provide adequate protein and energy intake.                                                                                               |
| <b>Enteral Nutrition (Tube Feeding)</b>             | For CKD stages 1–5D patients with persistently inadequate intake where requirements cannot be met by counseling or oral supplements, a trial of enteral tube feeding may be appropriate.                                                                                                                                                                                     |
| <b>Parenteral Nutrition (IDPN/TPN/AA Dialysate)</b> | <p>In adults on maintenance haemodialysis (MHD) with protein-energy wasting, IDPN may be trialed.</p> <p>For CKD patients unable to meet needs otherwise, TPN can be considered, while amino acid dialysate may be used for peritoneal dialysis (PD) patients. These options aim to improve and sustain nutritional status when oral and enteral intake is insufficient.</p> |

Adapted from Ikizler et al., 2019, National Kidney Foundation & Academy of Nutrition and Dietetics

## 12.8.6 Other dietary recommendations in adults with CKD

| Nutrient / Electrolyte / Fatty Acid | Key Guidance                                                                                                                                                                                                                                                                                                                                                                                                                                  | Suggested Intake / Range                                   |
|-------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------|
| <b>General Micronutrients</b>       | CKD 3–5D or post-transplant patients meet RDAs for vitamins and minerals. Periodic assessment is advised; multivitamins may be considered if intake is insufficient. Dialysis patients with chronic low intake may need supplements.                                                                                                                                                                                                          | Based on RDA for age/sex (varies by nutrient).             |
| <b>Folic Acid</b>                   | <ul style="list-style-type: none"> <li>Do not routinely supplement for hyperhomocysteinemia (no CV benefit).</li> <li>Folate, B12, or B-complex may be prescribed if deficiency is present.</li> </ul>                                                                                                                                                                                                                                        | Only if deficiency; individualised dosing.                 |
| <b>Vitamin C</b>                    | Supplement if risk of deficiency to meet daily needs.                                                                                                                                                                                                                                                                                                                                                                                         | Men ≥90 mg/day;<br>Women ≥75 mg/day                        |
| <b>Vitamin D</b>                    | <ul style="list-style-type: none"> <li>Use cholecalciferol/ergocalciferol to correct deficiency.</li> <li>In nephrotic proteinuria, consider vitamin D precursors.</li> </ul>                                                                                                                                                                                                                                                                 | Correct low 25(OH)D; dose individualised.                  |
| <b>Vitamins A &amp; E</b>           | Avoid routine supplementation in dialysis due to toxicity risk. If required, monitor closely.                                                                                                                                                                                                                                                                                                                                                 | Avoid high doses; monitor serum levels.                    |
| <b>Vitamin K</b>                    | Avoid supplements in patients on anticoagulants (e.g., warfarin).                                                                                                                                                                                                                                                                                                                                                                             | Contraindicated with anticoagulants.                       |
| <b>Selenium &amp; Zinc</b>          | Routine use not recommended; limited benefit evidence.                                                                                                                                                                                                                                                                                                                                                                                        | Not routinely prescribed.                                  |
| <b>Acid Load (NEAP)</b>             | <ul style="list-style-type: none"> <li>CKD 1–4: More fruits/vegetables to reduce acid load and slow CKD progression.</li> <li>CKD 3–5D: Use bicarbonate or citrate supplements; maintain bicarbonate at 24–26 mmol/L.</li> </ul>                                                                                                                                                                                                              | Serum bicarbonate 24–26 mmol/L                             |
| <b>Calcium</b>                      | <ul style="list-style-type: none"> <li>CKD 3–4: Total 800–1000 mg/day from diet + supplements.</li> <li>CKD 5D: Adjust based on vitamin D analog/calcimimetic use to avoid hypercalcemia.</li> </ul>                                                                                                                                                                                                                                          | 800–1000 mg/day (CKD 3–4); adjust in CKD 5D.               |
| <b>Phosphorus</b>                   | <ul style="list-style-type: none"> <li>CKD 3–5D: Adjust intake to keep phosphate normal. Total 800–1000 mg/day from diet + supplements.</li> <li>Consider source bioavailability.</li> <li>Post-transplant hypophosphatemia: Increase phosphorus via diet or supplements.</li> </ul>                                                                                                                                                          | 800–1000 mg/day<br>Maintain 2.5–4.5 mg/dL                  |
| <b>Potassium</b>                    | <ul style="list-style-type: none"> <li>Adjust intake to keep serum potassium normal.</li> <li>Individualise intake/supplementation for hypo- or hyperkalemia.</li> </ul>                                                                                                                                                                                                                                                                      | Target Serum 3.5–5.0 mmol/L (<2g/day)                      |
| <b>Sodium</b>                       | <ul style="list-style-type: none"> <li>CKD 3–5, 5D, post-transplant: Restrict to control BP and fluid balance.</li> <li>CKD 3–5: Also reduces proteinuria.</li> <li>CKD 5D: Supports fluid control and dry weight.</li> </ul>                                                                                                                                                                                                                 | <100 mmol/day (<2.3 g/day)                                 |
| <b>Fatty Acids (LC n-3 PUFA)</b>    | <ul style="list-style-type: none"> <li>Do not routinely prescribe to reduce mortality or CV events in CKD 5D or post-transplant.</li> <li>CKD on HD and PD, Fatty Acids may be used to improve lipid profiles: <ul style="list-style-type: none"> <li>1.3–4 g/day lowers TG, LDL and raises HDL.</li> <li>~2 g/day reduces TG.</li> </ul> </li> <li>Not recommended solely to maintain vascular access or prevent graft rejection.</li> </ul> | 1.3–4 g/day (for lipid effects);<br><br>~2 g/day lowers TG |

