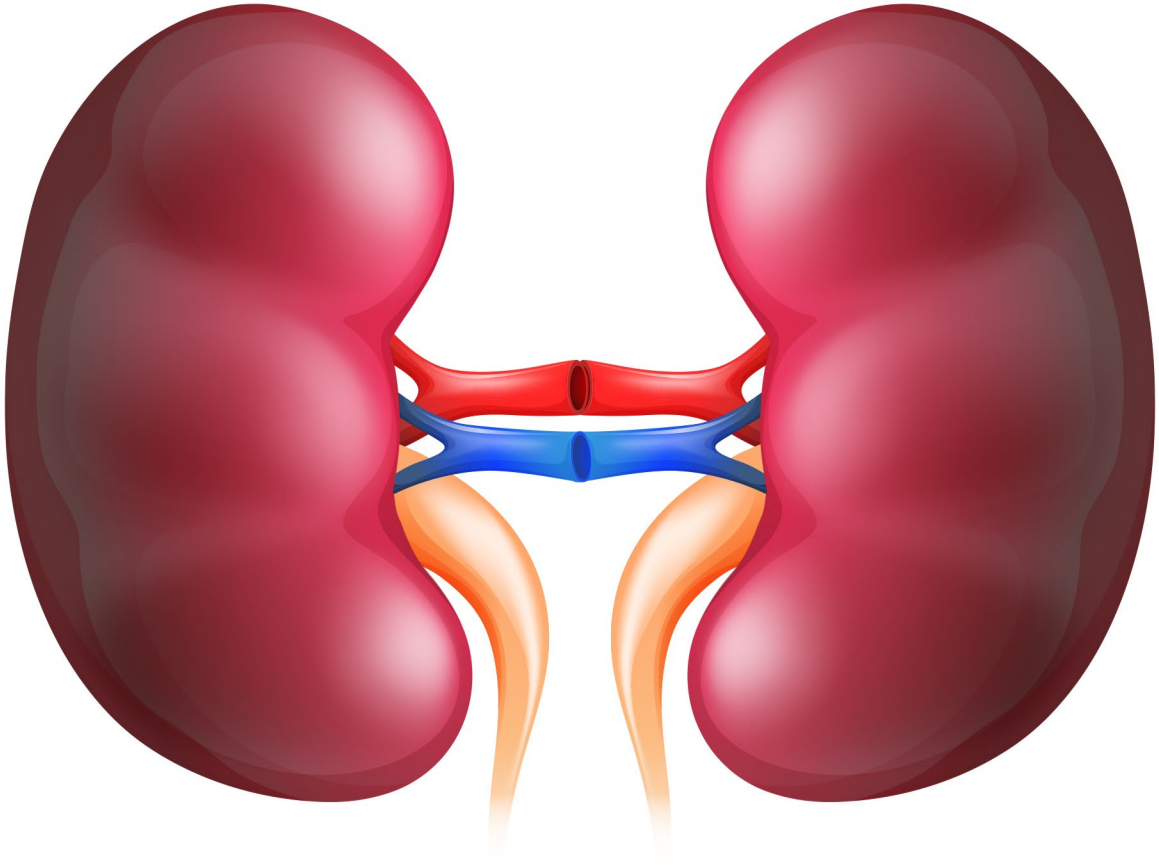
Malawi Government
Ministry of Health

National Renal Protocols



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ACRONYMS

ACEi Angiotensin Converting Enzyme inhibitors, AKI Acute Kidney Injury,
AKIN Acute Kidney Injury Network AOC Acute on Chronic Kidney Disease ARB Angiotensin Receptor Blockers,
ATI Acute Tubular Injury

ATN Acute Tubular Necrosis,

BP Blood Pressure,

BUN Blood Urea Nitrogen

Ca Cancer

CCF Congestive Cardiac failure

CKD Chronic Kidney Disease

COX II Cyclooxygenase -2

CT Computed Tomography

Cx Cervix

CXR Chest X-ray

DAMN Diuretics, ACEi/ARBs, Metformin, NSAIDs DIC Disseminated Intravascular coagulation
DM Diabetes Mellitus

ECG Electrocardiogram

EPO Erythropoietin

ESRD End Stage Kidney Disease,

ESR Estimated sedimentation rate

eGFR Estimated Glomerular Filtration Rate GFR Glomerular Filtration Rate,
GIT Gastrointestinal tract

Hb Haemoglobin

HBA1c Glycated Haemoglobin HCO3 Bicarbonate
HCG Human Chorionic Gonadotropin

HTN Hypertension

HUS Haemolytic Uremic Syndrome

ICU Intensive Care Unit

iPTH intact Parathyroid Hormone

IV Intravenous

K+ Potassium

KDIGO Kidney Diseases Improving Global Outcomes L Lumbar
LFTs Liver Function Test

MAP Mean Arterial Pressure Mmol/L Millimoles per liter
MRI Magnetic Resonance Imaging

Na+ Sodium

NGT Nasogastric tube

NSAIDs Non-Steroidal Anti-inflammatory Drugs, OD Once a day
PEW Protein Energy Wasting

RAAS Renin Angiotensin Aldosterone System RBCsRed Blood Cells
RIFLE Risk, Injury, Failure, Loss, End Stage Renal Disease RRT Renal Replacement Therapy
SBP Systolic Blood pressure

SC Subcutaneous

sCR Serum Creatinine

STOP Sepsis, Toxicity, Obstruction, Paranchymal T Thoracic
TB Tuberculosis

TIN Toxic Ischaemic Nephropathy

TTP Thrombotic Thrombocytopenic Purpura U & Es Urea and electrolytes
UTI Urinary Tract Infections

µmol/L micromole per Liter

WBCs White Blood Cells

FOREWORD

The burden of kidney disease is increasing in Malawi and causes are multifactorial. Most of the patients with kidney disease in our setting are aged below 45 years and usually present late during advanced disease stage requiring kidney replacement therapy which is very expensive and not always available. This document aims to provide guidance on the prevention of kidney disease. It also provides evaluation, management and treatment of all patients with both acute kidney injury (AKI) and chronic kidney disease (CKD).

The guidelines present an enhanced classification framework for acute and chronic kidney disease; elaborates on the identification and prognosis of kidney disease; discusses the management of progression and complications of kidney disease; expands on the continuum of kidney disease care: timing of specialist referral, ongoing management of people with progressive kidney disease, timing of the initiation of dialysis and the implementation of a treatment program which includes comprehensive conservative management.

Ministry of Health is committed to improving the quality of care delivered to all patients with kidney disease. Therefore, developing renal protocols was important not only to equip staff with knowledge, but also to improve patient outcomes, avoid complications and slow disease progression, and, subsequently, reduce financial burden which the hospital incurs when managing renal patients.

Honourable Khumbize Kandodo Chiponda,
MP. MINISTER OF HEALTH

ACKNOWLEDGEMENT

The Ministry of Health would like to acknowledge the efforts of the Individuals and organizations that contributed to the success in finalizing these national renal protocols. It is the Ministry's desire to improved quality of clinical care for people with Acute Kidney Injury and Chronic Kidney Disease in Malawi. The technical and financial support from individuals and institutions aprovided during the development of these protocols are well appreciated.

The protocols have been developed through the scientifically rigorous exercise and based on a critical appraisal of the available evidence. The participants involved in the developing of the protocols composed of multidisciplinary teams. Thus, the development process was open, more inclusive and these guidelines are responsive to guide practice for all levels of healthcare delivery in the country. It is believed that if these guidelines are put to best use. They will be key to prevention of kidney disease, guide early detection and linkage to care for all people with kidney disease with the aim of improved quality of life and survival for a better Malawi.

Special gratitude goes to the department of Curative and Medical Rehabilitation Services through the Non-communicable diseasess and mental health (NCDs & MH) Division for providing leadership in the development of this document. The efforts of coordinating meetings, putting together vital pieces of information, comments, criticisms and suggestions and final compilation of the document is well appreciated.

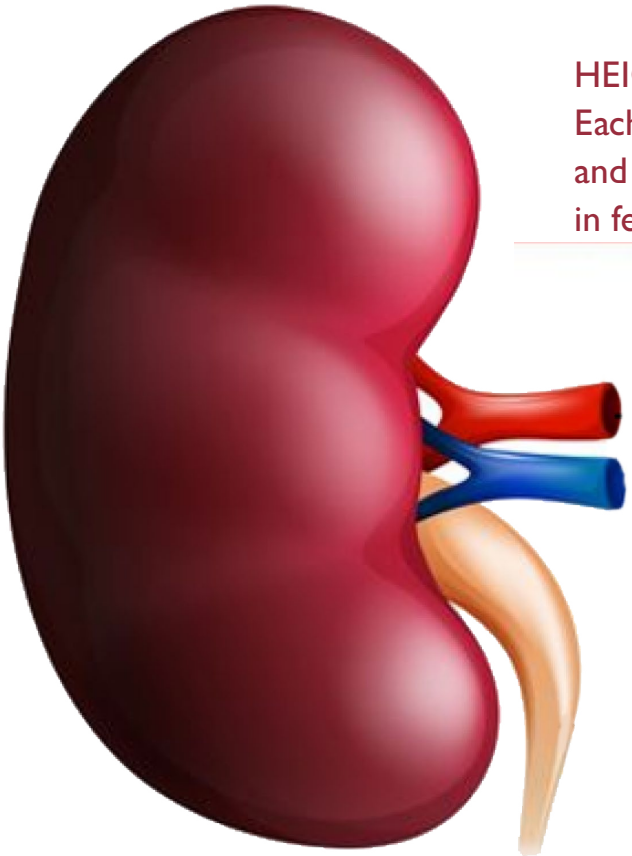
We would like therefore to acknowledge various stakeholders involved in the development of these protocols. We further recommend for collaborative efforts towards utilization of these guideline for improved service delivery to people with kidney disease.

Dr Charles Mwansambo SECRETARY FOR HEALTH

CHAPTER 1: ANATOMY AND PHYSIOLOGY OF THE KIDNEYS

Anatomy of the Kidneys

- Kidneys are paired bean shaped organs, and are also known as "renes" in Latin, Nephros in Greek.
- They lie behind peritoneum in the abdomen either side of the vertebra column (posterior abdominal wall, above the waist)
- Typically extending border of T12 to L3 vertebra
- They are partially protected by 11th and 12th pairs of ribs
- Right kidney is situated slightly lower than the left owing to the presence of the liver which occupies considerable space on the right-side superior to the kidney
- It is size of a fist weighing about 150gIn adults, the kidneys measure from 9cm-12cm long
- Each is connected to your bladder by ureters



HEIGHT & WEIGHT

Each kidney is 11 cm (4-5") long, 6 cm (2-3") broad and 3cm (1") thick, weight 150 g in males and 135 g in females

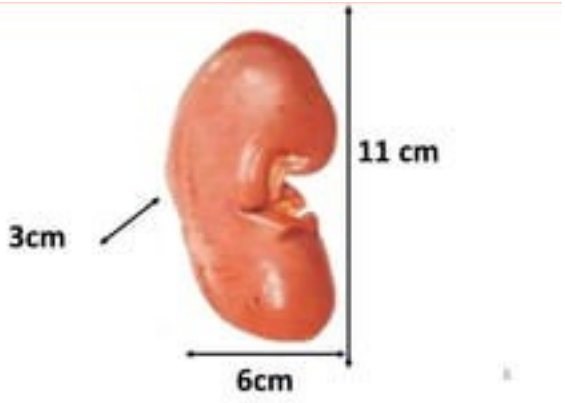


Figure 1: External appearance of Kidneys

Functions of Kidneys

Functions of kidneys are mainly; Excretory, Regulatory and Endocrine

1. Excretory

- Excretion of waste products of metabolism by filtering blood passing through them.
- Excretion of end product of protein metabolism such as urea (BUN), uric acids, phosphates, organic acid, creatinine and sulphates.
- Excretion of hormones such as Human Chorionic Gonadotropin (HCG)
- Excretion of excess water which forms urine

2. Regulatory

- Regulation of acid - base balance
- Regulation of Fluid and electrolyte balance

3. Endocrine

- Secretes renin which controls blood pressure by the mechanism of renin angiotensin aldosterone system (RAAS)
- Secretes Erythropoietin (EPO) hormone for production of red blood cells
- Activation of Vitamin D (Calcitriol) for bone health

Structures of the Kidneys

External Structures

Renal capsule – Kidneys are covered by fibrous tissue called renal capsule which protects the kidney from trauma.
Renal Hilum

Gives the kidney its shape and it is where the renal blood and lymph vessels, ureters and nerves enter.

Internal Structures

A dissected kidney has:

- **Cortex** - an outer reddish-brown layer of tissue with little obvious structures, blood filtration occurs between capsule and medulla. It contains renal corpuscles and tubules.
- **Medulla** - the middle layer consisting of pale shaped striations called renal pyramids that transport urine to the renal pelvis via the pyramids
- **Renal pelvis** - the inner layer, funnel shaped structure that collects urine formed by the kidney.
- **Calyces** – propels urine through the pelvis, ureters to the bladder. This is where kidney stones usually form.

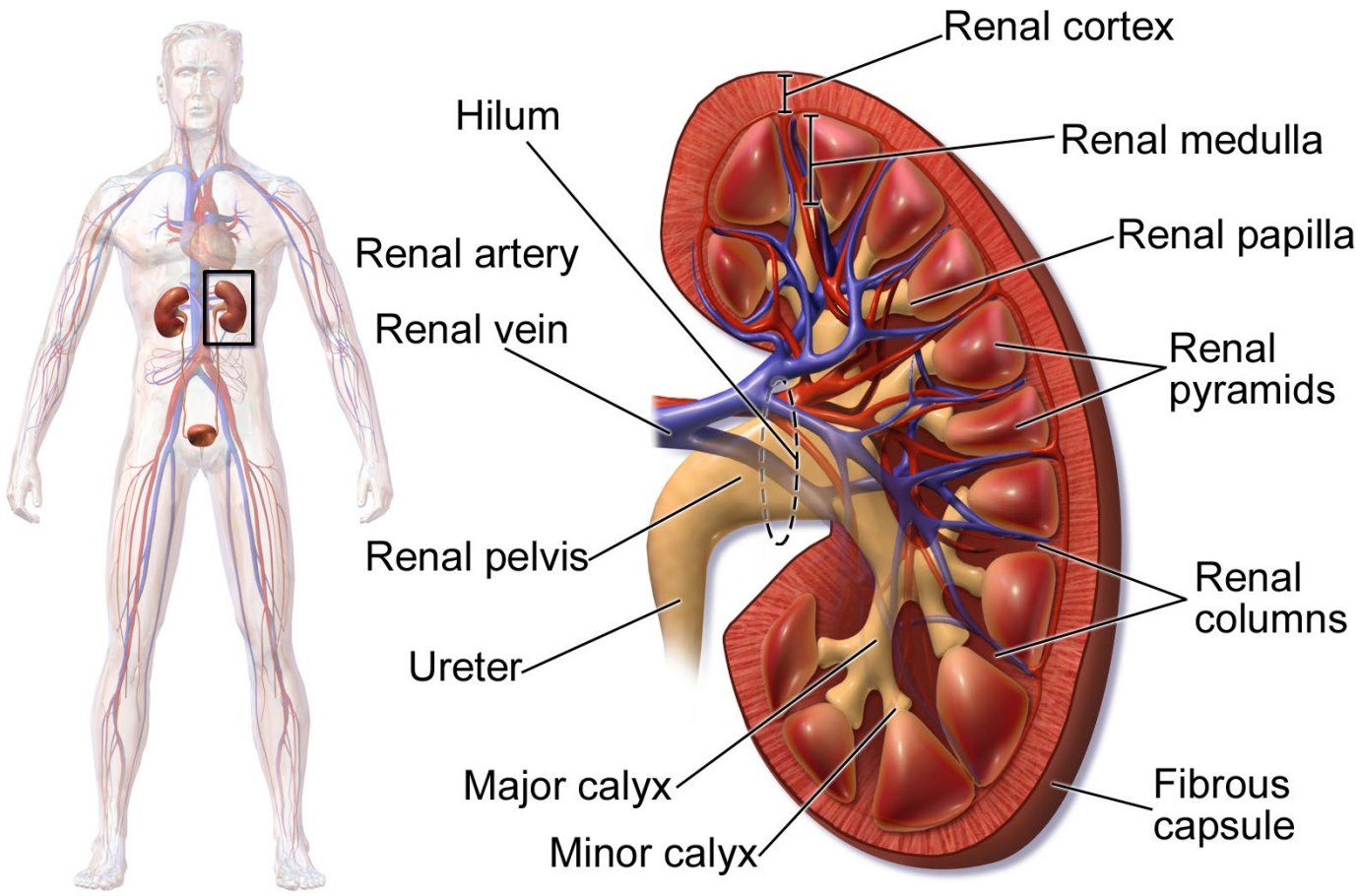


Figure 2: Intenal Structure of kidneys

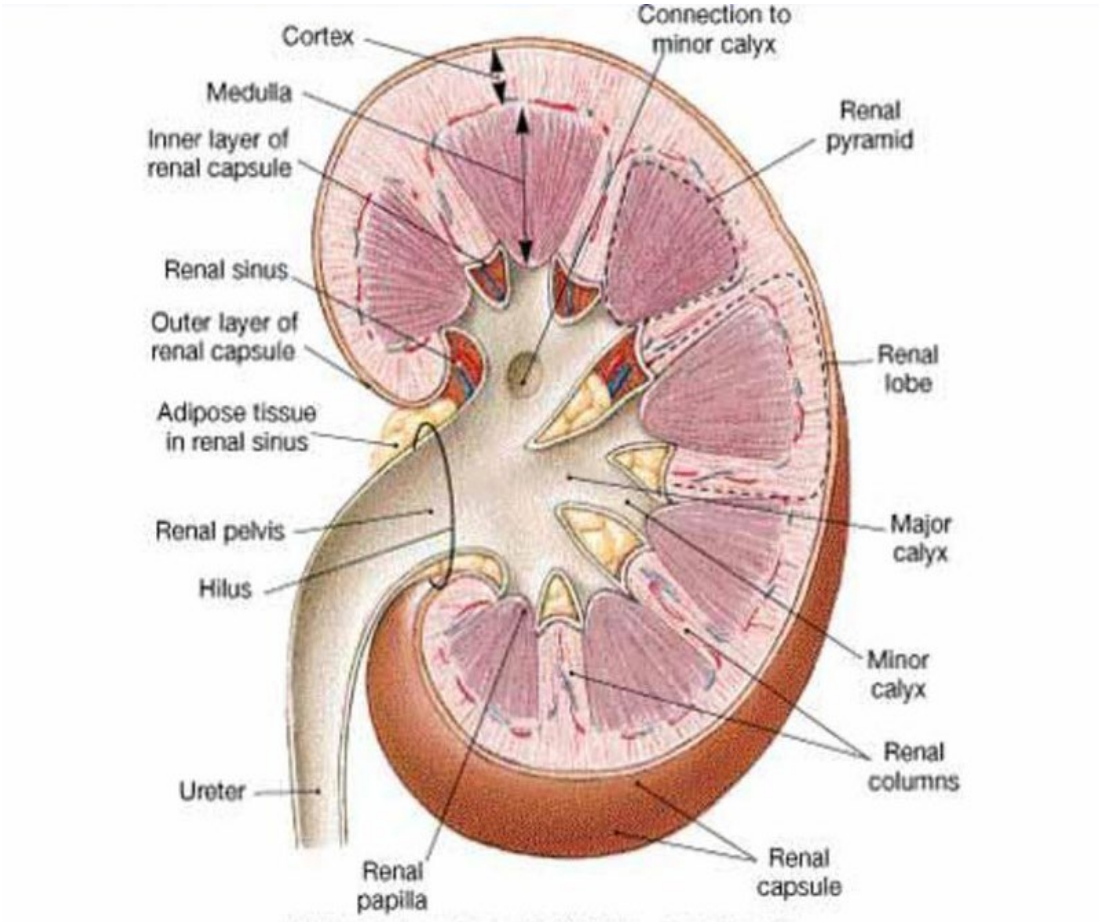


Figure 3: Frontal Section Of the left, anterior View

Microscopic Anatomy of the Kidney

The Nephron:

