

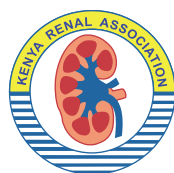
# GUIDELINES FOR MANAGEMENT OF EMERGENCIES IN DIALYSIS

2017

## EMERGENCIES IN DIALYSIS



MINISTRY OF HEALTH



KENYA RENAL ASSOCIATION

## FOREWORD: MINISTRY OF HEALTH

### **Message from the Chairman, Kenya Renal Association**

The Kenya Renal Association (KRA) is pleased to partner with the Ministry of Health in the formulation of the dialysis guidelines. This is the first edition of the guidelines covering many areas relevant in dialysis care. The writing of these guidelines was motivated by the realization of the existing human resource capacity gap in the country. The expansion in installed hemodialysis infrastructure has far out - paced the requisite trained human resource complement needed to offer these services. As a result, services are offered by personnel who have only undertaken short preceptorship courses. These short courses in no way substitute formal training. These guidelines attempt to provide a guide to management of common conditions and situations that may be encountered during the course of dialysis and in management of dialysis dependent patients.

These guidelines are deliberately simplified to make them easy to use. In coming up with these guidelines, we have borrowed from other major guidelines which we acknowledge at the references section. KRA is grateful to all who contributed their time and effort to ensure that these guidelines come to fruition. Special gratitude goes to the pioneer EAKI Nephrology Fellows who took the time to painstakingly go through these guidelines as well as the Nephrologists who mentored them.

These guidelines are by no means exhaustive and the user must not hesitate to ask for help or consult more detailed nephrology texts if they encounter situations not envisioned or well captured in these guidelines. KRA hopes to review these guidelines periodically as and when significant changes to best practice recommendations occur.

It is hoped that these guidelines will prove educative and practical to the user and help improve the quality of care offered to the dialysis patient.

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## Abbreviations and acronyms

ACLS	-	Advanced cardiac life support
aPTT	-	Activated partial thromboplastin time
AV	-	Arterio-venous
BLS	-	Basic life support
CO <sub>2</sub>	-	Carbon-dioxide
CPR	-	Cardio-pulmonary resuscitation
dDAVP	-	Deamino delta-D-arginine vasopressin
DDS	-	Dialysis disequilibrium syndrome
ESA	-	Erythropoiesis stimulating agent
ETO	-	Ethylene oxide
FFP	-	Fresh frozen plasma
HD	-	Haemodialysis
HIT	-	Heparin-induced thrombocytopenia
HUS/TTP	-	Haemolytic uremic syndrome/ Thrombotic thrombocytopenic purpura
INR	-	International normalized ratio
KDOQI	-	Kidney Disease: Outcome Quality Initiative
MAP	-	Mean arterial pressure
mls	-	Millilitres
PaO <sub>2</sub>	-	Partial pressure of oxygen in arterial blood
UF	-	Ultra-filtrate
VQ	-	Ventilation-perfusion

## **Introduction**

Although dialysis machines are equipped with a fail-safe mode, a self-test and alarms, to attain a safety profile of less than 1 event per 100 million treatments, emergencies related to human error, and patients' medical conditions, ranging from minor discomfort to cardiac arrests have been reported in dialysis units.

Common haemodialysis emergencies include: -

- Hypotension
- Intra-dialytic hypertension crises
- Sudden cardiac arrest
- Disequilibrium syndrome
- Intra-dialytic seizures
- Intra-dialytic hypoxemia
- Air embolism
- Intra-dialytic hyperkalemia
- Dialyzer reactions
  - o Type A (anaphylactic reaction)
  - o Type B (nonspecific reaction)
- Blood loss
- Febrile reactions
- Haemolysis
- Acute coronary events e.g. myocardial infarctions
- Arrhythmias
- Muscle Cramping
- Metabolic acidosis

## **Emergency response preparedness**

All staff working in dialysis units must be trained and updated in basic live support(BLS) and advanced cardiac life support(ACLS).