# Chapter 13: Obesity Prevention and Treatment in Renal Disease (CKD)

## 13.1 Introduction

The prevalence of overweight and obesity continues to rise globally, as it does in Kenya, where rates are 4.2% in men and 17.3% in women (KDHS,2022). Obesity remains the greatest risk factor for chronic kidney disease (CKD). Excess adiposity has also been independently associated with an increased risk of hypertension, albuminuria, hyperfiltration and diabetes, conditions that further increase the risk of CKD. An increase in obesity, therefore, threatens to increase the health care burden in the management of CKD. Management of obesity requires more attention, as evidence already suggests that the presence of obesity in renal patients worsens outcomes, delays treatment, such as transplants due to eligibility issues, and complicates dialysis adequacy, thus compromising quality of life.

## 13.2 Renal Care for Obese Patients

1. Obesity screening should be incorporated in all treatment areas, and should include body composition analysis to help in:
  - Early diagnosis of sarcopenia and malnutrition, especially in older patients and those on dialysis.
  - Predicting mortality risk, which is often missed with BMI (in cases of overhydration and low lean mass)
  - Precise fluid management to prevent cases of overhydration
2. Individualised weight management of patients in the CKD pathway should be prioritised to meet the individual requirements of patients at every stage of the disease.
3. Appropriate physical activity that takes into consideration muscle preservation opportunities should be carefully incorporated, and emphasis should be given to gradual and sustainable weight loss.
4. Evidence-based pharmacological and surgical interventions should be accessible for treatment where an indication for use has been identified.
5. As opposed to blanket restriction on carbohydrate intake, meal planning should carefully balance intake by considering glycaemic control, potassium and phosphorus reduction rather than elimination of food.
6. Management should include at all times a multidisciplinary team with an experienced nephrologist, obesity physician, clinical dietitian, surgeon and a psychologist.