- It is the basic functioning unit of a kidney
- Each kidney contains approximately one million nephrons
- There are two types of nephrons – cortical and juxtamedullary
- Nephrons form urine by the process of filtration, secretion and reabsorption

Renal Corpuscle

- Renal corpuscle (the filtering unit) consists of glomerulus which contains bowmans capsule and Glomerular Basement Membrane where filtration occurs

Glomerular

- Blood enters renal corpuscles via glomerulus (ball of capillaries)
- The filtrate consists of electrolytes, glucose, some drugs, hormone insulin, sodium, potassium, magnesium, calcium, phosphorus, water
- Glomerular basement membrane prevents passage of large protein molecule while endothelium of glomerulus prevents RBC, WBCs passage
- The filtrate eventually forms urine.

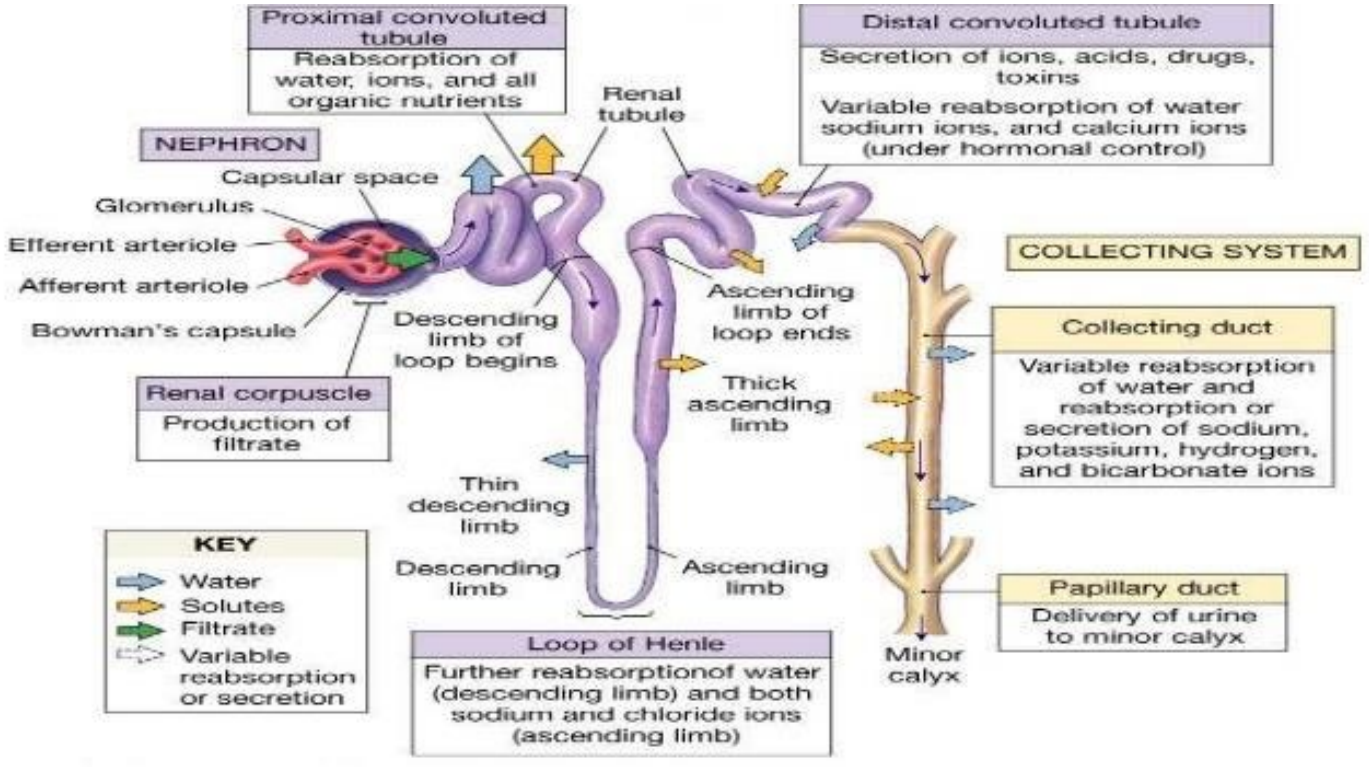


Figure 4: The Nephron

Renal Tubules

Proximal loop

- Longest part of renal tubule and first to receive fluid filtered by bowman's capsule
- Reabsorbs most of the useful substances of the filtrate i.e. sodium, chloride, bicarbonate, water, glucose. The primary site for secretion of drugs, waste and hydrogen ions

Loop of Henle

- U-shaped tube that consists of a descending limb and an ascending limb.
- Reabsorbs water and ions from the urine and controls the concentration of urine.
- The descending limb of the loop of Henle is fully permeable to water and completely impermeable to solutes (salt particles).
- This where loop diuretics works i.e Furosemide

Distal loop

- Regulates the electrolytes, pH and further dilution of urine.

Collecting Duct

- Receives fluid from the distal convoluted tube which excretes urine into the renal pelvis even water continues to be reabsorbed and other electrolytes

Keys Steps in Urine Formation

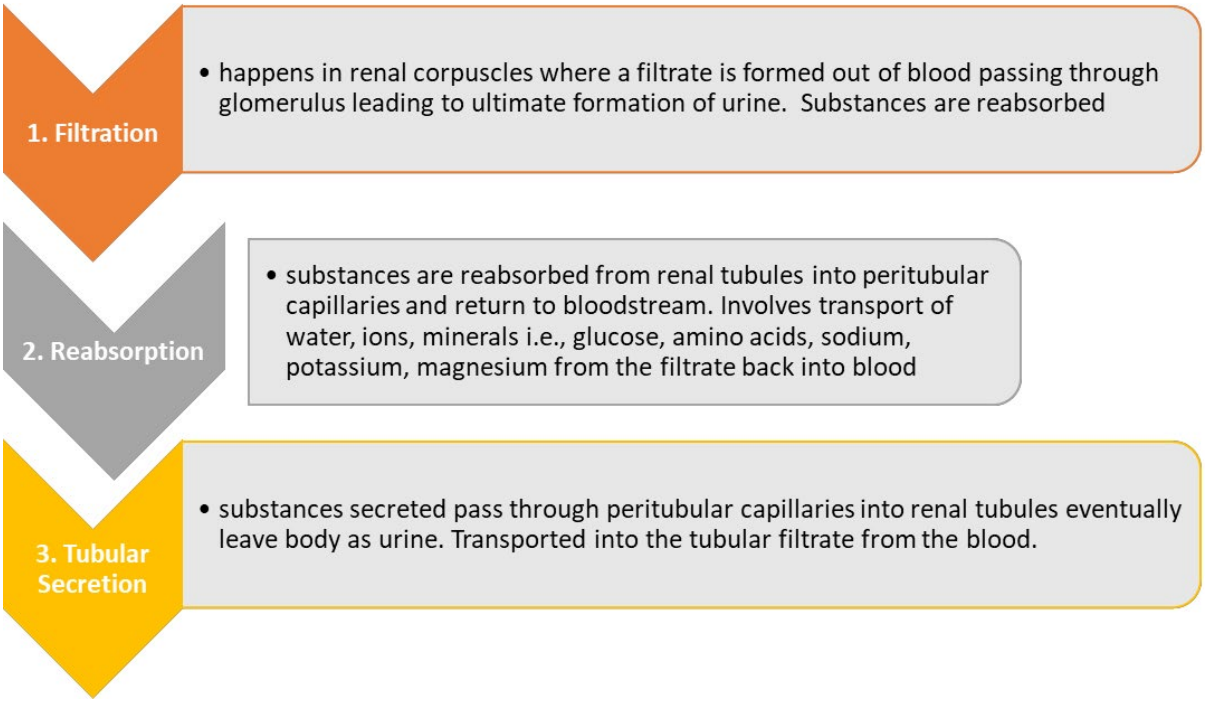


Table 1 : Amount of urine Formed in 24hours

Age	Amount of Urine/ day
New born and infants	2mls/kg/hr
Toddler (1-3yrs)	1.5mls/kg/hr
3- 12 yrs	1ml/kg/hr
Adult	0.5-1ml/kg/hour

CHAPTER 2: ACUTE KIDNEY INJURY (AKI)

Acute kidney injury network (AKIN)

AKIN defines AKI as an abrupt reduction in kidney function over a few hours or days (within 48 hours) with an absolute increase in serum creatinine of either ≥0.3mg/dl (≥26μmol/L) or a percentage increase of ≥50% or a reduction in urine output of ≤0.5ml/kg/hour for >6hours.

Incidence

-
- AKI replaced the term of Acute Renal Failure to recognize varying degrees of kidney injury severity, and also to encourage early identification and management
- Acute loss in AKI occurs within hours to days
- Commonly in those critically ill and hospitalized, with frequencies of 3-10% among hospitalized and can rise up to 10-30% in those admitted in ICU
- Increases mortality rate up to 35- 86%.
- May last a few days or up to 3 months.
- AKI can be reversed if underlying cause is quickly identified and addressed
- Can be expensive to manage and prolongs hospitalization

Classification for Acute Kidney Injury According to Kidney Diseases Improving Global Outcomes (KDIGO) Staging

- This is a grading system for AKI severity stratification in three stages
- Changes in serum creatinine and urine output are used to define and classify AKI
- These stages recognize that AKI subsequently leads to CKD and ESRD requiring long term dialysis

Table 2: Classification of AKI

Creatinine is more reliable than urea because urea is influenced by other factors i.e. critical illness, volume status, protein intake and medications, while Serum creatinine is influenced by creatinine generation, volume of distribution and excretion. Thus, a sudden fall in GFR is accompanied by a slow rise in serum creatinine.

Risk Factors for Acute Kidney Injury

Background factors

- CKD (GFR ≤60mL/min)
- Age ≥ 65years
- Co-morbidity (CCF, Diabetes Mellitus, HTN)

Acute Context

- Sepsis
- Peri-operative period

Illness severity

- Hypovolaemia
- Systolic Bp <100mmHg
- Deteriorating early warning score

Medication use

- NSAIDS, COX II, ACEi, ARB,
- Tenofivir
- Aminoglycosides
- Iodinated contrast
- Amphotericin B

Causes of Acute Kidney Injury

Majority of causes of AKI are not renal specific.

Too little blood flow/ volume depletion/Hypoperfusion (prerenal causes)

- Low blood pressure (Hypotension)
- Hypovolemia
- Bleeding too much – acute bleeding, loss from internal/external bleeding
- Severe diarrhoea/ severe dehydration
- Heart diseases, cardiac tamponade
- Infection, Liver failure, Serious burns
- Severe allergic reaction
- Major surgery
- Urinary loss from diuretics or diabetic insipidus
- Unreplenished insensible losses i.e. excessive sweating
- Diseases – hepatorenal syndrome, sepsis/septic shock, pancreatitis
- Drug overdose

Table 3: Factors increasing susciptibility to renal hypoperfusion Factors increasing susceptibility to renal hypoperfusion Failure to decrease arteriolar resistance
Structural changes in renal arterioles (old age, atherosclerosis, hypertension, CKD)

Reduction in vasodilatory prostaglandins (nonsteroidal anti-inflammatory drugs, cyclooxygenase-2 inhibitors
Afferent glomerular arteriolar vasoconstriction (sepsis, hypercalcaemia, hepatorenal syndrome, ciclosporin /tacrolimus, radiocontrast agents)
Failure to increase efferent arteriolar resistance

Angiotensin converting enzyme inhibitors

Angiotensin receptor blockers

Renal artery stenosis

B. Direct damage (intra renal causes)

Vasculitis

- Glomerulonephritis
- Acute Tubular Injury
- Interstitial Nephritis

Vascular causes –

- Blood clots in or around the kidney
- Diseases of small vessels renal arterial obstruction e.g. vasculitis, emboli, dissection, thrombosis, TTP, HUS, Sclerodema renal crisis
- Venal obstruction e.g. thrombosis
- Microambiopathy – i.e. DIC, preeclampsia
- Malignant hypertension
- Transplant rejection

Glomerular causes –

- Glomerular Basement Membrane disease
- Infections
- Glomerulonephritis
- Immunecomplex conditions i.e. Lupus
- Nephrotic syndrome with super-imposed hemodynamic insult

Tubular causes

- Prolonged hypovolaemia or renal ischaemia
- Toxic Drugs – Amphotericin B, lithium, gentamycin, NSAIDs,
- Contrast media used in radiology
- Megadose of vitamin C (more than 1000mg/day), acyclovir
- Haemipigment in rhabdomyolysis, intravascular haemolysis
- Tropical infections such as malaria,
- Tumor lysis syndrome, seizures,

Interstitial causes

- Systemic diseases – lymphoma, leukemia, lupus, Sarcoid
- Infections- pyelonephritis, viral nephrotitis
- Drugs e.g. penicillins, NSAIDs, protopumps, TB drugs eg rifampicin

Blockage (post renal causes)

- Bladder outlet obstruction
- Bilateral ureteric obstruction
- Obstruction of a single functioning kidney
- obstruction of flow of urine due to:
    - ◊ Some cancer i.e. Cancer prostate, Cancer Cervix, bladder carcinoma
    - ◊ Blood clots
    - ◊ Renal-vein thrombosis
    - ◊ Renal stones
    - ◊ Enlarged prostate (in men)
    - ◊ Tumors e.g. retroperitoneal tumours, bladder tumours
    - ◊ Ligation from pelvic surgical errors
    - ◊ Neurogenic bladder
    - ◊ Strictures
    - ◊ Phimosis
    - ◊ Obstructed foleys catheter
    - ◊ Tense ascites

Summary of AKI pathophysiology

AKI can result from decreased renal perfusion in the setting of undamaged parenchymal tissue (pre-renal), resulting also from structural damage to the kidneys, most commonly the tubule from an ischemic or toxic insult (intrinsic), and from obstruction of urine flow downstream from the kidney (post-renal).

Phases of Acute Kidney Injury

Initiating Phase Time of insult until signs and symptoms become apparent Phase lasts for hours to days depending on cause (toxic causes last longer)
Oliguric-anuric Phase Usually appears 1-2 weeks of initiating event, can last 10-14 days Urine output 400 ml/24hrs (oliguria) or less than 100mls/24hrs (Anuria). GFR is greatly reduced which leads to BUN and creatinine; electrolyte abnormalities
Diuretic Phase Usually within 10-12 days of onset oliguric phase Urine output 2 to 4L in 24hrs can be produced. High flow of urine can contribute to volume depletion. Hence, administration of IV crystalloids is vital to maintain hydration Tubular function returns slowly

Recovery

Renal function slowly returns to normal and near normal

Lab values stabilizes, usually within a month, recovery takes up to 12 months.