It is recommended that the following equipment be readily available in the unit, and well-maintained: -

- Portable oxygen cylinder with cylinder key, oxygen concentrators or piped oxygen.
- Manual and/or automated external defibrillator, with pacing function if possible.
- Glucometers.
- Emergency resuscitation trolley containing: -
  - Pocket mask with oxygen port
  - Self-inflating resuscitation bag with oxygen reservoir and tubing (e.g. Ambu bag)
  - Clear oxygen mask with reservoir bag
  - Appropriate size clear face masks
  - Nebulisation kits
  - Oropharyngeal airways
  - Suction machines (at least 2)
  - Appropriate sizes of endotracheal suction catheters
  - Appropriate sizes of laryngeal mask
  - McGill's forceps
  - Appropriate sizes of endotracheal tubes (oral, cuffed)
  - Lubricating gel
  - Laryngoscopes (normal and long blades)
  - Spare laryngoscope bulb and batteries
  - Ribbon gauze

- Scissors
- Intravenous fluids including normal saline
- Circulation equipment/*(assorted)*: -
  - Intravenous cannulae
  - Hypodermic needles
  - Syringes
  - Cannula-fixing dressings and tapes
  - Central venous catheters
  - Intravenous giving sets

It is recommended that the following medications be available for emergency use: -

- Inotropes: dopamine, dobutamine, noradrenaline, vasopressin
- Solutions: 50% dextrose, 3% saline
- Protamine sulphate.
- Lignocaine
- Amiodarone
- Hydrocortisone
- Adrenaline
- Adenosine.
- Magnesium Sulphate 50%
- Potassium Chloride
- Calcium gluconate 10%
- Sodium Bicarbonate
- Atropine
- Chlorpheniramine maleate, enteral and parenteral formulations
- Nifedipine, Clonidine
- Paracetamol, enteral and parenteral



- Nitro-glycerine.
- Ondansetron, metoclopramide, PPIs, ranitidine.
- Vitamin K, tranexamic acid, desmopressin
- Anticonvulsants: -Midazolam, Diazepam.
- Salbutamol nebulization solutions.
- Naloxone 400 micrograms

All medications should be stocked in resuscitation trolley in adequate quantities depending on patient load.

Expiry dates of medications should be verified periodically.

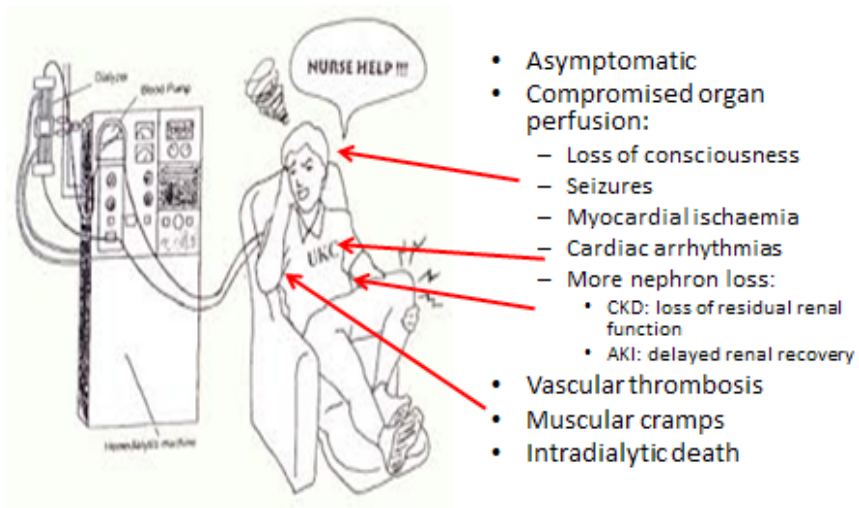
Stocks are to be verified every morning and replaced if used.

A critical care unit should be within reach.

### **Hypotension**

KDOQI defines hypotension as a decrease in systolic blood pressure by equal or more than 20mmHg or a decrease in mean arterial pressure(-MAP) by 10mmHg associated with symptoms.

# Effects of hypotension



*Figure 1: Effects of hypotension*

Treatment of hypotension occurring intra dialytically:

- Reduce blood flow rates.
- Reduce or stop ultra-filtration (UF).
- Place patient in the Trendelenburg position.
- Bolus with intravenous normal saline (200mls in adults), repeat if no response.
- If no response to saline boluses, give 30 mls of 50 % dextrose, slowly over 10min.
- If no response, discontinue dialysis and stabilize the patient.

- Evaluate for precipitants (including arrhythmias, acute coronary syndrome, anaemia, hypoglycemia).

Dialysis modification to prevent intra-dialytic hypotension in a predisposed patient include:

- UF profiling: - begin with higher UF rates and end with lower UF rates.
- Sodium profiling: - begin with higher dialysate Na concentration e.g. 145mEq/L and end with lower e.g. 135mEq/L.
- Dialysate cooling to 35.5 - 36.0°C.
- Frequent dialysis sessions for fluid overloaded patients to allow for lower UF targets per session.