## 13.3 Pharmacotherapy in Renal and Metabolic Care Pathways

| Medication/Class                                      | Mechanism of Action                                                | Adverse effects                                                                                                                      | Key considerations                                                                                                                                                                                                                |
|-------------------------------------------------------|--------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Metformin                                             | Improves insulin sensitivity                                       | Longterm use maybe associated with Deficiency in Vit B12<br>GI distrubances                                                          | Discontinue in patients with eGFR < 30 mL/min                                                                                                                                                                                     |
| SGLT2 inhibitors                                      | Promotes glycosuria                                                | Frequent urination, UTI and yeast infection                                                                                          | Provides renal and cardiovascular protection                                                                                                                                                                                      |
| Phentermine 15mg/30mg<br>eGFR 15-29 mL/min (15mg/day) | Increases norepinephrine and energy expenditure                    | GI distress, insomnia, Anxiety, tremors                                                                                              | Do not use in pts with insomnia, hyperthyroidism and uncontrolled HTN<br><br>Do not use when eGFR < 15 mL/min                                                                                                                     |
| Bupropion 90mg/<br>Naltroxene 8mg                     | Increases activity in the melanocortin system of the hypothalamus. | Risk of seizures, Insomnia, GI disturbance                                                                                           | Do not exceed a BD dose and watch out for toxicity.                                                                                                                                                                               |
| Topiramate 25-100mg daily                             | Unknown (antiepileptic drug)                                       | Paraesthesia, confusion, fetal abnormalities                                                                                         | Do not use in pregnancy or for patients with a history of kidney stones.                                                                                                                                                          |
| Orlistat 120mg<br>Requires no dose adjustment in CKD  | o Fat malabsorption<br>o Pancreatic & gastric lipase inhibitor     | o Faecal incontinence & steatorrhea<br>o Can promote gall & kidney stones<br>o Malabsorption of fat-soluble vitamins<br>o Flatulence | Do not use in Chronic malabsorption syndrome or cholestasis.<br><br>May cause Liver injury & Pancreatitis.<br><br>drug interaction with warfarin, oral contraceptives, Cyclosporine, thyroid hormones, & anti-seizure medications |

|                                                                  |                       |             |                                                                                                                                                                                                                                                                                                                                         |
|------------------------------------------------------------------|-----------------------|-------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Liraglutide 0.6mg SC injection daily but not exceeding 3mg daily | GLP1 Receptor agonist | GI distress | Do not use in patients with eGFR < 30 mL/min<br><br>Contraindicated with personal or family h/o medullary thyroid carcinoma, or Type 2 multiple endocrine neoplasia syndrome.<br><br>Stop with suspected pancreatitis/gall bladder disease/suicidal ideation.<br><br>Discontinue if volume depletion occurs (vomiting, diarrhoea, etc.) |
| Semaglutide 0.25mg, 0.5 mg, 1mg once weekly injectables          | GLP1 Receptor agonist | GI distress |                                                                                                                                                                                                                                                                                                                                         |
| Tirzepatide 2.5mg, 5mg, 2.5mg, 7.5mg once weekly injectables     | Dual Incretin Agonist | GI distress |                                                                                                                                                                                                                                                                                                                                         |

*Adapted from WHO guidelines on GLP use, obesity med. assoc*

## 13.4 Surgical Intervention in Obese Renal Patients

Growing evidence continues to support the use of Bariatric/Metabolic surgery for weight management in obese patients with renal disease as a safe and effective intervention. It is, however, recommended that such an intervention be carefully planned in a multi-disciplinary renal team together with a metabolic and surgical team for optimal results.

Sleeve gastrectomy and Roux-en-Y remain the most popular procedures as they are associated with a greater reduction in weight, enabling especially transplant candidates to achieve an admissible BMI for a safe transplant procedure.

In dialysis patients as well, bariatric surgery helps reverse comorbidities such as diabetes and Hypertension, and lowers hyperfiltration, which in turn reduces systemic inflammation, thereby slowing the progression of kidney disease. Roux-en-Y, although associated with increased muscle catabolism, thus a risk of sarcopenia as well as long-term calcium and iron deficiencies requiring rigorous and lifelong monitoring, is still a beneficial surgical procedure with significant and sustainable weight loss.

Intragastric Balloon offers moderate and effective weight loss with minimally invasive technology and has also been shown to provide a safe, effective, and relatively affordable intervention for weight loss in renal patients compared to surgical options.