Urine Output- Normal; oedema diminishes

Period when BUN and Creatinine stabilizes. If significant renal damage has occurred, BUN and creatinine may never return to normal levels

Clinical presentation of AKI

Presentation varies and dependent on underlying cause

- Symptoms may include;
    - Reduced urine output
    - Flank pain
    - Confusion
    - Shortness of breath
    - Vomiting
- Signs may include;
    - Weight gain accompanied by oedema
    - Coloured or foamy urine
    - Orthostatic hypotension especially in volume depleted patients
    - Kussmaul breathing

Clinical Assessment of AKI

Obtained thorough medical and medication histories, physical exam, laboratory and imaging findings

- Search for clinical evidence of hypoperfusion (volume depletion and hypotension)
    - ◊ Dry mucus membrane
    - ◊ reduces skun turgor of more than 2 seconds
    - ◊ reduced jugular Vein pressure
    - ◊ tachycardia
    - ◊ low blood pressure (less 90/60mmHg)
    - ◊ lethargic
    - ◊ reduced level of consciosness
    - ◊ central venous pressure of less than 8 mmHg
    - ◊ Search for urinary tract obstruction. distended urinary bladder on
        - inspection
        - palpation
        - ultra sound scan
        - catheterisation
- Hypotension may suggest significant underlying sepsis and the following test should be carried out:
    - ◊ Full blood count
    - ◊ chest xray
    - ◊ urine culture
    - ◊ blood culture
- Rash, uveitis and/or arthropathy may suggest underlying Vasculitis
-
- Consider intrinsic renal in the absence of dehydration, hypotension and obstruction. The following tests should be done

- ◊ urine dipstick
- ◊ urine microscopy
- ◊ kidney biopsy

- Look for signs of fluid overload:
  - ◊ raised jugular venous pressure
  - ◊ high blood pressure
  - ◊ low saturation(hypoxia)
  - ◊ oedema

Altered mental status would suggest uraemic Encephalopathy

CHAPTER 3: MANAGEMENT OF ACUTE KIDNEY INJURY

Tests required

- Urine dipstick looking for blood, protein, glucose, specific gravity, leukocytes, nitrates
- Urine Microscopy for RBC, WBC, crystals, casts
- Sample for Blood and urine for Culture and sensitivity
- Arterial blood gases (ABG)
- Ultrasound scan (Kidney, Ureter, and Bladder) to exclude obstruction and renal diseases.
- Urea, Creatinine and Electrolytes beware of high potassium (K+)
- FBC to show infection, acute blood loss, chronic anaemia
- Peripheral smear (schistocytes)
- Serologic test
  - ◊ HIV
  - ◊ Hepatitis B and C
- LFTs, Clotting factor, ESR
- CXR if pulmonary oedema
- Aortorenal angiography to show renal vascular disease
- Renal biopsy

Management of Acute Kidney Injury

With prompt management of pre-renal, intra-renal and post-renal causes including restoration of intravascular volume and blood pressure, normal renal haemodynamics can be restored resulting in complete recovery of renal function.

Treat the treatable cause

- If patient in shock, manage according to protocols
- Urgent urinary bladder scan to exclude bladder retention.
- If urinary bladder is palpable, insert a catheter
- Stop nephrotoxic drugs (Gentamycin, NSAIDS, ACE inhibitors and others)
- Treat precipitating/ exerbating factors: Sepsis, UTI, CCF and high BP
- If electrolyte imbalances (hyperkalaemia, use management protocols)

Treatment in children

- monitoring of blood pressure, urine output, fluid balance (input and output), daily weight
- Avoid nephrotoxins (NSAIDs, gentamycin, tenofovir)
- Treat the underlying cause

Pre-renal AKI in children

- If the child is in hypovolaemic shock and /or severely dehydrated, treat according to protocol
- Refer to renal team

Renal AKI in children

◊ Treat hypertension with;

* Calcium channel blockers: Nifedipine initial dose 0.25-0.5mg/kg/day divided in 2 to 4 doses/day, titrate upwards up to 1mg/kg
* and if needed with Betablockers: Atenolol initial dose 0.5-1mg/kg 12 to 24 hourly

◊ Treat fluid overload

* salt restriction
* Furosemide 1-2 mg/kgIV 2- 4 times a day

◊ Refer patient

Post-renal AKI

Urgent catheterisation

Refer to tertiary facility

Non pharmacological

Avoid nephrotoxins

Adjust the doses of renally excreted drugs (penicillin, amoxicillin, Cotrimoxazole, Ciprofloxacin)

Nutrition

◊ Low salt diet
◊ Low potassium diet (no bananas, tomatoes, unboiled potatoes, citrus fruits)
◊ High caloric diet
◊ Breastfeeding can be continued

Complications

* Chronic kidney disease
* Pulmonary oedema
* Uraemic encephalopathy
* Bleeding diathesis

Refer all patients with AKI

Monitoring

- Check pulse, BP which is key to driving ultrafiltration at the glomerulus.
- Urine output hourly with daily fluid balance of intake and output plus weight chart, Match input to losses (urine, diarrhea, drains plus 500mL
- If hypovolaemic or hypotensive, resuscitate the patient per protocol
- Correct volume depletion with intravenous fluids, colloid, saline, or blood per protocol
- If patient is septic, take appropriate cultures and treat imperially
- Remove potential cause of sepsis when no longer required e.g. cannulae, catheters & NGT
- Take adequate calories (0.5g/kg/day). Increase when there is increased catabolism as in burns
- Daily U&Es follow-up
- ECG - In hyperkalaemia, the following changes may be seen; small or absent P-waves, wide QRS, shortened or absent ST segment, wide tall and tented T-waves, ventricular fibrillation, atrial fibrillation

Treat complication if any;

1. Hyperkalemia (potassium ≥6.5 mmol/l)

    - Stabilize the cell
    - Shift the potassium
    - Remove the potassium
    - C BIG K DROP

Stabilize the cell

- Give 10ml of 10% Calcium Gluconate over 10 minutes or Calcium Chloride 10ml over 10 minutes.
- Does not change the K level but calcium is cardiac stabilizer.

Shift the potassium into the cell

- Administer Soluble insulin 10 units and 50ml of 50% dextrose same time. Only if glucose is <250mg/dl. Insulin drops K by 1mmol/L in 1 hour. Glucose is given to maintain blood glucose levels.
- Give Sodium Bicarbonate – only if in acidosis. Sodium bicarbonate 8.4%, (50mEq) 1 ampoule slow IV push over 5 minutes. Cause temporary potassium intracellular shift.
- Buffers excess H+ ions and shifts K+ into cells. Bicarbonate do not work if no excess in H+.
- Administer Beta agonists: Salbutamol 10-20mg in 4mL normal saline Nebulized over 10 minutes. Salbutamol can lower K level by 1mmol/L in about 30minutes and maintain it for up to 2 hours. Very effective for renal patients that are fluid overloaded.

Remove the potassium

- Administer Furosemide 40-80mg IV push depending on hydration status. Facilitates renal removal. Helps to urinary excrete K especially in fluid overloaded patients
- Administer Sodium polystyrene Sulfonate (Kayexelate potassium binding resin) 15- 30g in 15-30mL (70% sorbitol) or water can be used to dilute Kayexalate, PO. Repeated every six hours as necessary. Facilitates gastrointestinal potassium removal. Pulls K+ out through GI tract byexchanging Na+ for K+ in the colon.

- MNEMONIC - C BIG K Drop

  Calcium gluconate
  B- Beta - 2 agonist
  IG- Insulin + Glucose K- Kayexelate
  D- Diuretics (furosemide) or Dialysis
  ROP- Renal Unit for dialysis of Patient

Stop all potassium supplements or potassium sparing diuretics i.e Slow K and ACE inhibitors / NSAIDS / ARBS.

Restrict potassium intake by restricting fruits, vegetables, meat and feezy drinks (Refer to dietary guide)

In severe AKI, Consider renal replacement therapy (RRT) such as haemodialysis and peritoneal dialysis if refractory despite medical treatment.

- Pulmonary Oedema
  - High fowlers position
  - Give high O2 flow by face mask
  - Administer venous dilator e.g. morphine 2.5 mg iv plus metocloopramide 10mg iv stat dose
  - Restrict fluids
  - Ashort trial of loop diuretics should be considered in patients with features suggestive of pulmonary oedema provided the patient has a reasonable perfusion pressure (MAP
  - ≥ 65 mmHg, SBP ≥ 110 mmHg).
    - ◊ Iv furosemide (80-160mg), 5- 10 minutes, slowly
  - Consider CPAP
  - if not improving urgent dialysis

- Bleeding

  - Fresh frozen plasma and platelets
  - Blood transfusion of packed cells to maintain HB > 10g/dL- the blood should be recent (atleast 5 days from the time of donation)
  - Tranexamic acid IV 0.5-1g BD or TDS
    - ◊ In paeds 10-15mg/kg, PO or IV, BD or TDS
  - Control of epistaxis
    - ◊ Ice pack placed on the forehead
    - ◊ Cotton with adrenaline placed in the nostril
    - ◊ Ballooning Foleys catheter placed at the back of nostril (should not be in for more than 30 minutes).

- Indication for dialysis in patients with uraemic gastritis

Pharmacological therapies

- Discontinue medications associated with diminished renal blood flow i.e ACEi/ARBs, NSAIDs (refer to MSTG)
- If BPs <130/80 hold antihypertensives unless clear medical indications
- If fluid overload, loop diuretics i.e. Furosemide 40 – 80mg may be used

Management of Hypovolaemia

- Promptly correct hypovolaemia with repeated boluses of 250 – 500 mL of isotonic crystalloid normal saline 0.9% up to an initial total of 2L over 2 hours.
- Strict monitoring of urine output
- Avoid potassium-containing solutions such as Ringer's lactate
- Large volumes of 0.9% sodium chloride can provoke a hyperchloraemic metabolic acidosis.
- Failure to maintain an effective blood pressure following this regime should raise the possibility of underlying sepsis or significant ongoing losses.
- Refer to seniors for assessment rather than continuing to prescribe increasing large volumes of fluid in the face of poor urine output.
- Fluid accumulation resulting in a positive fluid balance is a frequent event in critically ill patients with AKI.
- Fluid accumulation (>10% fluid weight gain) is independently associated with increased mortality, failure to improve renal function and is associated with worsening respiratory function.
- Euvolaemia is characterised by an absence of clinical signs of dehydration, haemodynamic stability and an absence of volume overload.
- Oliguria in this context often reflects established ATN and will not respond to increasing fluid challenges, which put the patient at risk of fluid overload.
- During oliguric phase, recovery of an adequate urine output is impossible to predict.
- Fluid intake should be restricted to a match daily output / loss.
- For patients who require ongoing maintenance IV fluids, one regime is to prescribe hourly crystalloid at a rate of the previous hour's urine output + 30 mL.
- Patients should be carefully assessed for signs of hypervolaemia. Features may include a raised JVP, peripheral and pulmonary oedema (clinically and radiologically).
- Calculation of total fluid balance from admission should alert clinicians to the potential of fluid overload.

Non-dialytic management of AKI

- Preventive measures
- Fluid and electrolyte balance
- Acid-base balance
- Nutritional balance
- Drug management
- Management of uremia
- Indications of dialysis in AKI

Dialysis indications: A, E, I, O, U	Clinical setting
A: Acid-base balance abnormalities	Severe metabolic acidosis from the accumulation of organic and inorganic acids
E: Electrolyte Imbalance	Electrolyte abnormalities in particular; Refractory Hyperkalaemia
I: Intoxications	Severe acute intoxications: Salicylates, theophylline, poisons, herbs

O: Overload of fluid	Volume overload refractory to diuretic therapy Pulmonary oedema

U: Uremia Severe accumulation of uremic toxins; complications such as altered sensorium, unexplained decline in mental status, pericarditis, uremic bleeding diathesis
Azotemia

Optimization of fluid balance

- Fluid volume status should be assessed both fluid depletion and fluid overload.
- Patients at risk of dehydration due to prohibited or poor oral intake should be prescribed maintenance IV fluids.

Optimization of blood pressure

Hypotension SBP < 110 mmHg or MAP <65 mmHg needs urgent assessment and treatment with IV fluid challenges and vasopressor agents where indicated.

Medication review

Temporary cessation of ACEi and ARBs is appropriate in patients with dehydration, hypotension SBP < 110 mmHg and / or deteriorating renal function.

Reducing the risk of contrast induced AKI

AKI secondary to radiological contrast media typically occurs within 72 hours of receiving such agents (Harty, 2014). The risk of contrast nephropathy can be reduced by temporary cessation of potentially nephrotoxic medication and adequate volume expansion

Prevention of Contrast Nephropathy

Identify risk
GFR ≤ 30 mL/min/1.73m2
GFR 30 – 60 mL/min/1.73m2 and risk factors
The CKD-EPI equation i.e., eGFR = 141 × min (Scr × 0.0113/k, 1)α × max(Scr × 0.0113/k, 1)-1.209 × 0.993Age × 1.018 [if female] × 1.159 [if black], where Scr is serum creatinine, k is 0.7 for females and 0.9 for males, α is -0.329 for females and -0.411 for males, min indicates the minimum of Scr/k or 1, and max indicates the maximum of Scr/k or 1.

Manage risk	Ensure adequate hydration - IV fluid 1L 12 hourly prior to exposure and then 1L post exposure Use of sodium bicarbonate pre-exposure if in emergence situation Omit nephrotoxic medications (ACEi/ARBs/NSAIDs/metformin) on day of procedure and do not restart until renal function stable at 48 – 72 hrs Use low osmolar agents in the lowest dose. Recheck renal function 48 – 72 hours following the procedure

Guidelines for COVID-19 In AKI

Kidney involvement in patients with coronavirus disease 2019 (COVID-19) is common. COVID-19-associated AKI (COVID-19 AKI) is associated with high mortality and serves as an independent risk factor for all-cause in-hospital death in patients with COVID-19.

Risks for AKI in COVID-19 Patients

- Pre-renal AKI is the most common contributor to AKI in COVID-19 patients.
- Volume depletion.
- Fever, history of vomiting and diarrhoea, dehydration from reduced fluid intake and bleeding are risks for AKI
- Older age, diabetes, hypertension, existing chronic kidney disease (CKD), heart failure &
- COPD.
- Mechanical ventilation is often required as lifesaving intervention in critically ill patients. However, mechanical ventilation also increases the risk of acute kidney injury threefold
- Ventilator induced kidney injury is believed to occur due to changes in haemodynamics that impair renal perfusion, neurohumoral mediated alterations in intra-renal blood flow, and systemic inflammatory mediators generated by ventilator induced lung injury
- Use of Vasopressors

Management

In the absence of haemorrhagic shock, use isotonic crystalloids rather than colloids (albumin or starches) as initial management for expansion of intravascular volume in patients at risk of AKI or with AKI

Open lung protective ventilation with low tidal volumes and high positive end expiratory pressure.

CHAPTER 4: CHRONIC KIDNEY DISEASE

CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. CKD is classified based on Cause, GFR category (G1-G5), and Albuminuria category (A1-A3), abbreviated as CGA.

Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012

GFR categories (ml/min/1.73 m²) Description and range			Persistent albuminuria categories Description and range		
			A1	A2	A3
			Normal to mildly increased	Moderately increased	Severely increased
			< 30 mg/g < 3 mg/mmol	30–300 mg/g 3–30 mg/mmol	> 300 mg/g > 30 mg/mmol
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			

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

Determination of albuminuria, for which the preferred method is the measurement of the albumin/creatinine ratio in the urine of an isolated urine sample due to its ease and good correlation with the excretion in the 24-hour urine.

Categories Albuminuria: A/C Ratio = Albumin-Creatinine Ratio in Isolated Urine Samples

Category	24-hour Albuminuria mg/24hr	A/C Ratio	Classification
A1	<30	<30	Normal to discrete
A2	30 – 300	30 – 300	Moderate
A3	>300	>300	Severe

Risk factors

i. History of diabetes. Diabetes injures the small blood vessels due to the high levels of sugar in the blood, therefore the kidneys cannot properly clean the blood which leads to high water and salt retention resulting to weight gain and swelling.
ii. Hypertension. High blood pressure results into increased intraglomerular pressure which then damages the glomeruli basement membrane. The impairment of the glomeruli causes high protein filtration which then increases the amount of protein in the urine.
iii. Cardiovascular disease (CVD)
iv. HIV or hepatitis B and C virus infection
v. Malignancy
vi. Autoimmune diseases
vii. Nephrolithiasis,
viii. Chronic glomerulonephritis
ix. Recurrent urinary tract infections.
x. Family history of renal disease
xi. Smoking
xii. Obesity
xiii. Abnormal kidney structure
xiv. Old age (above 60)
xv. Acute renal failure from which the patient fails to recover.
xvi. Low birth weight.

NB: Among all the risk factors, hypertension and diabetes remain the most common causes of chronic kidney failure. Diabetes contributing about 30% cases of end stage kidney disease in developing countries.

Multiple guidelines recommend that patients with diabetes or hypertension be screened annually for CKD. Furthermore, patients with other risk factors, including cardiovascular disease, older
age, history of low birth weight, obesity, and a family history of CKD, warrant consideration for screening (Ferenbach et al., 2017).

Presenting Signs and Symptoms According to CKD Stages and Their Physiology

Stage 1

Usually there are no symptoms that indicate kidney damage. At this stage the GFR maybe normal or high level greater than 90ml/min/1.73m2. When presented with symptoms, patients are screened for other conditions such as DM or BP.

Symptoms may include;

i. Tiredness- it comes in as a result of impurities and toxins in blood. Anaemia also causes weakness and fatigue.
ii. Trouble sleeping due to presence of toxins in blood.
iii. Dry itchy skin-
iv. Frequent micturation
v. Haematuria (blood in urine)
vi. Foamy urine - – it is because the presence of protein in urine.
vii. Puffiness around eyes – this is because of leakage of large amount of proteins in urine.
viii. Swollen ankles and feet – decreased kidney function causes sodium retention, consequently water retention causing edema.

Stage 2
The signs are similar to that of stage I but are different in severity but their physiology is the same. **(Refer stage one physiology of presenting signs and symptoms)**

- Swollen eyes, face, hand arm, ankles and feet and testes in males
- Tiredness and fatigue
- Frequent micturation at night and sometimes could also be decreased urine output.
- Insomnia.
- Dry, itchy and hyper pigmented skin
- Severe BP, foamy urine and blood in urine.

Stage 3
The signs are similar to that of stage 2 but are different in severity but their physiology is the same.

- Swelling and oedema of the extremities.
- Foamy urine – damage of kidney cause of leakage of proteins in urine.
- Haematuria
- Fatigue and weakness
- High BP –– Fluid retention as a result of damaged kidneys raises blood pressure and also lack of kallikrein and postaglandins.
- Loss of appetite
- insomnia
- Muscle cramp,
- Itchy skin
- Restless legs.