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Prevention of hypotension:

- Avoid excessive interdialytic weight gain by avoiding excess salt and free water intake.
- Ensure accurate evaluation and re-evaluation of patient's dry weight (use a bio-impedance machine if available).
- Hold use of anti-hypertensive medications just before dialysis unless advised.
- Avoiding heavy meals during dialysis.
- Correction of anaemia and hypoalbuminaemia.
- Recognise and Refer patients with congestive cardiac failure or arrhythmias for appropriate care.

## **Intra-dialysis hypertensive crises**

Severe hypertension occurs in 8-30% of haemodialysis treatments. It is associated with increased risk for cardiovascular mortality. Aetiologies include volume overload, hyperactive renin-angiotensin system, erythropoietin stimulating agents, increased dialysate sodium concentration, increased sympathetic hyperactivity, dialytic removal of anti-hypertensives.

Treatment:

- It is recommended that treatment be instituted if systolic BP is >180mmHg.
- Short acting anti-hypertensives (e.g. nifedipine 20mg, clonidine 25-50µg) may be used
- ESA should not be given if BP is more than 180/110mmHg.

## **Sudden cardiac arrest**

This can happen during the dialysis procedure, immediately after dialysis or outside the dialysis unit. Risk factors include: old age, diabetics, cardiovascular disease, anaemia, use of catheters compared to arteriovenous fistulas, long inter-dialytic period.

The most common mechanism is cardiac arrhythmias. Other aetiologies are: -

- Myocardial ischaemia
- Electrolyte abnormalities e.g. hyperkalaemia, hypocalcaemia
- Air embolism
- Unsafe dialysate composition
- Overheated dialysate

- Line disconnection
- Sterilant in dialysate

Treatment:

- Disconnect the patient from the machine
- Resuscitation according to the ACLS protocol
- Do not remove the AV fistula needles.
- If there is no obvious identifiable cause, do not reinfuse blood back to the patient.

### **Dialysis disequilibrium syndrome**

Dialysis disequilibrium syndrome(DDS) is a clinical syndrome of neurological deterioration that is seen in patients who undergo dialysis. It is more likely to occur in patients during, or immediately after the first treatment, but can occur in any patient who receives haemodialysis. It usually occurs towards the end of HD but may be delayed for up to 24 hours.

Risk factors:

- Initial dialysis session
- Long inter-dialytic period
- Severe azotaemia (high urea levels)
- Dialyzer membrane: high-flux, large surface area
- Low dialysate sodium concentration
- Extremes of age
- Pre-existing neurological disorders

Presentation:

- Mild: Restlessness, headache, nausea, blurred vision, tremor, muscle twitching.
- Severe: Disorientation, obtundation, seizures, coma.

Disequilibrium syndrome is best prevented by ensuring slow urea removal through initiating low efficiency dialysis as below:

- Use small surface area dialyzers (0.9-1.2m<sup>2</sup>).
- Use slow blood flow rates (150-200mL/min).
- Use concurrent rather than counter current blood flow.
- Perform short and frequent HD e.g. 2hours daily.

Treatment:

Symptomatic and supportive treatment is the mainstay of management.

For Severe disequilibrium syndrome: -

- Stop dialysis
- Give oxygen, intubate, anticonvulsants e.g. phenytoin
- Give 50% dextrose (30-50mls), or
- Give mannitol 12.5gms, or
- Give 30% saline (3-4mls).

DDS is self-limited, but full recovery may take several days.

### **Intra dialytic seizures**

Seizures occur in less than 10% of dialysis treatments. They are usually generalized and are easily controlled. If focal or refractory, evaluation for

focal neurological disease is necessary.

Aetiologies:

- Metabolic encephalopathy: Hypoglycaemia, hypocalcaemia, hypomagnesaemia, hypernatremia (hyper osmolality), hyponatremia, severe acid-base disturbance.
- Hypoxic encephalopathy (sustained hypotension): Cardiac arrhythmia, sepsis, haemorrhage, hypersensitivity reaction.
- Hypertensive encephalopathy.
- Stroke (focal neurological deficit): Intracranial haemorrhage, ischaemic stroke.
- Uraemic encephalopathy.
- Dialysis disequilibrium syndrome.
- Drugs and toxins: Epileptogenic drugs e.g. theophylline, pethidine, penicillin, ESAs, aluminium, carbapenems; withdrawal e.g. dialytic removal of anticonvulsants; alcohol withdrawal.

Treatment:

- IV Diazepam 10mg.
- IV Phenytoin infusion if persisting or repetitive seizures.
- Treatment of specific cause.
- If recurrent seizures occur, long term treatment with anticonvulsants is necessary.