## 13.5 Surgical Considerations

For all renal patients meeting the criteria for surgical interventions for weight management, the following factors must be considered beyond the BMI cutoffs:

- A comprehensive micronutrient assessment must be conducted before the procedure, and monitoring post-operatively must be done.
- A comprehensive renal nutrition assessment preoperatively to establish gaps that require attention.
- Body composition analysis to establish muscle mass and employ strategies for sarcopenia prevention.
- Availability of long-term nutrition follow-up by a registered and experienced dietitian.

# Chapter 14: Mental Health Support for Kidney Patients

## 14.1 Introduction

- Kidney disease is a chronic and life-altering condition associated with significant psychological, social, and emotional burden.
- Mental health support is a core component of holistic renal care.
- Addressing psychosocial needs improves treatment adherence, clinical outcomes, quality of life, and family functioning.
- Renal counselling is a structured therapeutic approach aimed at supporting patients and their families throughout the disease continuum—from diagnosis to dialysis and transplantation.

## 14.2 Objectives of Mental Health Support in Kidney Disease

Mental health and psychosocial support aims to:

- Promote psychological well-being among kidney patients.
- Identify and manage mental health challenges early.
- Enhance adherence to medication, diet, dialysis schedules, and transplant management.
- Support family members and caregivers to prevent burnout.
- Prepare patients for renal replacement therapy (RRT) and associated procedures.
- Facilitate patient self-management, coping skills, and problem solving.
- Address psychosocial barriers that may affect engagement in care.

## 14.3 Components of Psychosocial Support for Kidney Patients

| Component of support                         | Scope                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |
|----------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Patient and Family Education                 | <ul style="list-style-type: none"> <li>• Creating awareness about kidney disease and its progression.</li> <li>• Early counseling on renal replacement therapy options.</li> <li>• Education on vascular access preparation.</li> <li>• Preparing patients and families for dialysis initiation.</li> <li>• Counseling on the financial implications of kidney disease management.</li> <li>• Guidance on procedures including kidney biopsy, AVF creation, HD/PD catheter insertion, and dialysis processes.</li> <li>• Counselling for transplant preparation and post-transplant care.</li> <li>• Navigating life adjustments after initiating dialysis.</li> </ul> |
| Treatment Support                            | <ul style="list-style-type: none"> <li>• Promoting treatment adherence (drugs, diet, dialysis, lifestyle).</li> <li>• Supporting continuous problem-solving as needs arise.</li> <li>• Encouraging patient self-care and self-management.</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                   |
| Psychosocial and Mental Health Interventions | <ul style="list-style-type: none"> <li>• Identification of psychosocial challenges.</li> <li>• Individual counselling to promote mental wellness.</li> <li>• Family conferences for shared decision-making.</li> <li>• Caregiver support to prevent burnout.</li> <li>• End-of-life discussions and advanced care planning where appropriate.</li> </ul>                                                                                                                                                                                                                                                                                                               |
| Collaboration and Multidisciplinary Care     | <ul style="list-style-type: none"> <li>• A multidisciplinary team should include:</li> <li>• Nephrologists</li> <li>• Renal nurses</li> <li>• Clinical psychologists</li> <li>• Psychiatrists</li> <li>• Social workers</li> <li>• Nutritionists</li> <li>• Counsellors</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                     |

## 14.4 Psychological Issues Associated with Chronic Kidney Disease in Children

### 14.4.1 psychological and social effects of kidney disease in children

Children with kidney disease experience psychological issues that affect their mental wellbeing

A. When children are distressed:

- o their level of interaction with other children is affected
- o their quality of life is affected
- o their image and self-esteem is lowered

B. When children experience challenges:

- o their level of adherence to treatment is low
- o they are likely to be delinquent
- o somatic illness can increase