Stage 4
- There is high BP
- Haematuria
- Nausea and vomiting – due to high toxins in blood
- Tiredness and fatigue
- Bad taste in mouth (metallic taste)- due to high Blood Urea Nitrogen (BUN)
- Itchy skin

Stage 5
- Uremia- elevated concentrations of urea in the blood associated with fluid, electrolyte and hormone imbalances. Urea causes a range of direct ad indirect toxic effects on different tissues. Findings include:
  - ◊ Skin: Pruritus, Uremic Frost
  - ◊ Eyes: icteric sclera (yellow eyes)
  - ◊ Mouth: oral lesions
  - ◊ Cardiovascular: pericardial rub or effusion

Gastrointestinal: abdominal pain, nausea, vomiting,

- Uraemic Bleeding- High levels of Urea Causes Platelet dsyfuntion (Bloody stools, vomiting blood bleeding from venipunctures and nose bleeding)
- Mental status changes due to increased Urea (drowsness, seizures, confusion and Coma).
- Uremic fetor -urine like odor due to uremia

Investigations

These include; Blood Studies, Urine Test, Radiology Imaging, Intravenous Pyelogram and Kidney Biopsy.

1. Blood Studies

- **Serum creatinine**
- The most important determinant of renal function. Increase in serum creatinine level indicates decreased renal function. Since creatinine is also excreted by the kidneys. Creatinine excretion is not affected by dietary or fluid intake and is thought to be a more accurate indicator.
- **BUN**
- Urea is an end product of protein metabolism and is normally excreted through the kidneys, any renal function compromise results in increased plasma urea level (Polaski and Tatro, 1996)
- **Full blood count**
- May help provide some information about renal function as well as the progress of the disease. Decreased hemoglobin level indicates decreased erythropoietin function of the kidney.

2. Urine Test

i. Urine dipstick (normal findings)

Table 4: Urine Characteristics

Color Pale-

yellow/deep amber (normal)
Cloudy may indicate infection
Foamy when albumin is present
Dark-red (smokey)may indicate bleeding from kidneys
Bright-red urine – bleeding from lower urinary tract
Dark-yellow urine presence of urobilinogen or bilirubin
Dark-brown may indicate myoglobin, malaria

Opacity Clear

Specific gravity 1.002- 1.035 Indicates urine concentration. A higher specific gravity

indicates very concentrated urine evidence of fluid depletion

		and dehydration.
pH.	4.5-8	
Glucose	Negative	
Ketones	Negative	Ketones are produced when the body's fat store are metabolized for energy mostly due to uncontrolled diabetes, pregnancy, and fasting.
Proteins	Negative	The protein that is usually checked is albumin and increased levels indicates abnormal glomerular permeability, decreased tubular reabsorption or an overflow or protein in the plasma.

Bilirubin	Negative	They are found in the urine when there is hepatic biliary obstruction.
RBCs	None-3	
WBCs	None- 3	
Casts	Negative	
Crystals	Negative	

i. U+Es (increased urea and increased creatinine)

3. Radiology Imaging

Radiology imaging is very helpful in assessment of renal failure patients by the use of different imaging modalities.

i. Ultrasound

It distinguishes AKI and CKD by evaluating renal size, renal texture changes and cortical thickness

Renal Size: Kidney length is the most clinical useful measurement of the kidneys in patients with renal failure. The increase size may result from infiltrative diseases like multiple myeloma amyloidosis lymphoma or renal thrombosis.

Renal Texture Changes: The echogenocity or brightness of the renal tissue on ultrasound signifies renal failure such as hypoechoic or dark changes of the kidney texture suggests AKI while hyperechoic or brighter texture of the renal tissue suggests CKD.

Cortical Thickness: The increase in cortical thickness also suggests AKI due to disease infiltration within renal cortical while dilatation of calyceal indicates hydronephrosis due to blockage of urinary tract by stone(s) in CKD.

ii. Doppler

It helps to have a resistive indices (RI) of renal blood flow velocity within pre-renal or intra-renal veins or arteries in patient with renal failure. The RI greater than 0.75cm/s is more likely associated with AKI. RI is calculated by the measurement of (peak systolic velocity minus peak dystolic velocity divided by peak systolic velocity).

iii Computed Tomography (CT) –Scan: Mainly to provide the location of the stone(s) in CKD and the degree of stenosis in AKI.

iv. Abdominal Magnetic Resonance Imaging (MRI)

It demonstrates changes in renal medullary oxygenation in response to ingestion of different drugs and water lodding in AKI, also in disease states such as obstruction and vascular disease in CKD. It further characterizes mass lesions in the kidney, such as renal cell carcinoma.

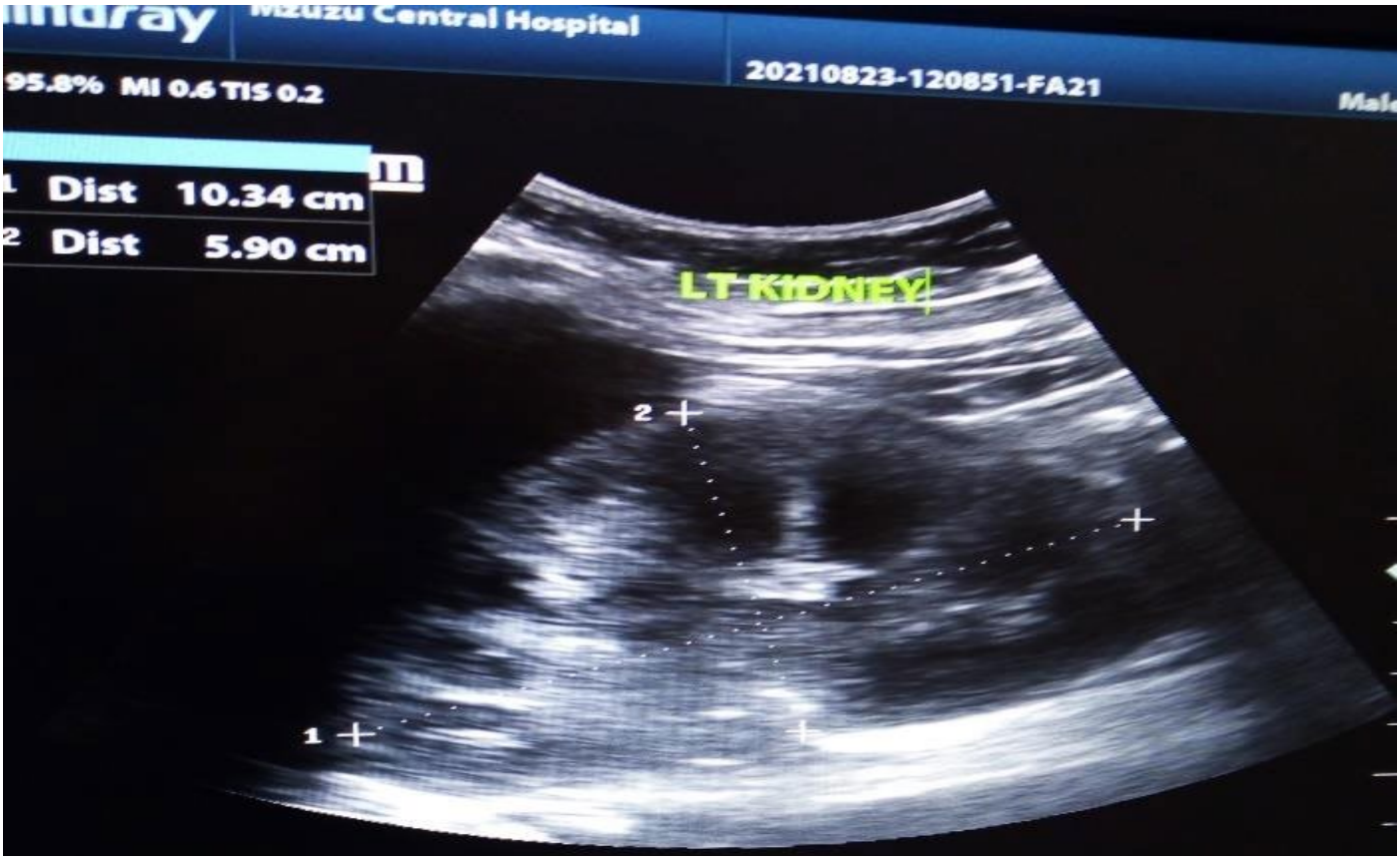
4. Intravenous Pyelogram

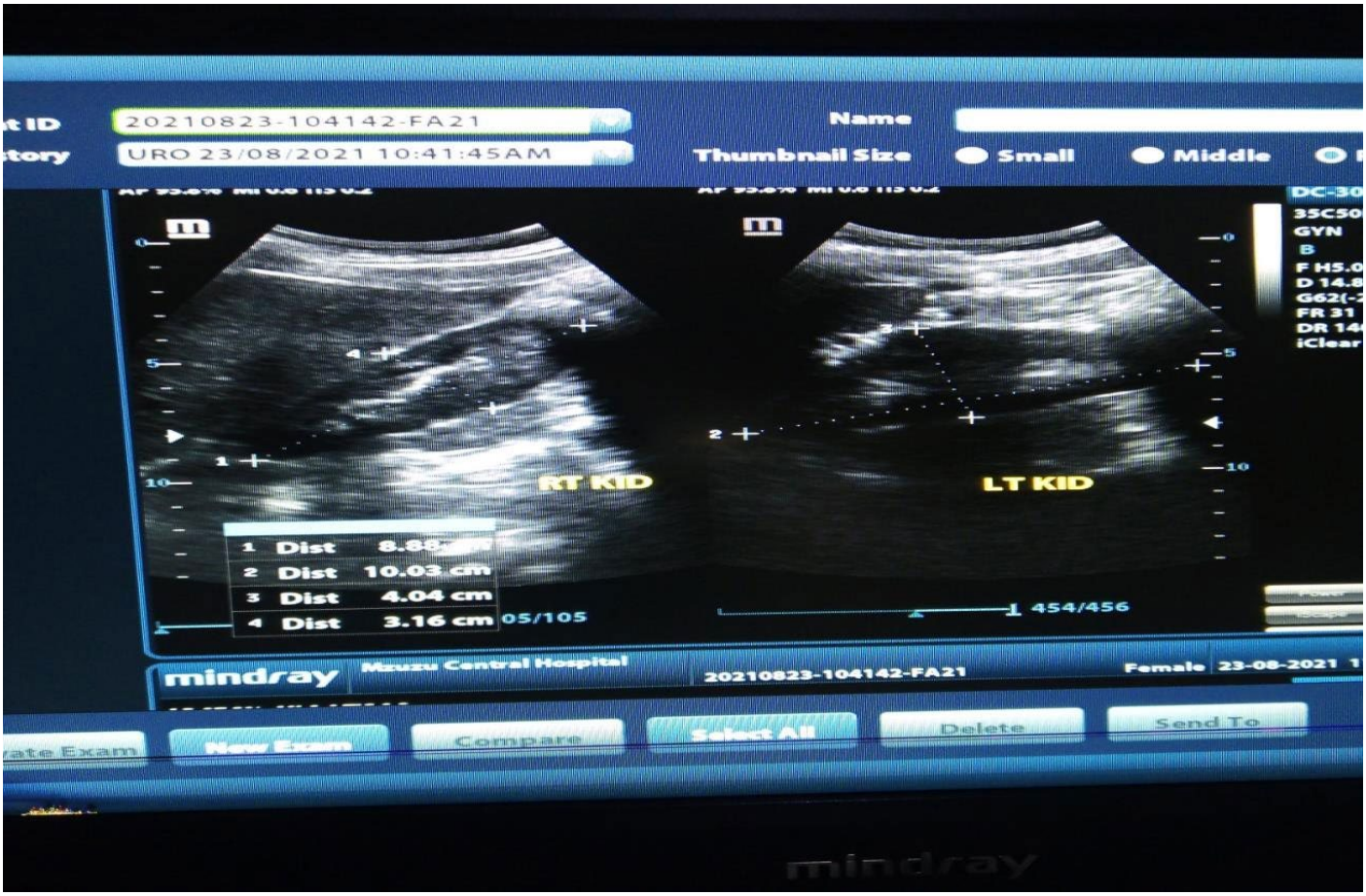
Serial KUB xrays are taken to visualize the flow of contrast through the kidney, bladder and ureter. It can help to identify point or level of obstruction. A contrast is injected into the vein.

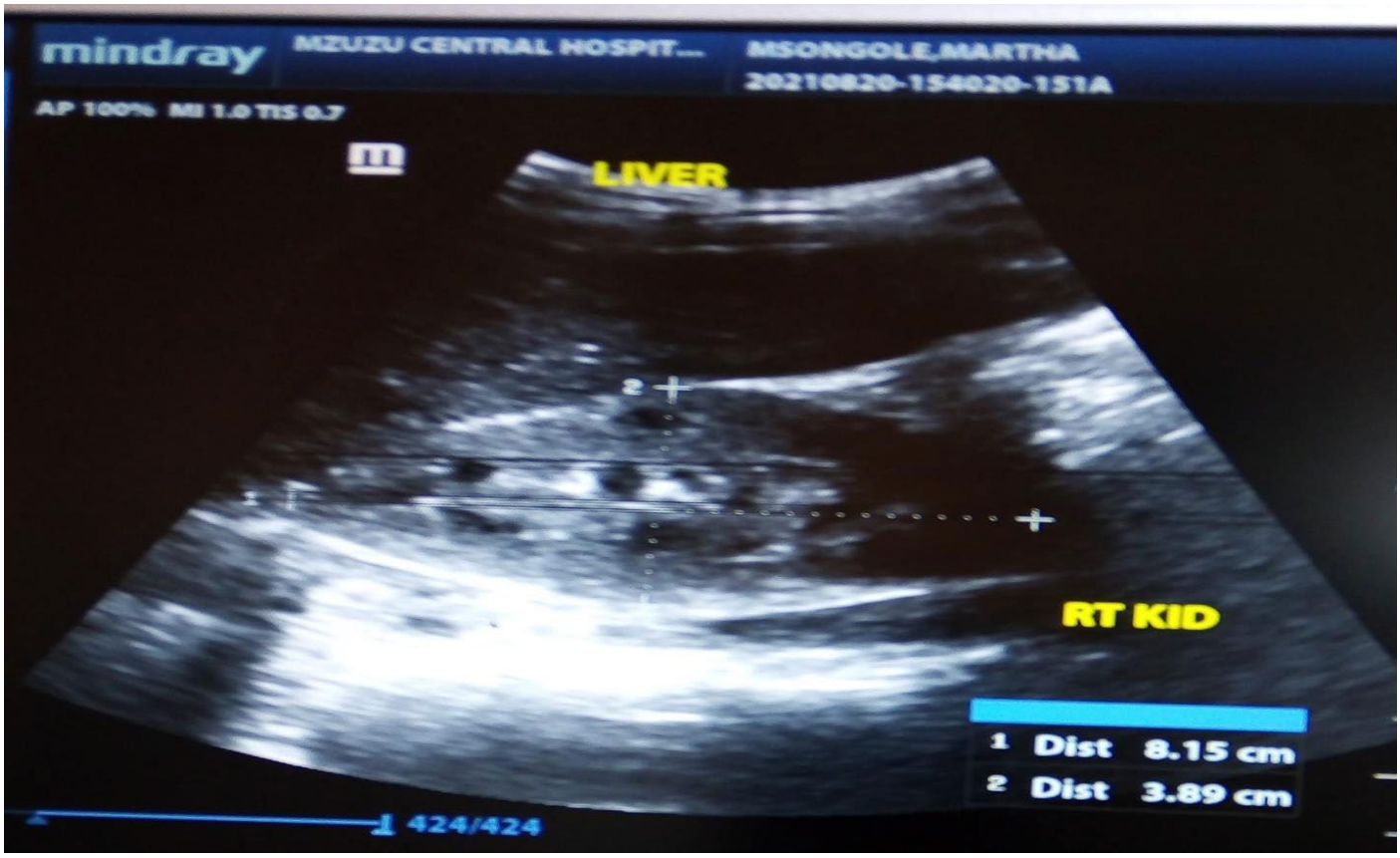
5. Kidney biopsy

This test is done to control specific types of kidney problems or to see how much damage has occurred in order to plan for a further treatment.