### **Dialysis-associated hypoxemia**

Most patients on HD develop a decline in their partial arterial oxygen pressure(PaO<sub>2</sub>) by 5-20mmHg, reaching a nadir at 30-60minutes and re-

solving within 60-120minutes after HD. This is often of no clinical significance unless there is pre-existing chronic cardiopulmonary disease.

Aetiology:

Hypoventilation:

- Central: Triggered by loss of CO<sub>2</sub> in dialyzer and/or rapid alkalization of blood.
- Peripheral: Ventilation-perfusion (V-Q) mismatch due to pulmonary leuko-agglutination secondary to complement activation and impaired cardiac output. It can also result from respiratory muscle fatigue.

Treatment in high-risk patients:

- Intra-dialytic oxygen supplementation.
- Use biocompatible membranes.
- Optimize the patient's haemoglobin.
- Use sequential isolated UF followed by HD.

### **Air embolism**

Introduction of more than 50 mls of air into the blood circulation can be a serious event. The sources of this air include:

- Pre-pump tubing (where there is the highest negative pressure).
- Intravenous infusion lines.
- Air bubbles in dialysate.
- HD catheter.

Presentation:

The presentation is dependent on the volume of air introduced, speed of air



introduction site of air introduction and position of patient.

- Sitting position: Peripheral vein air bypasses the heart and ascends into the cerebral veins, resulting in acute-onset seizure and coma.
- Supine position: Peripheral vein air gets trapped in the right ventricle, leading to a decrease in the cardiac output and causing obstructive shock. From the right ventricle, air can enter the pulmonary circulation resulting in dyspnoea, dry cough, chest tightness and even respiratory arrest. If the air bypasses the lungs, it enters the cerebral or coronary arteries, leading to seizure, coma, chest pain or cardiac arrest.
- Left Trendelenburg: Peripheral vein air ascends into the lower extremity veins resulting in lower limb ischemia.

Treatment:

- Clump the venous blood line.
- Stop the blood pump.
- Place the patient in the left Trendelenburg position.
- Institute CPR if necessary.
- Use high-flow oxygen or hyperbaric oxygen.
- Endotracheal intubation and mechanical ventilation when necessary.
- Consult cardiologist or cardiothoracic surgeon (air can be aspirated from the right ventricle using a lumbar puncture needle or from the right atrium using in-situ a HD catheter).

### **Intra-dialytic hyperkalaemia**

Dialysis induced hyperkalemia is rare. It can result from high dialysate potassium concentration, haemolysis, accidental potassium infusions.

Treatment:

10%, 1-gram (10 mls) calcium gluconate, 10 I.U soluble insulin and 50 mls of 50% dextrose infusion. Nebulization with beta 2 agonists, sodium bicarbonate and cation exchange resins e.g. resonium may be given.

### **Dialyzer Reactions**

Reactions attributed to the hemodialyzer are generally divided into two types:

- *Type A* - Anaphylactic reactions. Increased risk in patients with a history of atopy, eosinophilia and allergic reactions during dialysis.
- *Type B* - Mild reaction. Pathogenesis of type B reaction is not clear. It may be related to complement activation. Current data do not support the role of membrane biocompatibility in development of type B reactions.

Diagnosis:

*Type A reaction*: - Severe and rapid in onset. Established by three major criteria or two major and one minor criterion:

Major criteria:

- Onset within 20 minutes of starting dialysis.
- Dyspnoea.
- Burning/heat sensation at the access site or throughout the body.
- Angioedema.

Minor criteria:

- Reproducible during subsequent dialysis when using the same type or brand of dialyzer.
- Urticaria.
- Rhinorrhoea or lacrimation.

- Abdominal cramping.
- Itching.

### *Type B reaction*

- Primary symptoms are chest and back pain.
- Occurs 20-40 minutes into the dialysis treatment.
- Disappears or lessens dramatically during the subsequent hours of dialysis.

### Treatment:

- Symptomatic and supportive.
- Discontinue HD and discard the blood. Give oxygen, give anti-histamines, epinephrine and corticosteroids.
- HD can be initiated after stabilization with a more biocompatible membrane and a haemodialyzer not sterilized with ETO (ethylene oxide).

### ***Haemorrhage***

### Risk factors include:

- Platelet dysfunction.
- Ineffective platelet-vessel wall interaction and heparin induced thrombocytopenia (HIT).
- Use of anti-coagulation during HD.
- Co-morbid conditions e.g. uncontrolled hypertension, liver disease, sepsis, certain medication (especially anti-platelet drugs), HUS/TTP, malaria associated renal failure.
- Poorly secured access site.
- Venous needle falling out or catheter connection disrupted (venous pressure may fall too little to cause an alarm).