### 14.4.2 Issues affecting children

- **Denial**
  - o This will lead to poor adherence to drug, diet, dialysis and even transplantation
- **Uraemia**
  - o High urea will cause change in skin color and pruritus which affect their esteem and body image.
  - o It may also cause low concentration, confusion and delirium
  - o Poor grades may lead to low self-esteem
- **Frequent hospitalization**
  - o This may lead to social isolation and feeling of loneliness and depression
- **Invasive procedures:**
  - o This may lead to fear anxiety and panic attack, may also cause poor self-image
- **Repeated routine**
  - o Dialysis routine and frequent medication and may cause burn out, leading to poor adherence
- **Blood sampling**
  - o May cause fear, anxiety and panic attack
- **Disease process**
  - o May cause somatic illness, burn out, insomnia, anger, depression
  - o It may also lead to behavior problem
- **Family dysfunction**
  - o If the family is in conflict, the child may be emotionally drained
- **Peritoneal dialysis**
  - o This may cause social isolation and limit ability to play with agetmates, causing more distress

## 14.5.2 Psychosocial intervention (psychotherapy)

### Psychosocial intervention

- Psychotherapy
- Family therapy
- School reintegration program
- Peer support group
- Collaboration and multi-disciplinary approach: physician, dietician, psychiatrist, social worker, clinical psychologist

## 14.5 Psychosocial Issues Associated with Chronic Kidney Disease in Adults

### 14.5.1 Issues affecting adults

- **Denial**
  - Refusal to accept the truth
  - This may delay initiation of treatment
  - May cause poor adherence
- **Anxiety**
  - Due to uncertainty, comorbidity, disease process, treatment process
- **Stress:**
  - Due to regular appointment, dietary restriction, financial burden, disease process, comorbidity
- **Cognitive impairment**
  - Patients may have poor memory, brain fog, low concentration and slow in decision making
  - This may be due to uremia
- **Anger**
  - May be due to loss of control, disease process, comorbidity, financial burden, unfinished business, routineness of treatment, guilt and shame
- **Depression**
  - Being in low mood
  - Due to uncertainty, comorbidity, disease process, routineness, life in general, sexual dysfunction, biochemical imbalance
- **Fatigue**
  - Due to anemia, uremia, dialysis process
- **Sexual dysfunction**
  - Due to hormonal changes, anemia, infection, fatigue, drugs, disease process
  - Stigma in disclosing sexual matter
  - This affects self-esteem

- **Family and relationship strain**
  - o Due caregiver burden and role changes, and financial strain
- **Employment and financial strain**
  - o Due to treatment schedule and high cost, loss of income
  - o This may lead to depression
- **Identity alteration**
  - o Due to altered body images and loss of identity
- **Insomnia**
  - o fear, worry and anxiety, high urea, fluid overload, disease process
- **Loss and grief**
  - o Due to loss of kidney function, disease process
- **Withdrawal from dialysis**
  - o Due to social economic status, burnout, depression, culture, religion, ethical and legal issues, lack of support
- **Panic attack**
  - o Due to invasive procedures causing worry and anxiety

### 14.5.2 Psychosocial intervention (psychotherapy)

- Support group
- Health education
- Selfcare strategies.
- Collaboration and multi-disciplinary approach

## 14.6 Mental Health Screening in Kidney Disease

Mental health challenges such as depression and anxiety are common in people living with kidney disease and can negatively affect treatment adherence and overall wellbeing. Integrating standardized mental health screening tools enables early detection and timely intervention for these conditions.

Facilities should incorporate standardized mental health screening tools such as:

- **Beck Depression Inventory (BDI)**
- **Patient Health Questionnaire (PHQ-9)**
- **Generalized Anxiety Disorder Scale (GAD-7)**

These tools allow early detection, monitoring, and referral for mental health conditions.