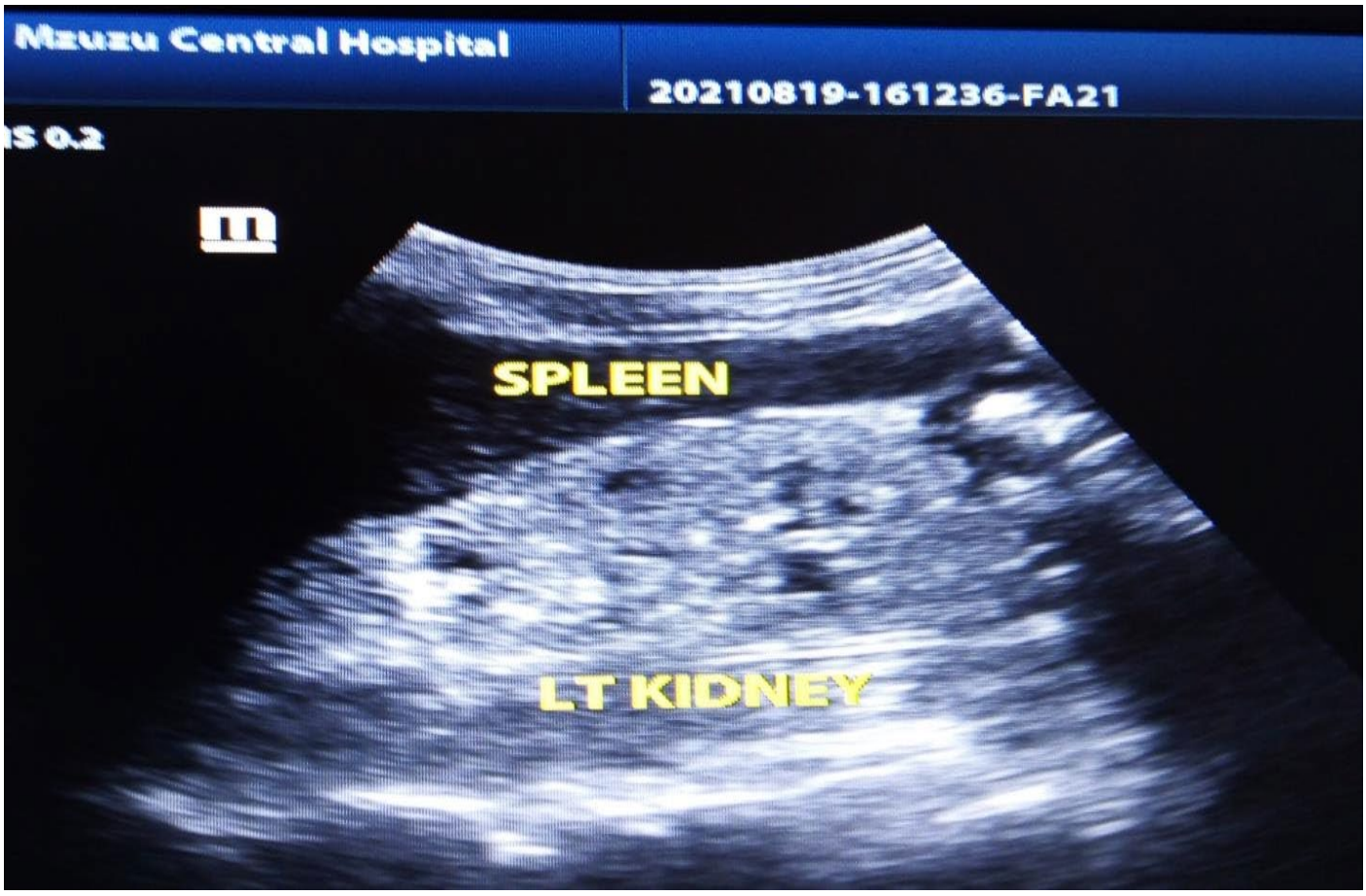
RENAL SIZES (VARIES)



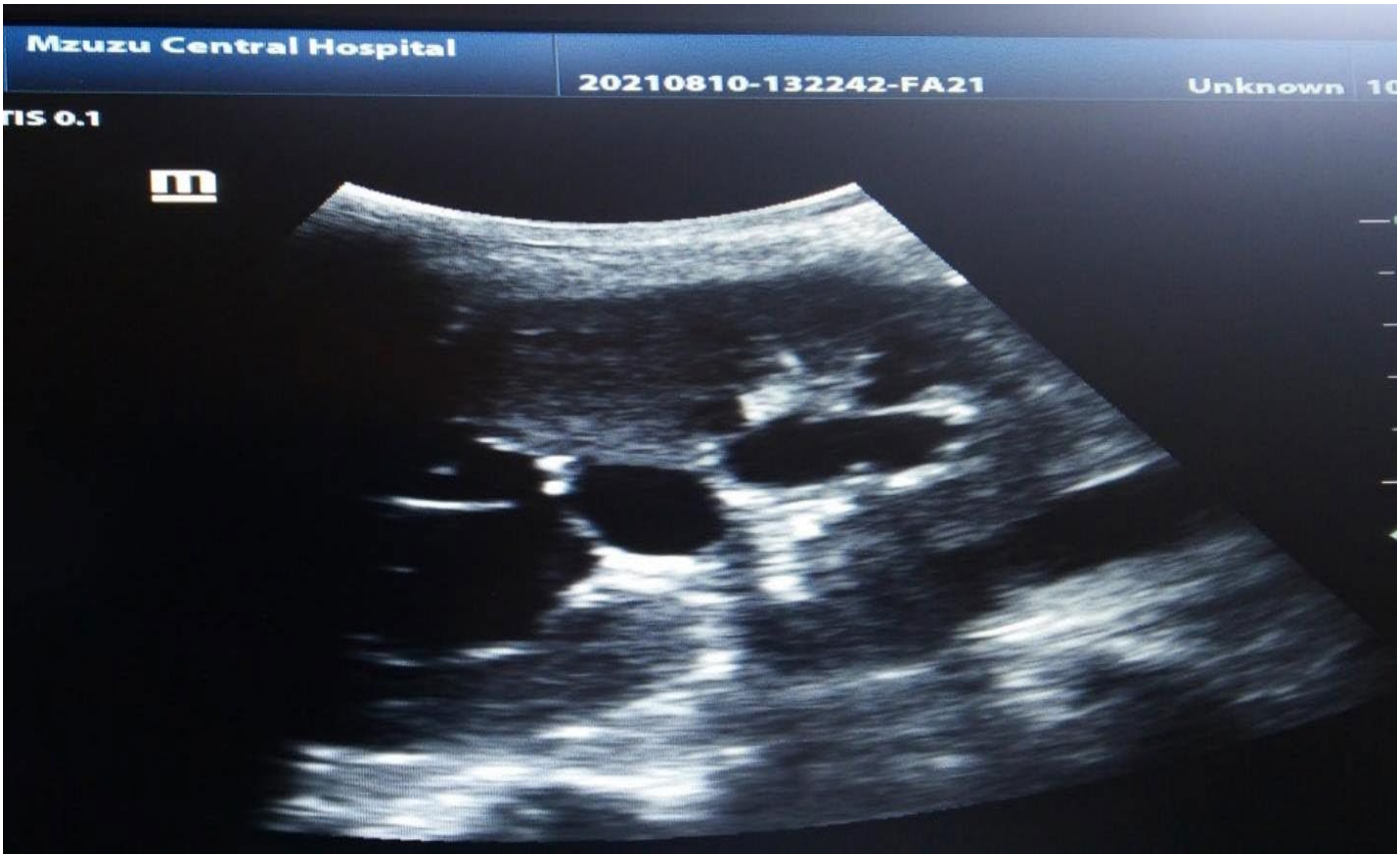




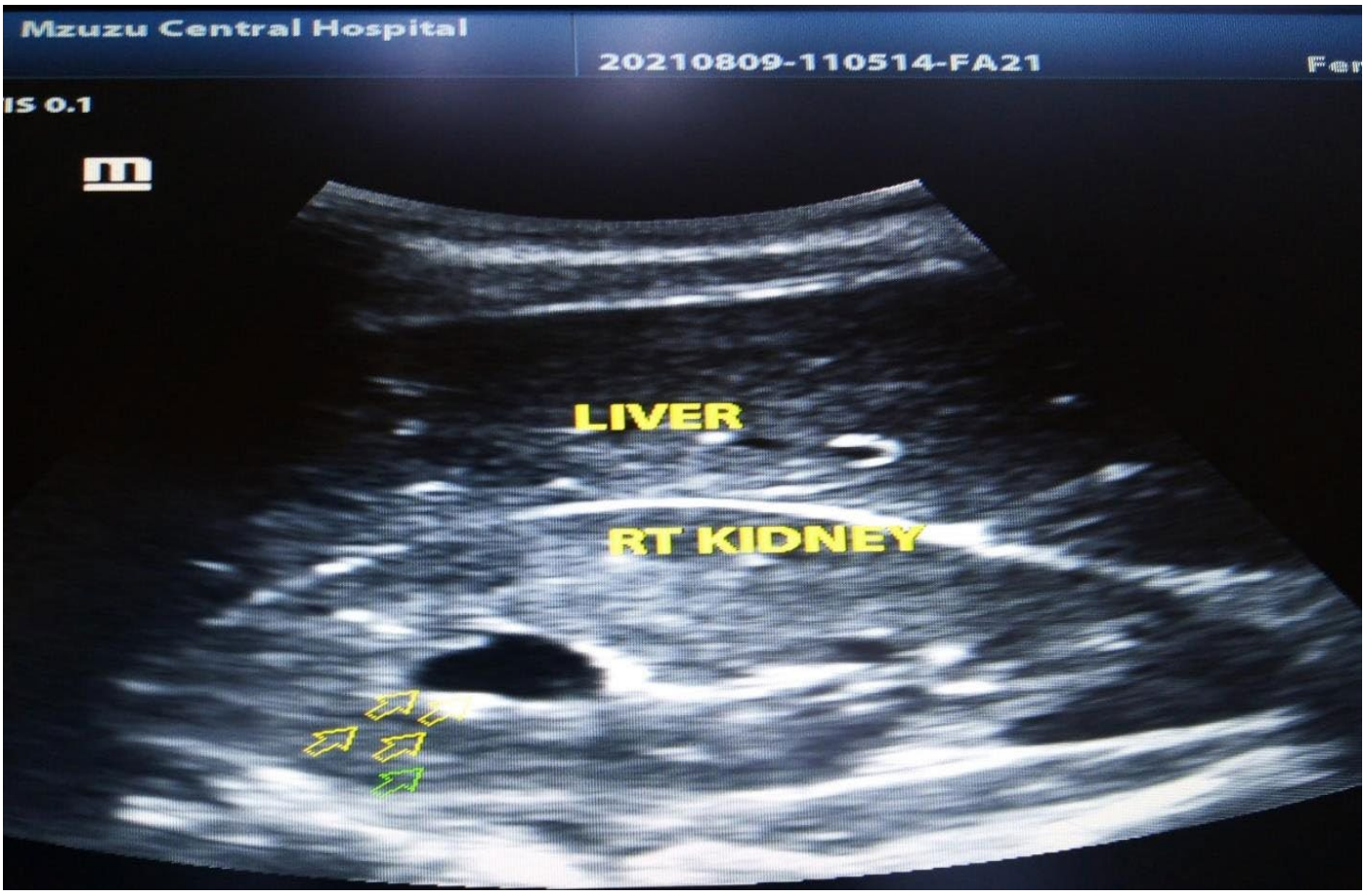
Cortical Echogenicity



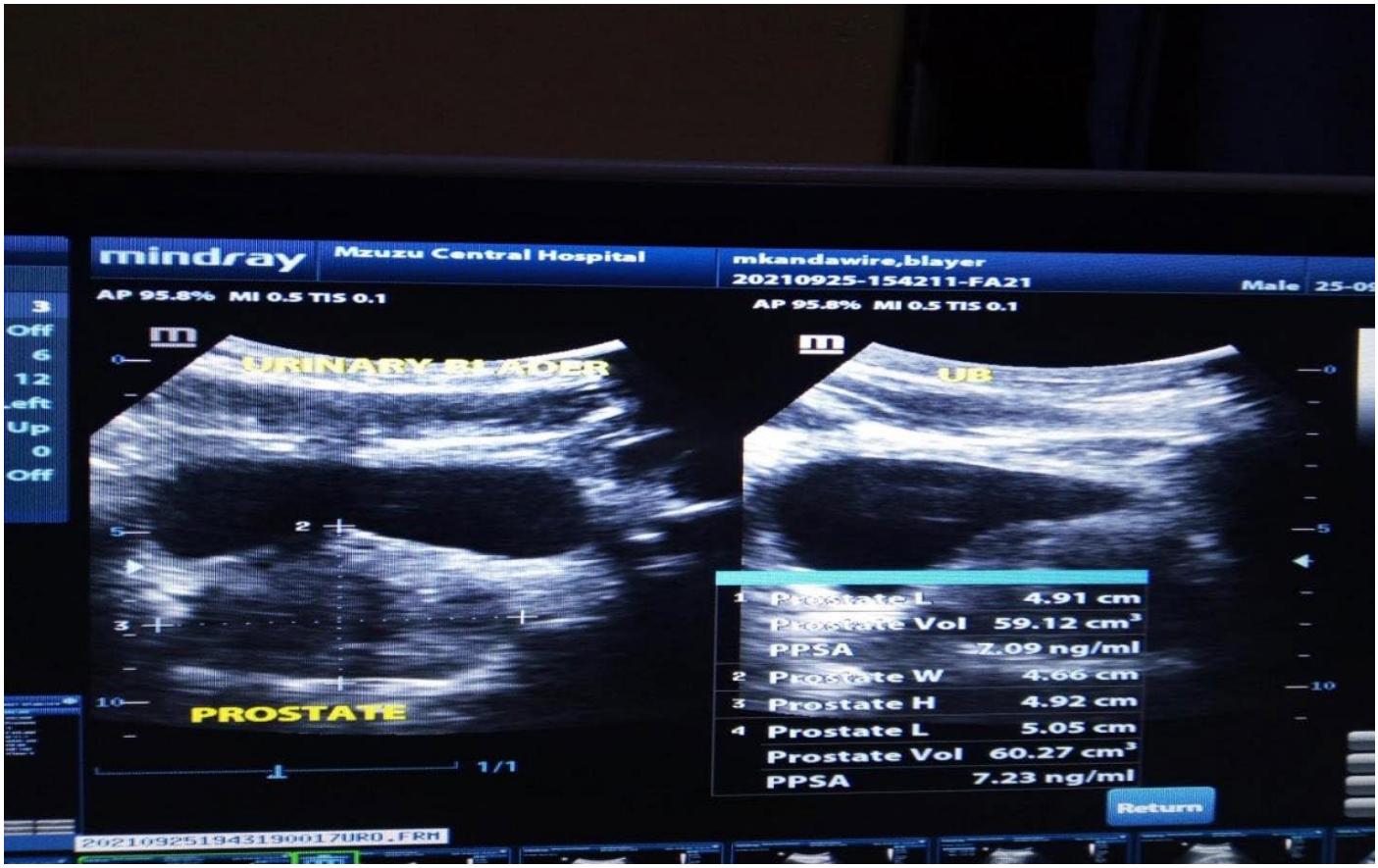
HYPERECHOIC (LOSS OF CORTICAL-MEDULARY BOARDERS)