## Diagnosis and Treatment:

- Screen using bedside-bleeding time, activated partial thromboplastin time(aPTT) and INR.
- Prolonged bleeding time - may require platelets, cryoprecipitate, DDAVP, FFP.
- Prolonged PTT (heparin induced) - require protamine (1mg/ 100 units of heparin), FFP.
- Tranexamic acid when indicated.
- Vitamin K in prolonged INR.
- Transfuse blood if necessary.

## Prevention:

- Should never keep access site covered during dialysis.
- Heparin dose should be reviewed.
- Strategy based on risk assessment:
  - o Low risk - low dose, low molecular weight heparin.
  - o Very high/high risk - regional anticoagulation with heparin and protamine, heparin- free dialysis, regional citrate anticoagulation, prostaglandin (PGI<sub>2</sub>), peritoneal dialysis.

## **Febrile reactions**

Febrile reactions are defined as a rise in temperature during HD of at least 0.5° C or a rectal or axillary temperature during dialysis of at least 38.0 or 37.5° C respectively. The majority (70%) of febrile reactions are associated with preexisting infections (vascular access, urinary and respiratory). HD related febrile reactions can be associated with localized infection of the vascular access site (especially catheters and grafts) or

products from the dialysate and/or the apparatus used for HD treatment.

#### Diagnosis and Treatment:

- Obtain blood cultures.
- Begin broad spectrum antibiotics immediately.
- Treatment is largely supportive and empirical.
- Cluster of similar cases should prompt a review of water used for reprocessing, dialysate, processing procedure, bicarbonate system.

#### Prevention:

- Reduce the use of catheters for HD.
- Reduce the susceptibility to infections by:
  - Provide adequate HD.
  - Prevent and/or treat malnutrition.
  - Optimize haemoglobin concentration.
  - Avoid iron overload.
- Use biocompatible dialysis membrane.

### **Muscle cramping**

Approximately 20% of dialysis sessions are accompanied by muscle cramps.

#### Etiology:

- Cramps are more pronounced in patients who require high ultrafiltration rates and are possibly dialyzed below their dry weight. They are presumably related to reduction in muscle perfusion that occurs in response to hypovolemia. Compensatory vasoconstrictive responses

may shunt blood centrally during treatment, and could play a role in promoting muscle cramps.

- Changes in intra or extracellular balance of potassium and concentration of ionized calcium can disturb neuromuscular transmission and produce cramps.
- Peripheral vascular disease, although common in dialysis patients, may not be associated with increased prevalence of intradialytic cramps which confirms that processes related to the dialytic treatment are responsible for the cramps.

### Differential Diagnosis:

While the majority of cramps are associated with dialysis treatment, the differential diagnosis is extensive and includes the following conditions:

- 
- Idiopathic cramps.
- Contractures (occurring in conditions such as metabolic myopathies, and thyroid disease)
- Tetany (due to hypocalcaemia or alkalosis).
- Dystonias (anti-psychotic medications).
- Other leg problems such as restless leg syndromes and periodic leg movements, must be distinguished from cramps.

### Treatment and Prevention:

- Many of the treatment strategies are similar to those used to treat intradialytic hypotension (see intradialytic hypotension above).
- Physical manoeuvres such as massage of the calf muscles and dorsiflexion of the foot are not very helpful.
- Immediate treatment is to increase intravascular volume by interrupt-

ing or slowing ultra-filtration and administering saline, mannitol or glucose. In addition to effecting an intravascular shift of water, hypertonic solutions may directly improve blood flow to the muscles.

- Use of quinine 300mg as a start dose may help.
- Use of dialysate sodium, potassium or calcium modelling. The concept of individualization of dialysate composition seems to be a good preventive method.
- Careful reassessment of the dry weight by bio-impedance machine where possible, counselling the patient to reduce inter-dialytic weight gain and using bicarbonate dialysis.

### **Metabolic acidosis**

- Can occur accidentally as a consequence of dialysate fluid containing improper ratios of acid and base concentrates in the form of acetate or bicarbonate.
- Can also develop as a result of the accidental use of an acidic concentrate instead of acetate or bicarbonate and due to computer software malfunction of the machine.
- Severe metabolic acidosis has been reported during first 2 hours of HD using sorbent regenerative hemodialysis in mechanically ventilated patients.
- Treatment consists of intravenous administration of bicarbonate and dialysis with bicarbonate dialysate of a correct concentration (38-40 mEq/L.)
- The mainstay of prevention is to fit all HD machines with a pH meter and alarms that will prevent the extreme acid load, which may be caused by an inappropriately prepared bicarbonate dialysate.

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