### 14.6.1 Beck depression inventory (BDI)

#### A. Overview of BDI

The Beck Depression Inventory (BDI) is a widely used self-report questionnaire designed to assess the presence and severity of depressive symptoms in adolescents and adults. It was developed by psychiatrist Aaron T. Beck, a pioneer in cognitive therapy. Table 44 presents an overview of the Beck Depression Inventory (BDI), while Table 45 outlines the corresponding BDI scoring tool.

| Purpose                                                                                                                                                                                                                                                                                                       | Structure                                                                                                                                                                                                                    | Key Features                                                                                                                                                                                                                                          |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <p>The BDI measures the cognitive, emotional, behavioral, and physical symptoms associated with depression. It is commonly used in:</p> <ul style="list-style-type: none"> <li>Clinical practice</li> <li>Research studies</li> <li>Mental health screening</li> <li>Monitoring treatment response</li> </ul> | <ul style="list-style-type: none"> <li>Consists of 21 items</li> <li>Each item has 4 response options (scored 0–3)</li> <li>Total score ranges from 0 to 63</li> <li>Takes approximately 5–10 minutes to complete</li> </ul> | <ul style="list-style-type: none"> <li>Simple and quick to administer</li> <li>Sensitive to changes over time (useful for follow-up)</li> <li>Not a diagnostic tool on its own — results should be interpreted by a qualified professional</li> </ul> |

Table 44: Beck Depression Inventory Tool

| Domain |                                                                  | Score |
|--------|------------------------------------------------------------------|-------|
|        |                                                                  |       |
| 1      | 0 I do not feel sad.                                             |       |
|        | 1 I feel sad                                                     |       |
|        | 2 I am sad all the time and I can't snap out of it.              |       |
|        | 3 I am so sad and unhappy that I can't stand it.                 |       |
|        |                                                                  |       |
| 2      | 0 I am not particularly discouraged about the future.            |       |
|        | 1 I feel discouraged about the future.                           |       |
|        | 2 I feel I have nothing to look forward to.                      |       |
|        | 3 I feel the future is hopeless and that things cannot improve.  |       |
|        |                                                                  |       |
| 3      | 0 I do not feel like a failure.                                  |       |
|        | 1 I feel I have failed more than the average person.             |       |
|        | 2 As I look back on my life, all I can see is a lot of failures. |       |
|        | 3 I feel I am a complete failure as a person.                    |       |
|        |                                                                  |       |
| 4      | 0 I get as much satisfaction out of things as I used to.         |       |
|        | 1 I don't enjoy things the way I used to.                        |       |
|        | 2 I don't get real satisfaction out of anything anymore.         |       |
|        | 3 I am dissatisfied or bored with everything.                    |       |
|        |                                                                  |       |
| 5      | 0 I don't feel particularly guilty                               |       |
|        | 1 I feel guilty a good part of the time.                         |       |
|        | 2 I feel quite guilty most of the time.                          |       |
|        | 3 I feel guilty all of the time.                                 |       |
|        |                                                                  |       |
| 6      | 0 I don't feel I am being punished.                              |       |
|        | 1 I feel I may be punished.                                      |       |

|    |                                                                                       |  |
|----|---------------------------------------------------------------------------------------|--|
|    | 2 I expect to be punished.                                                            |  |
|    | 3 I feel I am being punished.                                                         |  |
|    |                                                                                       |  |
| 7  | 0 I don't feel disappointed in myself.                                                |  |
|    | 1 I am disappointed in myself.                                                        |  |
|    | 2 I am disgusted with myself.                                                         |  |
|    | 3 I hate myself.                                                                      |  |
|    |                                                                                       |  |
| 8  | 0 I don't feel I am any worse than anybody else.                                      |  |
|    | 1 I am critical of myself for my weaknesses or mistakes.                              |  |
|    | 2 I blame myself all the time for my faults.                                          |  |
|    | 3 I blame myself for everything bad that happens.                                     |  |
|    |                                                                                       |  |
| 9  | 0 I don't have any thoughts of killing myself.                                        |  |
|    | 1 I have thoughts of killing myself, but I would not carry them out.                  |  |
|    | 2 I would like to kill myself.                                                        |  |
|    | 3 I would kill myself if I had the chance.                                            |  |
|    |                                                                                       |  |
| 10 | 0 I don't cry any more than usual.                                                    |  |
|    | 1 I cry more now than I used to.                                                      |  |
|    | 2 I cry all the time now.                                                             |  |
|    | 3 I used to be able to cry, but now I can't cry even though I want to.                |  |
|    |                                                                                       |  |
| 11 | 0 I am no more irritated by things than I ever was.                                   |  |
|    | 1 I am slightly more irritated now than usual.                                        |  |
|    | 2 I am quite annoyed or irritated a good deal of the time.                            |  |
|    | 3 I feel irritated all the time.                                                      |  |
|    |                                                                                       |  |
| 12 | 0 I have not lost interest in other people.                                           |  |
|    | 1 I am less interested in other people than I used to be.                             |  |
|    | 2 I have lost most of my interest in other people.                                    |  |
|    | 3 I have lost all of my interest in other people.                                     |  |
|    |                                                                                       |  |
| 13 | 0 I make decisions about as well as I ever could.                                     |  |
|    | 1 I put off making decisions more than I used to.                                     |  |
|    | 2 I have greater difficulty in making decisions more than I used to.                  |  |
|    | 3 I can't make decisions at all anymore.                                              |  |
|    |                                                                                       |  |
| 14 | 0 I don't feel that I look any worse than I used to.                                  |  |
|    | 1 I am worried that I am looking old or unattractive.                                 |  |
|    | 2 I feel there are permanent changes in my appearance that make me look unattractive. |  |
|    | 3 I believe that I look ugly.                                                         |  |
|    |                                                                                       |  |
| 15 | 0 I can work about as well as before.                                                 |  |
|    | 1 It takes an extra effort to get started at doing something.                         |  |
|    | 2 I have to push myself very hard to do anything.                                     |  |
|    | 3 I can't do any work at all.                                                         |  |