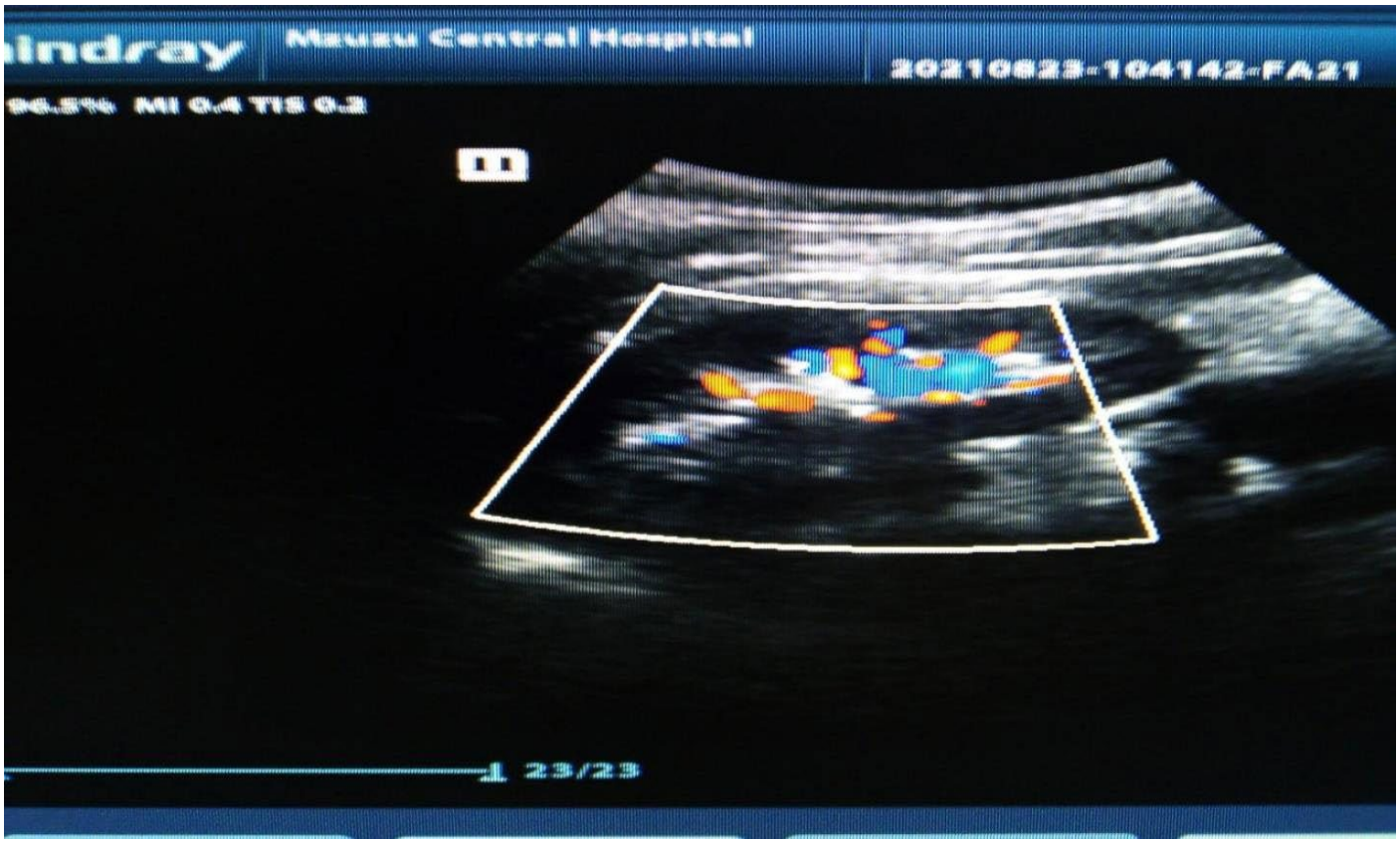
HYDRONEPHROSIS



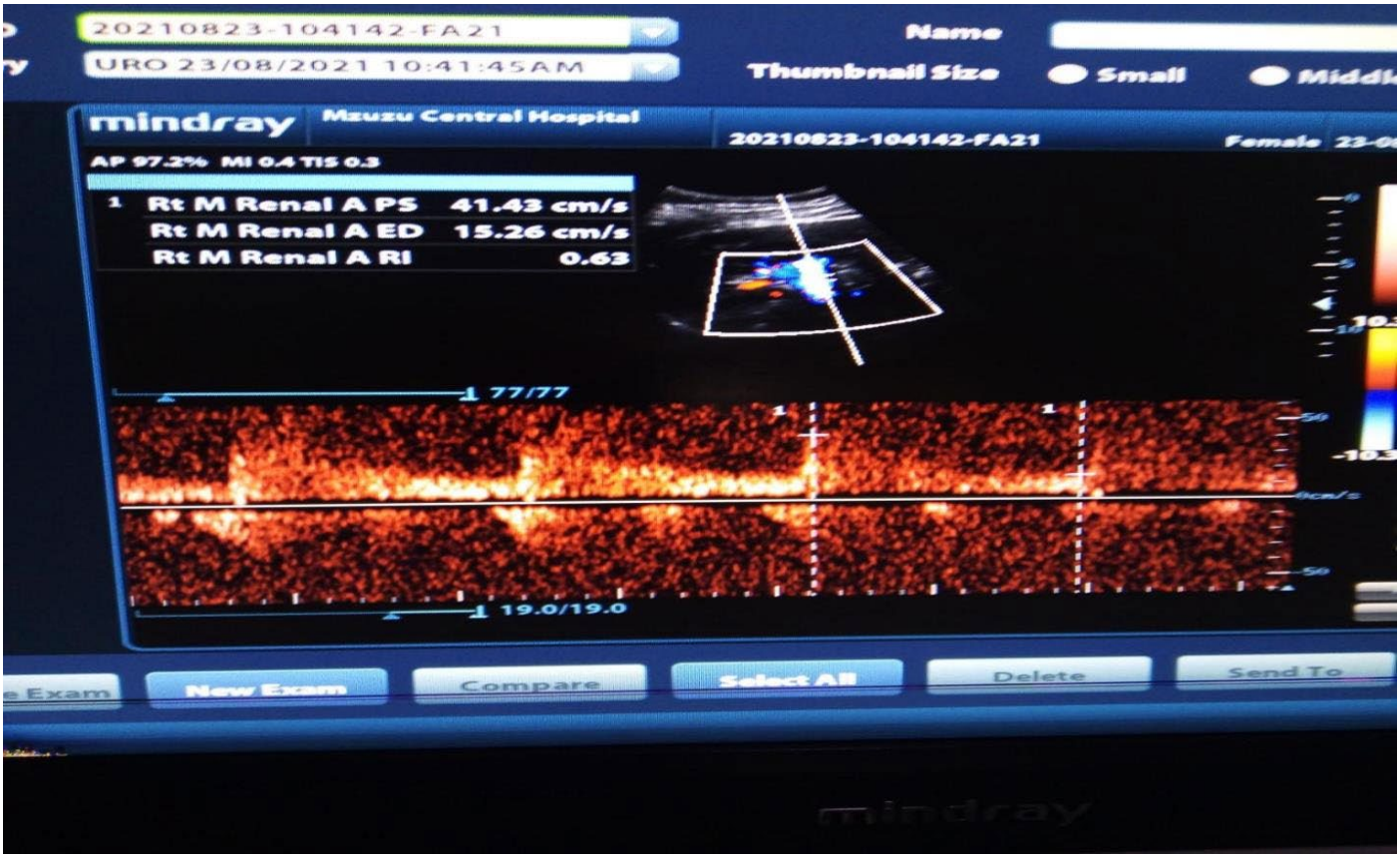
RENAL STONE



ENLARGED PROSTATE COMPRESSING UB
Resistive index (DOPPLER)



RENAL VASCULARITY



MEASURING RI

CHAPTER 5: MANAGEMENT OF CKD ACCORDING TO STAGES

It is suggested that people with progressive CKD should be managed in a multidisciplinary care setting. In terms of dietary management, it is recommended that individuals with CKD receive expert dietary advice and information in the context of an education program, tailored to severity of CKD and the need to intervene on salt, phosphate, potassium, and protein intake where indicated (KDIGO, 2013).

Note 1: At GFR <60 ml/min interventions to retard or prevent the progression of CKD should be instituted.

Note 2: At GFR <30 ml/min, options for renal replacement need to be considered. Consider preparing vascular access and working up for transplantation.

Note 3: At GFR <15 ml/min, be vigilant for complications such as hypertension, fluid overload, electrolyte disturbances and malnutrition.

Note 4: Avoid blood samples, tourniqueting, cannulating and putting a drip on non dorminant arm to prevent fibrosis of blood vessels. The fibrosis will affect the creation of arteriovenous fistulas (permanent haemodialysis vascular access)

Note 5: Need for procurement of analysers that will automatically calculate eGFR

Goals of Chronic Kidney Disease Management

- Delay progression to CKD
- Minimize severity of the complications

Stage 1 And 2

Non-pharmacological management

Generally, there is no diet changes for stages 1 and 2, except for a heart healthy diet (adequate fiber, <30% of kcals from fat, <10% from saturated fat) and diet changes needed for comorbidities e.g. diabetes, hypertension.

The recommended daily energy allowance among all CKD patients is at least 30 kcal/kg (30 – 35 kCal/kg/day) of ideal body weight. Education on diet should be based of patient's food availability. Educate on the need for adequate proportioning of carbohydrates and other food groups in a balanced diet

Pharmacological management

- Adequate Blood pressure control- optimum blood pressure 130/80mmHG
  - ◊ Initiate first-line therapy with an angiotensin converting enzyme inhibitor (ACEI) or an angiotensin II receptor blocker (ARB). Add a thiazide diuretic in combination with an ARB if additional reduction in proteinuria is needed.
  - ◊ Nondihydropyridine calcium channel blockers are generally used as second-line antiproteinuric drugs when ACEIs or ARBs are contraindicated or not tolerated.
  - ◊ Stop potassium sparing drugs

- Adequate Blood sugar control
  - ◊ Screen annually for diabetes with CKD in patients 5 years from diagnosis of type 1 diabetes or from diagnosis of type 2 DM Measure ACR, sCR & eGFR
  - ◊ Ensure adequate control with the indicated hypoglycemic agents: Insulin, glibenclamide, metformin etc.
  - ◊ Metformin: Continue in people with eGFR ≥45 mL/min/1.73 m2; review in those with eGFR 30–44 mL/min/1.73 m2, and discontinue in people with eGFR less than 30

CKG Stages 3 And 4

Non-pharmacological Management

For stages 3 and 4 (when GFR < 60 ml/min), there is the need to intervene on salt, phosphate, potassium, and protein intake.

Pharmacological Management

Blood pressure control

- ACE inhibitors and ARBs should be used . Consider second line therapy in Hyperkalemia: diltiazem 60mg tds (max 360mg/day) Verapamil 240-480mg daily in 3 divided doses.
- Stop potassium sparing diuretics

Blood sugar control

- CKD decreases renal elimination of insulin increasing the risk of hypoglycemia.
  Dose reduction of 25-50% of insulin may be required.
- Metformin should be discontinued when EGFR is less than 30 and replaced with another antihyperglycemic agent.

Management of anemia

- Initiate erythropoietic-stimulating agent (ESA) therapy 2000-10000 IU weekly in all CKD patients with low Hb less than 9 g/dL (Target Hb 9-12g/dl)
- Iron deficiency is the primary cause of resistance to treatment of anemia with ESAs. Iron supplementation is required by most CKD patients to replete iron stores depleted by ongoing blood loss and increased iron demands. Ferrous sulphate 200mg OD
- Subcutaneous (SC) administration of epoetin alfa is preferred. 10000-40000 iU weekly
- Darbepoetin alfa has a longer half-life than epoetin alfa and prolonged biologic activity. Doses are administered less frequently, starting at once a week when administered IV or SC. 40-300mcg sc

Management of renal osteodystrophy

- Evaluate Ca, P, iPTH every 2 weeks initially in stages 3-4 until normalized, then every 3-12 months depending on stage and trends.
- Calcitriol 0.25-0.50 mcg OD or Doxercalciferol 1.0 mcgOD
- Calcium acetate 1.0-1.5g OD or Calcium carbonate 1.0-1.5g OD

- Vitamin D 50,000U weekly or 250U daily
- Non calcium based phosphate binders i.e Sevelamer

Management of Metabolic acidosis

- Sodium bicarbonate supplements 0.5-2.0mEq/Kg OD
- Alternatively baking soda can be used. 1 tsp (2g) is equivalent to about 23mEq Na and HCO3 (in a glass of water).

Management of Hyperlipidemia

- Lipid lowering therapy in CKD is recommended if triglycerides >200mg/dl and/or LDL>100mg/dl. Lipid evaluation should be assessed at least once a year
- Treatment includes: Artovastatin 20mg nocte, Simvastatin 20mg nocte (can be increased to 80mg)
- Management of Disease Hyperkalemia (see flow Chart)

Hypertensive Emergencies in Kidney Disease

Life threatening elevation in BP necessitating emergency treatment within one hour to prevent severe end organ damage and death

- Systolic BP >180mmHg or diastolic BP >120mm Hg
- Hypertension can be a cause or an effect/consequence of CKD.
- Excessive arterial pressure leads to endothelial damage of arterioles and capillaries.
- Damages blood vessels making removal of body wastes difficult
- The ischemic of glomerular promotes further irreversible renal injury
- Symptoms may include blurred vision, confusion, decreased alertness, chest pain, headache, nausea and vomiting, numbness, reduced urine output, SOBs, seizure, weakness of arms, leg face or other areas
- Physical exam reveals high BPs, swelling in lower legs and feet, abnormal heart sounds and fluid in lungs, changes in thinking sensation and reflexes
- Eye exam will reveal bleeding of the retina
- Tests to determine kidney damage/impairment include ABG, creatinine, urea, electrolyte levels, kidney USS and Urine dipstick to detect haematuria or proteinuria
- Chest X-Ray may show congestion in the lungs and an enlarged heart

Management of hypertensive emergency in CKD

- Control of hypertension is important to prevent CKD progression, end organ damage as well as CVD risk.
- Patient will need to stay in the hospital until BP is under control.
- Admit patient in HDU. Create a calm, quiet atmosphere, conducive to ample rest
- BP should be measured in both arms
- Target BP in CKD <130/80 to improve clinical outcomes
- Reduce SBP to a maximum of 25% within the first hour, if clinically stable, lower BP to 160/100 – 110 mm Hg over the next 2-6 hours and then cautiously to normal over the following 24-48 hours.
- NPO initially, later a sodium restricted diet
- Obtain accurate intake and output, along with daily weights

Pharmacological Management

- Certain pharmacological therapies are renoprotective and/or cardioprotective
- Must be considered when instituting therapy i.e. Labetolol 40-80mg/hour IV, Hydralazine 5 - 20 mg IVdrugs Oral Hypertensives should be started together with IV
- Monitor U & Es, urinary output
- If BP remain uncontrolled, evaluate for a separate secondary cause of hypertension.

Oral drugs

- ACE inhibitor i.e. Enalapril 5mg OD or captopril 12.5mg BD. In pregnancy, give methyldopa, if no response, add hydralazine. Don't give ACEs or ARBs in stage 4 and 5
- Calcium channel blocker i.e. Nifedipine long acting 20mg BD
- Thiazide diuretic – Hydrochlorothiazide 25mg OD
- If pulmonary oedema or fluid overload give loop diuretic i.e. Furosemide
- Beta Blocker such as carvedilol 12.5mg BD increasing to 25mg OD increase to 50mg, alternatively use Atenolol 25mg OD

IV drugs

- Furosemide 40mg - 80mg IV. Watch for volume depletion
- Labetalol 10mg-20mg IV bolus, repeated after 10 minutes, then infuse at 2mg/min -use with caution if COPD/Asthma or bradycardia
- Hydralazine 5mg IV bolus, recheck BP at 0, 5 mins then 15 minutes.
  - ◊ 5mg Hydralazine boluses can be repeated every 15 minutes until 20mg is finished
  - ◊ If blood pressure is still not contolled to daistollic of 110mmhg, put Hydralazine 40mg infusion into 250-500mls of normal saline to run for 4 hours if Labetalol is out of stock
  - ◊ Stop Hydralazine when Diastollic BP is just 110mmHg
  - ◊ If there is a drop of blood pressure by 30%or more give a bolus of 250mls of normal saline IV
  - ◊ Before giving Hydralazine or Labetalol make sure you have inserted a high bore cannula and drip just incase of hypotension
- Close monitoring of BP every 15minutes, 30 minutes and hourly watch for side effects of medications, observe ECG for the T-wave inversions that occur with rapid BP reduction

In Kidney failure; dialysis may be required

If Proteinuria

- Start with ACEI or ARB i.e. captopril, lisinopril +/- calcium channel blocker.
- If not controlled add on;
  - ◊ calcium channel blocker i.e. Nifedipine
  - ◊ Thiazide/ thiazide like diuretic
  - ◊ Beta-blocker i.e. carvedilol or labetolol
  - ◊ Alpha blocker

If No Proteinuria; multiple groups of drugs may be used at once

- Angiotensin Converting Enzyme inhibitors (ACEi) or Angiotensin II Receptor Blockers
- Calcium Channel Blocker i.e diltiazem 90-120mg BD
- Thiazide/thiazide like diuretic

- If not controlled add on;
  - ◊ Beta-blocker
  - ◊ Alpha blocker

Non-Pharmacological Therapy

- Dietary salt restriction less a teaspoon/day will reduce SBP by 2-8mmHg
- Staying at a healthy weight by maintaining normal BMI (18.5- 24.9). Engage in regular aerobic physical activity i.e. brisk walking at least 30 mins / days in a week
- Get enough sleep 7-8 hours
- Explore stress reducing activities
- Adherence to prescribed antihypertensives
- Adopt the Dietary Approache
- Stop alcohol consumption

Management of Uremic bleeding in CKD

Uraemic bleeding syndrome is a recognized consequence of renal failure and can result in clinically significant sequelae.
Typical presenting symptoms include

- ecchymoses,
- purpura,
- epistaxis,
- bleeding from venipuncture sites
- gastrointestinal and intracranial bleeding might also be evident

Prevention and treatment options include

- Resuscitate the patient and follow ABCDE approach
- Tranexamic acid IV 0.5-1g BD or TDS
- Cryoprecipitate 10 units given over 30 minutes IV i.e FFPs and Platelets
- Desmopressin (DDAVP) 0.3 µg/kg to 0.4 µg/kg intravenously or subcutaneously as a single injection
- Conjugated estrogens 0.6 mg/kg intravenously over 30–40 min once daily for 5 consecutive days. The time to onset of action for conjugated estrogens is about 6 h; maximum effect is evident at 5–7 days with a duration of approximately 14–21 days.
- Control of epistaxis
  - ◊ Ice pack placed on the bridge of the nose
  - ◊ Cotton with adrenaline placed in the nostril
  - ◊ Ballooning Foleys catheter placed at the back of nostril (should not be in for more than 30 minutes).
  - ◊ Dialysis (anticoagulant–free dialysis should be considered)

Above methods may be used alone or in combination.

Chronic Kidney Disease and COVID- 19

- Evidence accumulating over time showed that although SARs COV-2 infection primarily causes respiratory illness with highly clinical manifestation, other organs may be damaged by the virus, the kidney being one of the main site of complication
- Patients requiring with end-stage kidney disease and kidney transplant recipients have been first identified as a sub group at higher risk for poor outcome and often presents atypical clinical features that constitute an additional challenge
- CKD and kidney failure are important comorbidities that are associated with unfavourable outcomes in patients with corona virus
- Patients with CKD also have a disproportion burden of other comorbidities such as coronary artery disease, hypertension, diabetes mellitus which are associated with more severe presentation of COVID
- Recently chronic kidney disease has been demonstrated to be a key risk factor for covid- 19 mortality as well as, with a clear association between the level of dysfunction and mortality rate
- Advise patients to continue taking their medicines including ACE inhibitors, angiotensin receptors blockers, diuretics as normal unless advised to stop by health personal. This includes CKD patients who have symptoms of COVID -19
- For the patients with CKD and suspected or confirmed COVID -19 review the use of medicines taking into account whether any have the potential to adversely affect renal function
- Reassess renal function in patients with CKD who have recovered from COVID -19. based the urgency of reassessment on the patient's GFR category, comorbidities and clinical circumstances
- For patients who are stable on treatment, assess whether it is safe to reduce the frequency of routine blood and urine tests during the COVID -19 pandemic just to avoid exposure to the hospital. Take into account any comorbidities and whether their CKD is progressive.
- During admission at Emergency treatment unit (ETU) CKD patients may need high flow oxygen as any other COVID -19 patient.
- Manage with the following medications;
  - ◊ Dexamethasone iv 6mg OD for 10 days
  - ◊ Low molecular heparin 60mg OD for 10 days
  - ◊ Zinc 20mg OD for 10 days
  - ◊ Omeprazole 20mg OD 10 days.

- CKD patients admitted with severe symptoms of COVID -19 despite above mentioned medications can also be managed according to the signs and symptoms they have presented with.

Management of Cardiovascular Disease in CKD

Patients with Chronic Kidney disease have more frequent and severe cardiovascular disease. The frequency is significantly higher in the early stages of CKD (stages 1-3) but in advanced CKD stages (4-5), patients have a markedly higher risk and cardiovascular disease is a leading cause of death as compared to end stage kidney disease.

CKD and CVD can initiate and perpetuate one another which means that interventions aimed at reducing the risk of CKD will also help reduce the risk of CVD and vice versa. The spectrum of CVD in CKD include:

- Ischemic heart disease
- Congestive heart failure
- Arrhythmias
- Peripheral vascular disease
- Myocardial fibrosis
- Calcification of cardiac valves

The following interventions are believed to reduce the risk of CVD morbidity and mortality in patients with CKD.