|    |                                                                                           |  |
|----|-------------------------------------------------------------------------------------------|--|
|    |                                                                                           |  |
| 16 | 0 I can sleep as well as usual.                                                           |  |
|    | 1 I don't sleep as well as I used to.                                                     |  |
|    | 2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.           |  |
|    | 3 I wake up several hours earlier than I used to and cannot get back to sleep.            |  |
|    |                                                                                           |  |
| 17 | 0 I don't get more tired than usual.                                                      |  |
|    | 1 I get tired more easily than I used to.                                                 |  |
|    | 2 I get tired from doing almost anything.                                                 |  |
|    | 3 I am too tired to do anything.                                                          |  |
|    |                                                                                           |  |
| 18 | 0 My appetite is no worse than usual.                                                     |  |
|    | 1 My appetite is not as good as it used to be.                                            |  |
|    | 2 My appetite is much worse now.                                                          |  |
|    | 3 I have no appetite at all anymore.                                                      |  |
|    |                                                                                           |  |
| 19 | 0 I haven't lost much weight, if any, lately.                                             |  |
|    | 1 I have lost more than five pounds.                                                      |  |
|    | 2 I have lost more than ten pounds.                                                       |  |
|    | 3 I have lost more than fifteen pounds.                                                   |  |
|    |                                                                                           |  |
| 20 | 0 I am no more worried about my health than usual.                                        |  |
|    | 1 I am worried about physical problems like aches, pains, upset stomach, or constipation. |  |
|    | 2 I am very worried about physical problems and it's hard to think of much else.          |  |
|    | 3 I am so worried about my physical problems that I cannot think of anything else.        |  |
|    |                                                                                           |  |
| 21 | 0 I have not noticed any recent change in my interest in sex.                             |  |
|    | 1 I am less interested in sex than I used to be.                                          |  |
|    | 2 I have almost no interest in sex.                                                       |  |
|    | 3 I have lost interest in sex completely.                                                 |  |
|    | Total Score                                                                               |  |

## B. Interpreting the Beck Depression Inventory

- Now that you have completed the questionnaire, add up the score for each of the twenty-one questions by counting the number to the right of each question you marked.
- The highest possible total for the whole test would be sixty-three. This would mean you circled number three on all twenty-one questions.
- Since the lowest possible score for each question is zero, the lowest possible score for the test would be zero. This would mean you circles zero on each question.
- You can evaluate your depression according to the Table below.