Treatment of Hypertension
Lowering of blood pressure is effective in preventing cardiovascular events and mild CKD. (For blood pressure management refer to the flow charts)

Statin Therapy
Lowering of lipid using statins is also beneficial in preventing CVD in patient with CKD as well those with normal renal function.

Bone Mineral Management
Bone and mineral metabolism are a complication of CKD. Hyperphosphatemia is a risk factor for CVD morbidity and mortality in patients with CKD. Standard dialysis treatment fails to maintain recommended levels of these parameters hence the need for pharmaceutical intervention. Phosphate binders and dietary counselling

Management of Anemia
Erythropoiesis stimulating agents reduce the need for blood transfusion. In patient with CKD, erythropoietin treatment should seek to achieve haemoglobin level of between 9-12g/dl. A higher haemoglobin level of more than 12g/dl is associated with higher rates of stroke and trends toward harm for total cardiovascular events.

Management of Diabetes In CKD

Diabetic Nephropathy in CKD
Diabetic nephropathy is a complication of DM that affects kidney structure and function, presents with;
- Persistent albuminuria (>300mg/d) that is confirmed on at least two occasions 3-6 months apart.
- A consistent decline in glomerular filtration rate
- Elevated arterial blood pressure (HTN)

Workup
- Urinalysis for proteins
- Urine protein creatinine ratio
- Blood tests for Urea and Creatinine, potassium
- Calculation of GFR to monitor the progression of kidney disease
- Renal ultrasound: observation of kidney size which is usually normal to increased in early stages and then decreased or shrunken in chronic renal disease.

Glycaemic monitoring
- Monitoring long-term glycaemic control by HbA1c twice per year is reasonable for patients with diabetes.
- HbA1c may be measured as often as 4 times per year if the glycaemic target is not met or after a change in antihyperglycemic therapy (KDIGO, 2020).
- In both Type1Diabetes(T1D) or Type2Diabetes (T2D), lower achieved levels of HbA1c <7% versus 8%–9% reduce risk of overall microvascular complications, including nephropathy and retinopathy, and macrovascular complications (KDIGO, 2020).

Glycaemic control
HbA1c target ranging from <6.5% to <8.0% in patients with diabetes and CKD not treated with dialysis (KDIGO, 2020)

Management of hypertension

- 1st line: ACE inhibitors (Enalapril 5-10mg OD; captopril 12.5mg BD)
- 2nd line: calcium channel blocker (Nifedipine SR 20 mg OD or Amlodipine 5-10mg OD) + diuretic (furosemide 40mg OD or BD)

Antihyperglycemic therapies in patients with T2D and CKD

Glycaemic management for patients with T2D and CKD should include lifestyle therapy, first- line treatment with metformin and a sodium–glucose cotransporter-2 inhibitor (SGLT2i), and additional drug therapy as needed for glycaemic control

- Treat patients with T2D, CKD, and an eGFR ≥30 ml/min per 1.73 m2 with metformin.
- Adjust the dose of metformin to be halved when the eGFR is between 30 to 45 ml/min per 1.73 m2, and for some patients when the eGFR is 45–59 ml/min per 1.73 m2 dose reduction may be considered depending on condition
- Monitor patients for vitamin B12 deficiency when they are treated with metformin for more than 4 years.
- Treating patients with T2D, CKD, and an eGFR ≥30 ml/min per 1.73 m2 with an SGLT2i because they are metabolized by liver or partially excreted by kidneys. Dose reduction or discontinuation especially when GFR is <30mL/min/1.73m2 may be required.
- Treatment can be discontinued if patient has been initiated on dialysis due severe reduction in kidney function
- Sodium-glucose cotransporter-2 (SGLT-2) inhibitors in those with severely increased albuminuria should be considered
- For patients in whom additional glucose-lowering may increase risk for hypoglycaemia (e.g., those treated with insulin or sulfonylureas and currently meeting glycaemic targets), it may be necessary to stop or reduce the dose of an antihyperglycemic drug other than metformin to facilitate addition of an SGLT2i.
- In patients with T2D and CKD who have not achieved individualized glycaemic targets despite use of metformin and SGLT2 treatment, or who are unable to use those medications, use a long-acting glucagon-like peptide – 1 Receptor Agonist (GLP-1 RA). E.g. Dulaglutide 0.75mg and 1.5mg once a week – no dose adjustment, use with GFR of <15mL/min/1.73m2
- To minimize gastrointestinal side effects, start with a low dose of GLP-1 RA, and titrate up slowly.
- The risk of hypoglycemia is generally low with GLP-1 RA when used alone, but risk is increased when GLP-1 RA is used concurrently with other medications such as sulfonylureas or insulin. The doses of sulfonylurea and/or insulin may need to be reduced. Statins can be used to lower cholesterol.

Lifestyle modification

- ◊ Reduction of dietary salt intake to <5 g per day.
- ◊ Maintaining a protein intake of 0.8 g protein/kg (weight)/d for those with diabetes and CKD not treated with dialysis. Patients treated with haemodialysis, and particularly peritoneal dialysis, should consume between 1.0 and 1.2 g protein/kg (weight)/d.
- ◊ Restriction of phosphorus and potassium
- ◊ Increased physical activity; moderate-intensity physical activity for a cumulative duration of at least 150 minutes per week
- ◊ Smoking cessation
- ◊ Reduce excessive weight

MANAGEMENT OF PRURITUS IN CKD

Pruritus in CKD may be caused by uremic toxins, electrolyte imbalances, immune dysregulation of uremia, calcium-phosphate deposition, metabolic abnormalities such as high phosphate levels, abnormal serum calcium, parathyroid and imbalance in opioids which activates itch fibers

Other contributing factors are dry skin (xerosis) due to changes in sweat glands and oil glands. CKD causes dry skin hence skin lacks lubrication. Uremic frost; white, powderly substance left on the skin due to severe uremia.

Some aggravating factors of pruritus are heat, stress, cold, physical activity, showering and dialysis

Clinical features

- ◊ Itching, worse at night
- ◊ Most common affected areas: back, arms and face
- ◊ Restless throughout day and night
- ◊ Scratching

Non-Pharmacological Measures

- Aggressive skin hydration with aqueous cream emollient two to four times daily
- Apply soothing skin lotions i.e. baby oil, creams with lanolin or camphor 2 to 4 times daily.
- Advise patient to apply a moisturizing, high water content gel, lotion or cream to the body right after bathing, while the skin is damp after bathing and other times to relieve itching
- Patient should avoid alcohol –based creams or lotions
- Advise patients to limit food high in phosphorus such as nuts, cola drinks, organ meat (offals, liver)
- ·Getting sunlight or ultraviolet from sun may lessen itching changes the chemicals on skin
- Advise patients to take morning baths to relieve itching and prevent secondary infections.
- Avoid long, hot showers or hot baths as they can make skin dry
- Advise patients to use soaps with natural, pure ingredients without harsh perfumes and chemicals. For sensitive dry, itchy skin; patient can use bath products made with oatmeal
- Educate patient to minimize scratching this can break the skin
- Rule out other causes of itching in patients who are refractory to a reasonable treatmentss

Pharmacological Measures

- Patients should use itch-relieving creams
  - ◊ Topical 0.025% to 0.03% Capsaicin cream/ointment can be applied
  - ◊ 1% Pramoxine hydrochloride lotion two times daily for 4 weeks
  - ◊ Tacrolimus cream 0.1% or 0.03% twice daily for seven days may be used in severe cases
  - ◊ – use with caution
  - ◊ Ergocalciferol
- Systemic therapies: use Gabapentin 100mg alternate days or Pregabalin 25 to 75mg Nocte
- Type B Ultraviolet light phototherapy (UVB) is a good option for treating uremic pruritus inhibits T-helper-1
- Mast cell stabilizers e.g. ketotifen for eight weeks
- Antihistamines such as Piriton when necessary
- Phosphate binders such as Calcium Carbonate to take with meals Opioid antagonists e.g. Naltrexone 50gm OD orally or Naloxone
- Optimize dialysis adequacy, calcium and phosphorus levels, skin hydration and nutrition

Table 5: Analgesia use in Patients with Renal Failure (CKD stages 4 and 5)

WHO Ladder	Medicine	Dose	Comments

Step 1	Paracetamol	Maximum daily dose 4g orally	Use maximum daily dose of 3g in liver impairment
Step 2	Tramadol	50mg-100mg 6-8 hourly (orally)	Only use immediate release preparation. Start at 50mg and titrate upwards. If creatinine clearance is less than 30ml/min give 12 hourly. May be poorly tolerated in stage 5 CKD
	Co Codamol 8/500 or 30/500	4 tablets in 24 hours	Caution: Accumulation of metabolites may cause narcosis and respiratory depression
	Codeine Phosphate 30mg	120mg in 24hrs (orally)	Use with caution
Step 3	Morphine	Oral Titrate with low starting dose 2.5mg-5mg 6-8 hourly	Caution: Accumulation of parent drug and metabolites reported to cause profound respiratory depression and narcosis
Parenteral			
Titrate with low starting dose 2.5mg as required			
	Fentanyl	Transdermal patch for stable pain only	10% parent drug excreted renally so may get some accumulation in renal failure
	Alfentanil	1/10th dose of morphine.	Do not use rescue doses of alfentanil to titrate the background dose because of its short half life

NOTE

NSAIDS - should be avoided, If prescribed: use lowest effective dose, monitor renal function

Renal Replacement Therapy if Conservative Management Fails

CHAPTER 6: PREVENTION OF KIDNEY DISEASES

The goal of prevention is to screen and identify patients at risk, monitor high risk patients, implement prevention strategies when appropriate. Prevention is based on
Acute Kidney diseases per STOP AKI strategy

1. Prevention of CKD progression
2. Prevention of CKD
3. STOP AKI strategy

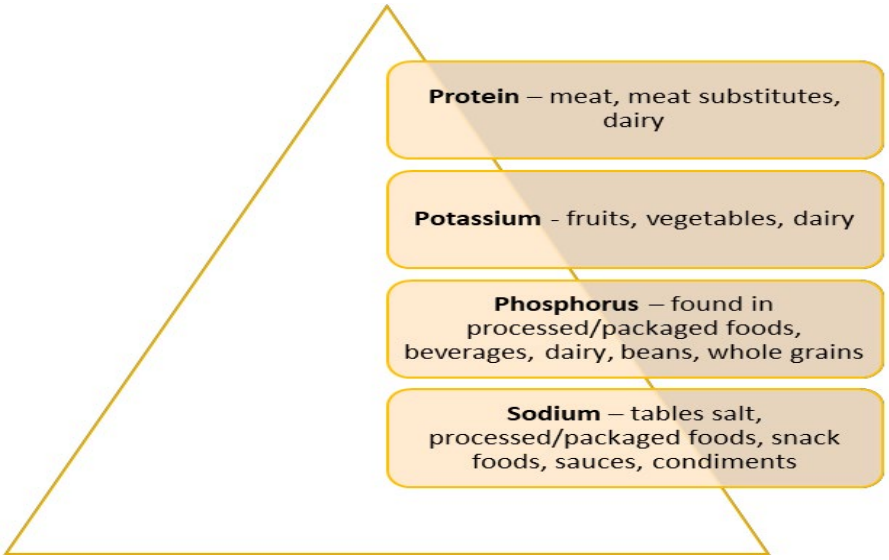
S – Sepsis and hypoperfusion Manage promptly acute illness such as; o Infections, malaria, dehydration, haemorrhage, heart failure and liver failure Maintain rehydration with IV fluids and oxygenation
T – Toxicity Minimise further kidney insults Drugs such as ACEi, NSAIDs, gentamycin, tenofovir, Amphotericin, radio contrast, herbal remedies and traditional medicine should be avoided
O – Obstruction Retention, mass, stone or any extrinsic compression (prostate/bladder/ureter) should be attended

Prevention of CKD Progression

Non-pharmacological Approaches

1. Diet restriction

The foods or food groups in which these nutrients are high include:



Proteins

Protein intake is restricted to 0.6–0.8 g/kg ideal body weight (IBW) for pre-dialysis patients (Brown & Compher, 2010). For hemodialysis patients, protein requirement is 1.2 g/kg IBW and
- 1.3 g/kg ideal body weight for peritoneal dialysis patients.

In general, 1 small chicken wing or drumstick is 30g meat and may contain 7 g of protein; 1 small chicken thigh or ½ cup tuna or 1 pilchard is 60g meat may contain 14g of protein; and 1 small chicken breast is 90g meat and may contain 21g of protein.

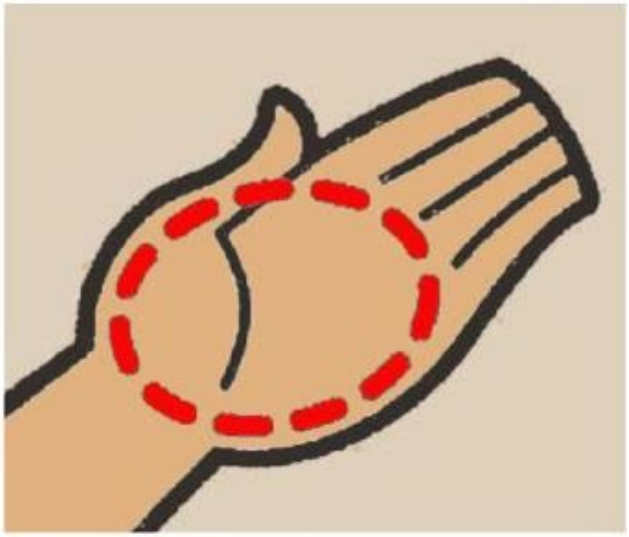
A patient can take

- 1 extra large egg or 2 egg whites per day
- ½ cup of low fat milk

A patient should avoid

- Nuts
- Offal, sausage, canned meat
- Pizza
- Milk and dairy products
- Chocolate and cheese,
- Meat tenderizer
- Salted dried fish. (Unpacked dried fish from the local market can be washed in warm water to remove any salts and/or oils added by sellers.)

A practical tool for controlling protein portion sizes can be the approximation of volumes from hand measurements, e.g., the palm equivalent to 90 g (3 matchboxes) of meat, chicken without skin or fish. The preferred cooking methods include boiling, roasting, grilling or stewing, e.g., boiled (or roasted) roasted meat, poultry, fish, egg.



Size of palm:
Useful for estimating:
Meat, fish, chicken

Phosphorus

The required amount of phosphorus in CKD patient is 800 – 1000 mg/day. Phosphorus is found in both animal and plant products. However, the phosphorus in animal sources may be absorbed better than that from plant products because the phytic acid or phytate found in plant-based foods can block some of the phosphorus absorption. In addition, commercial products such as beverages and packaged foods may contain hidden phosphorus from additives or enhancers containing phosphorus. Therefore, the patient may avoid all packed foods eg., canned fish,grocery sausages, canned beans. For beverages/fluids, a patient may take 1 cup 100% apple juice or pineapple juice per day or lemon water. Avoid all alcohol drinks.

Dried beans/peas are also high in both phosphate and potassium. Therefore, always soak the beans/peas for at least 8 hours and discard the water before cooking. Note, the protein restriction described above (under Protein) also helps to reduce phosphate intake.

Potassium

Potassium is mostly found in plant-based foods like fruits and vegetables (see table 3). Although high potassium levels are dangerous, a low potassium level can also be dangerous for the heart. If the patient has low potassium levels at any time, they may be requested to eat a certain amount of high potassium foods that are normally restricted. When the patient is hyperkalemic, potassium intake can be restricted 2000-4000mg/day (usually not needed until GFR <30 ml/m) in the following ways;

- Try to choose low/medium potassium fruit and vegetables such as apples, pineapples, cucumber and lemon (lemon water). Others fruits with high potassium like bananas/plantains, guava, avocado, mango, oranges/orange juice, papaya, and tamarind (Bwemba) may be avoided.
- Taking 4-5 servings of lower potassium fruit or vegetables per day. The portions should be small as a large

portion of a low potassium food can make it a high potassium food.