Table 45: BDI Scoring Criteria

| Total Score | Levels of Depression                      |
|-------------|-------------------------------------------|
| 1-10        | These ups and downs are considered normal |
| 11-16       | Mild mood disturbance                     |
| 17-20       | Borderline clinical depression            |
| 21-30       | Moderate depression                       |
| 31-40       | Severe depression                         |
| over 40     | Extreme depression                        |

## 14.6.2 Patient health questionnaire-9 (PHQ-9)

### A. Overview of PHQ-9

- The Patient Health Questionnaire-9 (PHQ-9) is a brief, validated self-report tool used to screen for and measure the severity of depression in clinical and primary care settings.
- It consists of 9 items, assessing symptoms experienced over the past two weeks, with each item scored from 0 to 3 (total score 0–27).
- Scores are categorized into severity levels (minimal, mild, moderate, moderately severe, and severe), with a score of  $\geq 10$  commonly indicating clinically significant depression requiring further evaluation.
- The PHQ-9 is widely used for screening, diagnosis support, and monitoring treatment response, and has strong evidence for reliability and validity (Kroenke et al., 2001).

Table 46: PHQ 9 Screening Tool

| No. | Item                                                                                                                                                                     | Not at all | Several days | More than half the days | Nearly every day |
|-----|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------|--------------|-------------------------|------------------|
| 1   | Little interest or pleasure in doing things                                                                                                                              | 0          | 1            | 2                       | 3                |
| 2   | Feeling down, depressed, or hopeless                                                                                                                                     | 0          | 1            | 2                       | 3                |
| 3   | Trouble falling or staying asleep, or sleeping too much                                                                                                                  | 0          | 1            | 2                       | 3                |
| 4   | Feeling tired or having little energy                                                                                                                                    | 0          | 1            | 2                       | 3                |
| 5   | Poor appetite or overeating                                                                                                                                              | 0          | 1            | 2                       | 3                |
| 6   | Feeling bad about yourself — or that you are a failure or have let yourself or your family down                                                                          | 0          | 1            | 2                       | 3                |
| 7   | Trouble concentrating on things, such as reading the newspaper or watching television                                                                                    | 0          | 1            | 2                       | 3                |
| 8   | Moving or speaking so slowly that other people could have noticed? Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual | 0          | 1            | 2                       | 3                |
| 9   | Thoughts that you would be better off dead or of hurting yourself in some way                                                                                            | 0          | 1            | 2                       | 3                |
|     | Total                                                                                                                                                                    |            |              |                         |                  |

#### Interpretation of results

| Score Range | Depression Severity |
|-------------|---------------------|
| 0–4         | Minimal or none     |
| 5–9         | Mild                |
| 10–14       | Moderate            |
| 15–19       | Moderately severe   |
| 20–27       | Severe              |

A score of  $\geq 10$  is commonly used as a threshold for clinically significant depression requiring further evaluation.

### 14.6.3 Generalized anxiety disorder 7 (GAD-7)

The GAD-7 is a 7-item, two-week recall screening tool used to assess and grade the severity of generalized anxiety disorder, with scores from 0–21 and  $\geq 10$  indicating clinically significant anxiety requiring further evaluation.

## GENERALIZED ANXIETY DISORDER 7 GAD-7

**PURPOSE** To screen for and measure the severity of generalized anxiety disorder

**7 ITEMS**

- 1 Feeling nervous, anxious, or on edge
- 2 Not being able to stop or control worrying
- 3 Worrying too much about different things
- 4 Trouble relaxing
- 5 Being so restless that it is hard to sit still
- 6 Becoming easily annoyed or irritable
- 7 Feeling afraid as if something awful might happen

**INTERPRETATION**

- 0–4** Minimal anxiety
- 5–9** Mild anxiety
- 10–14** Moderate anxiety

**SCORING**

|                           |          |
|---------------------------|----------|
| Not at all                | <b>0</b> |
| Not at one day            | <b>1</b> |
| Nearly every day          | <b>2</b> |
| 10–14<br>Moderate anxiety | <b>3</b> |
| 15–21<br>Severe anxiety   |          |

A score of 10 or greater suggests the presence of clinical anxiety



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