- A patient can take 1 ½ cup of unsalted popcorn.
- Because potassium can dissolve into water, the potassium content of vegetables can be decreased by carrying out the following steps:
  - ◊ Wash and peal vegetables and chop them finely
  - ◊ Soak vegetables in water for 2 hours
  - ◊ Discard the water that the vegetables were soaked in
  - ◊ Discard the water that the vegetables were cooked in.
- Always peel, chop and soak potatoes for at least 8 hours before cooking. Only one serving of potato per day as these are very high in potassium.
- Dried peas and vegetables are high in potassium. Always soak the beans/peas for at least 8 hours before cooking.
- Avoid chips bought from take away shops and restaurants. If you have homemade chips or roast potatoes then soak them first and parboil before cooking.
- Avoid baking, frying or steaming raw vegetables or potatoes as this doesn't reduce the potassium content.
- Avoid mushrooms, tomato, tomato sauce, kamba puffs, okra (Therere)
- Avoid nut bread, salted crackers and whole wheat bread, cereals andpasta
-

Size of the patient's fist: useful for estimating rice and nsima

Size of the patient's fist: useful for estimating rice and nsima

Table 6: Examples of Potassium Foods

Low potassium foods	Mg (K+)	Medium potassium foods	Mg	High potassium foods	Mg
Fruits Lemon (1) 2 rings pineapple (½ cup) Tangerine (1) Cucumber ½ Cup (1 slice) 1 medium Apple	Mg 85 115 125 110 158			 Apricots (3)	 300

Vegetables (cooked) ½ Cup bell peppers Cabbage ½ cup Cauliflower 1/2cup serving Garlic 1 clove Onions 1/2cup Beans green ½ Cup Bean sprouts, raw ½ C up Lettuce, raw 1Cup Asparagus (5spears) Olive oil 1tablespoon Corn cob ½ Cup	 88 60 88 12 116 100 115 130 140 <1mg 130	Carrots ½ cup Beets ½ cup Egg plants ½ cup Mushroom ½ cup Okra 8 pods Lentils ½ cup 1 raw Green pepper Mixed vegetables ½C Peanut butter 2 teaspoon Broccoli ½ cup	170 180 150 170 250 170 250 190 200 210	Beans ½ cup Greens, turnips ¾ cup Pumpkin ½ cup Spinach ½ cup Tomato, raw (1) Nuts ¼ cup Yams ½ cup Potatoes ½ cup Potato chips 1 oz	350 280 295 295 350 335 300 325 290		
Bread and Cereals Rice 1 C Doughnut (1) White bread 1 slice Whole grain bread 1 slice Cereals 1 cup Crackers: snack-15 Pasta ½ C	 50 40 30 70 <140 50 50	Raisin Bran 1 cup	240	All Bran ½ cup	260		
DRINKS (LOW POTASSIUM) Apple juice ½ Cup Cranberry juice ½ Cup Grape juice ½ Cup Lemonade 1Cup Tea 1Cup Coffee 1Cup Wine 4oz (120 Ml)	 125 22 145 15 25 85 110	MODERATE POTASSIUM Apricot nectar ½ C Orange juice ½ C Pineapple juice Ice cream 1 cup Custard ½ cup Pudding ½ cup	 150 250 185 230 175 170	HIGH POTASSIUM Milk 1 cup Cocoa 1 cup ½ Cup Tomato or vegetable juice ½Cu Prune juice Yoghurt 1 cup Buttermilk 1 cup	 350 365 270 300 400 345		

Sodium

The required amount of sodium is 2 – 2.3 g/day (1 teaspoon of table salt). A diet high in salt (sodium) can lead to fluid retention and high blood pressure. Salty foods can also make you thirsty and this can be a problem if you need to limit your fluid intake. Salt/sodium can be found naturally in many foods; therefore, it is impossible to avoid it completely but the following can help you to reduce your intake:

- When cooking, use very little or none at all. You can use herbs, curry powders, chilli powders, garlic, ginger and lemon juice to help add flavor to food.
- Don't add salt to your meals at the table
- Avoid hot spices
- Avoid flavored salts such as garlic salt or celery salt.
- Reduce salty foods in your diet, e.g., processed meats (ham, polon, bacon, sausage, noodles, canned fish), salty snacks (chips, salty biscuits or crackers) other processed foods, and spreads such as Bovril, marmite, cheese spread.
- Avoid salt substitutes such as low sodium salts as they are often high in potassium.
- Convenience foods, ready prepared meals and take away foods are generally high in sodium, so should be limited/avoided.

Fats and oils

1 teaspoon of margarine/day
1 teaspoon of oil in cooking/day
1 teaspoon of peanut butter as condiment/day

Table 7: Example of days meal Plan

Meal Meal Item (Examples)

Breakfast 2 slices of bread/ 250 mls of porridge 2 mandazi
1 teaspoon of margarine

½ cup (125 mL) fresh milk (may be diluted with water to one full cup) 2 teaspoons sugar
Morning Snack 1 medium size apple

Cucumbers 1 tangerine
A slice of pineapple

A wedge of watermelon

Lunch 1 lump of nsima (size of a fist) 2 medium cooked fresh fish 1/2 cup cooked cabbage
1 medium apple

Afternoon Snack 1 cup (250mL) pineapple/apple juice

Dinner 1 lump of nsima (size of a fist) 1 drum stick
1/2 cup leached vegetables

1/2 cup soaked & cooked beans 1 medium apple

Fluid

When kidneys are not working properly, not enough urine is made by the kidneys. When the amount of urine passed is less than the amount of fluid taken in, it means fluid is retained in the body. This is dangerous as it causes overloading, swelling of the ankles and around the eyes, high blood pressure, shortness of breath and may result in heart failure.

If you gain weight quickly, it usually means that fluid is building up in your body. If there is no water retention, then you do not need to restrict fluid intake.

It is important to check the amount of fluid taken in and compare it to the amount of urine passed. Water should always be taken in sips, no gulping.

A minimum of 500 ml (2 cups) of water/fluids should be taken daily to make up for fluids lost from the body through breathing and sweating.

METHOD	EXAMPLE
Measure the total amount of urine passed on day 1	Urine passed on day 1 = 250 ml (1 cup)
On day 2 add this 500 ml to give a total amount of fluids that can be taken per day	Fluid allowed on day 2: 250 ml + 500 ml = 750 ml (3 cups)

- Patients should be advised to perform exercises regularly. Patients should undertake physical activity at least 30 minutes per week.
- Patients should be advised to reduce excessive weight.
- Diet: low in salt (less than a teaspoon in a day), red and processed meat and saturated fat, and high in fruit and vegetables.
- Prevent high protein intake of >1.3 g/kg/day and 0.8g/kg/day in patient with GFR
- <30mL/min (meat equivalent to the size of the 3 matchboxes, dry legumes equivalent to half a cup, eggs 1-2)1
- Advise patient to quit smoking.
- Recommend to stop alcohol intake.
- Control high blood pressure. KDIGO recommends keeping blood pressure <140/90 in nonproteinuric and <130/80 in proteinuric
- Patients with diabetes should stay in their target blood sugar range, FBS 70mg/dl to 130mg/dl, achieve optimal glycaemic control targeting HBA1c <7%
- Patient should be advised to drink plenty of water at least (weight x 30mls) in 24 hours.
- In terms of number of cups to drink in 24 hours = body weight kg divided by 8(250mls cup)

Prevention CKD

Get tested for CKD regularly (yearly) if you are at risk and treat early (Center for Disease Control and Prevention (CDC), 2020). Screening if family history of kidney diseases. Some people inherit kidney disease from their parents

Avoid NSAIDs like ibuprofen, Indocid, diclofenac and naproxen and take medicines as prescribed.

Avoid herbal medicine including local herbs

Control of hypertension is the single most important intervention to reduce both albuminuria/proteinuria and subsequent development of CKD in patients with and without diabetes alike (Bello et al., 2005). It is therefore imperative to detect prehypertensive states, aim at lower blood pressure levels in the general population, and be even more aggressive with lowering blood pressure in patients with hypertension and underlying CKD.
The control of glycemia has been shown to be a major factor in the prevention and progression of diabetic nephropathy (Center for Disease Control and Prenvention, 2020).
Minimize the risk of contrast induced renal injury

Encourage patients to get vaccinated against diseases such as hepatitis, SARs COV2.

Prompt management of AKI.

Control of albuminuria/proteinuria is also an important factor in slowing the progression of diabetic and nondiabetic CKD.
Establishment of functioning renal clinics in District and Central Hospitals

CHAPTER 7: RENAL REPLACEMENT THERAPY

It is the aim of the government of Malawi to provide all Malawian citizens equitable access to renal dialysis. Dialysis is a method of removing waste products from the body for patients with kidney failure. The settings where dialysis may be undertaken are: Hospitals, satellite units and homes.

The renal replacement therapy is currently being provided in tertially public hospitals and some private facilities in form of haemodialysis and peritoneal dialysis. There are future plans for kidney transplant. The services can be accessed for free and paying services.

However, due to the lack of resources, it has to be accepted that there is a need to set boundaries for medical treatment, including renal dialysis.

Dialysis guidelines have been formulated to optimize the use of scarce resources, cost- effectiveness, promote public/private partnership and improve service provision to patients.

Guiding principles

All treatment options for chronic dialysis should be discussed with the patient and the family members. They should be allowed to choose the technique that is optimal for the patient with due consideration of medical, social and geographic factors. Treatment that is offered should be cost- effective both to the healthcare system and the patient. In order to make informed choice the potential impact on the patient's life and that of the families should be explained.

Physical and psychological symptoms related to chronic renal dialysis should be treated appropriately and monitored. Public Private Partnerships should be encouraged where possible as a model for service delivery in chronic renal dialysis. The service providers must take reasonable measures, within its available resources, to achieve the progressive realization of the services to be offered.

Patients with diabetes and patients with acceptable co-morbid conditions may be considered for long-term renal dialysis although research shows that they do not respond well in the long term.

The current method of providing kidney therapy has been faced by challenges of resource and by patients who have started treatment in private sector and would want to proceed receiving care at public tertially hospital.

Inclusion and exclusion criteria for acceptance into the chronic dialysis programme

In order to make efficient use of healthcare resources and to ensure that patients receive optimal benefit of chronic dialysis exclusion rather than inclusion criteria should be applied for the selection of a suitable patient.

Before it is decided that dialysis is a suitable option for an individual with chronic kidney disease there should be done a full assessment of the patient's healthcare needs such as economic, social, school and work circumstances. The consequences of long- term dialysis are significant on the patient and their families.

There should be the following:

1. Renal board
2. Indication for dialysis
3. Categorization strategy for patients with end stage renal disease (category 1-3)
4. Renal replacement therapy

Renal Board

- There shall be a renal board in public dialysis centres who shall oversee and make decisions on categorising of patients who need to access renal replacement therapy and termination of the treatment.
-
- The renal board should make a decision of categorizing patients and communicate decision within 6 weeks.
-
- After 6 weeks' further treatment will be determined according to categorization by renal board.

    ◊ Patient in category A will continue with dialysis according to category A instruction
    ◊
    ◊ Patients in Category B can either continue with dialysis or go on waiting list for transplantation according to the category instructions
    ◊
    ◊ Patients in category C the treatment will be terminated permanatly.

- Patients in category C who opt for renal replacement therapy privately should remain the responsibility of the private service provider.

- The board shall report to the Hospital director.
-
- The board will be responsible for recommending patients for transplantation to the transplantation committee
-
- The Ministry shall develop terms of reference (TORs) which will include;

    ◊ Chair for the board
    ◊
    ◊ Membership to the board will include;

        » Hospital director
        »
        » Head of Department
        »
        » Head of internal medicine
        »
        » Nephrologist/ Renal Physician
        » Physician attending to the patient

» Head of Nursing department
»
» Nurse incharge of Dialysis
»
»
» PyschoSocial worker
»
» Dietician
»
» Hospital Administrator

◊ Quorum should be 50 % +1

Indication for Dialysis

Those with AKI are recommended for renal replacement therapy unless they have multi -organ failure

Dialysis is recommended when the eGFR is less than 15 ml/min/1.73m2 and the patient has one or more of the following:

- Symptoms or signs of uremia
- Diuretic resistant fluid overload
- Poorly controlled blood pressure
- Evidence of malnutrition
- Refractory metabolic acidosis
- Refractory hyperkalemia
- For acute dialysis to consider dialyzable toxins and drugs

Categorising of Patients with End Stage Renal Failure

CATEGORY A

These are patients who meet the criterion for renal replacement programme as below;

Age of less than 50 years
BMI less than 30kg/m2
Gainfully employed
HIV-negative
HBSAG- negative
Malawian Citizen
Avoidability ® Able to meet transport costs ® Able to be away from work to attend sessions ® Able to follow changing treatment schedules

If patient meets the category the renal board should make sure that the patient starts dialysis either at the public hospital or is sent to private sector to be supported by government of Malawi or patient's health insurance depending on availability of resources

CATEGORY B

In this category, the patient is accepted on the renal replacement therapy programme pending the availabity of space in the hospital according to the following:

MEDICAL

- Age range of 51-60 years
- BMI- 30- 35Kg/m2
- Hypertension with target severe organ damage
- HBSAG/HCV positive and no cirrhosis
- Diabetes Mellitus
- HIV positive provided CD4 count more than 200 and undetectable viral load. If on ARVs demonstrate good adherence and clinical response in 6 months
- Late presentation requiring urgent dialysis
- Comorbid diseases e.g stable Ischaemic Heart Disease (IHD)
- Previous Kidney Transplant
- SOCIAL
- Poor home circumstance; lack of storage space, running water, sanitation and electricity
- Not gainfully employed
- Poor social network/support
- Fails to enhance others
- No proximity to dialysis unit

Whilst on the waiting list the patient should find his/ her own means to access the renal replacement therapy.

The Renal board should ensure that first come first serve on the waiting list.

CATEGORY C

» Patients will be given temporaly dialysis for 4-6weeks whilst giving time for further investigations and decision by renal board
» Transplantation contraindicated or carries unacceptable risks
» HIV infection other than as described in category 2
» Age more than 65 years
» Active substance abuse or dependency
» Morbid Obesity (BMI more than 35kgs/m2
» Cirrhosis
» Diabetes Mellitus and above 60 years
» Active uncontrollable malignancy with short life expectancy
» Non-Malawian Citizen
» Advance irreversible progressive vital organs disease e.g CVA, lung disese, Liver disease, unresponsive infection
» Psychological exclusion criteria
» Mental illness resulting in diminished capacity to take responsibility for his/her action
» Habitual no adherence with any medical management

RENAL REPLACEMENT THERAPY (RRT)

Renal replacement therapy replaces non- endocrine functions of the kidney. It is the essential requirement for relieving patients suffering due to ESRD and works as a bridge to renal ransplantation or when transplantation is not available. Similary, it can be used to support patients with AKI for a temporaly period.

RRT are of various types:
1. Haemodialysis
2. Peritoneal
3. Kidney transplantation

Haemodialysis

Haemodialysis is a medical procedure to remove fluid, waste produet from the blood and to correct acid-base and electrolytes imbalance using a machine and a special filter called an artificial kidney(dialyser). The procedure is mostly done 3 times a week on a patient and runs for 4 to 5 hours.

The machine is connected to the patient through a vascular access (VA) which lets large amounts of blood flow continuously during haemodialysis. Their two types of VA which are Arteriovenous Fistula and dialaysis catheters. Dialysis catheters should be inserted in large veins such as Femoral vein, Internal jugular vein and Subclavian Vein.

Dialysis Catheters also known Central Venous Catheters (CVCs) are of two types; temporary and Permanent Catheters

Temporary Catheter

- These catheters are called Uncuffed catheters. They are usually used in emergency conditions or when dialysis is needed only for a few days.

- Double lumened uncuffed non tunnelled soft catheters inserted in the Internal Jugular vein should be the temporary access of choice either when;

  ◊ Permanent catheter should be done as soon as the patient is stable and can tolerate the procedure.
  ◊ A permanent access has been created and is expected to mature within 90 days.
  ◊ A period of waiting before the permanent access can be created is anticipated.
  ◊ Partial or complete recovery is expected.

- The sub-clavian vein should not be cannulated as a temporary access unless the internal jugular is unusable and no permanent access is possible ipsilaterally. Even a single cannulation is associated with a 35% risk of stenosis.
- The femoral vein on the left side may be used as a temporary vascular access with rigid single lumened cannulae in an emergency situation only. Cannulae in the femoral vein should not be retained for longer than 5 – 7 days and should never be used in the outpatient setting. Right sided femoral vein cannulation should be avoided if the patient is to undergo a future renal transplantation.
- Permanent Vascular Access

1. Permanent dialysis catheter

These catheters are used incases where dialysis needs to extend from the previously stipulated time or when patients are in a waiting period for AV fistula maturation. Permanent catheters are cuffed and tunnelled.

2. AVF

An arteriovenous fistula (AVF) is the vascular access of choice in haemodialysis patients and should be fashioned timeously (CKD stage 4, preferably when required.

Note 1:The order of placement of AVF is at the wrist and then the elbow starting with the non-dominant arm. The advantages of fistulae include ;

1. excellent patency once established,
2. improved flow over time
3. lower incidence of stenosis, infection and vascular steal phenomenon.

Note 2: If it is not possible to establish a fistula, access may be established using an arteriovenous graft of synthetic material (preferably PTFE) upon vascular surgeon assessment

Note 3: Cuffed tunnelled central venous catheters s not be the first choise as a permanent vascular access.

Note 4: The initial cannulation of a native AVF must be performed by an experienced person.

Note 5: Patients with AVF must adopt good personal hygiene habits; clean technique should be used before cannulation

Note 6: The placement of subclavian vein catheters for acute dialysis should be avoided as should venepuncture of antecubital fossa veins, in patients who are potential candidates for haemodialysis.

Note 7: The AVF should be monitored by regular physical examination, and surveillance for stenosis can be done by monitoring static dialysis venous pressure, by duplex ultrasound and by assessing for recirculation.

Note 8: Use tourniquette during initial cannulation of an AVF

The following should be followed in deciding when to use an AV fistula;

6 weeks from the time of creation and when matured,

A cannulation length of at least 3 to 6 cm from the anastomosis,

A vein of at least 6 mm in diameter with clearly distinguishable margins,

Flow of at least 350 ml/min and a depth of not more than 6 mm from the skin. Numerous collateral veins should not be visible and there should be no evidence of venous hypertension

Cannulation for VA

- Initial cannulation should be with 17G needles equipped with a "back eye". Flows of up to 250 ml/min can be obtained with a 17G needle. Subsequent cannulation should be with a 16G needle to obtain flows of 300 ml/min and with a 15G needle to obtain flows of > 300 ml/min.
- A railroading technique rather than a button hole technique should be followed for cannulation.
  - ◊ Railroading – At each dialysis session, puncture of the fistula should be done 1 to 2 mm away from the previous point and a return to the original site should occur after 6
  - ◊ – 7 sessions.
  - ◊ Buttonhole – Every puncture is done through an identical point. This eventually leads to decreased pain sensation at the site but also to weakening of the vein wall and aneurismal dilatation. However, not a recommended method due to increased risks of infection and requires use of blunt needles

- Removal of needles should follow aseptic technique.
  - ◊ Firm digital pressure over a sterile gauze should be given to the site of needle insertion for 10 minutes after removal of the needle followed by application of a sterile adhesive dressing.

- Tight tourniquets should not be applied to a fistula limb.
- Avoid inserting a drip, drawing blood from a fistula site and measuring blood pressures.

Monitoring of permanent VA

All care givers should learn the methods of examining a vascular access; this will include the following steps;

Look

- ◊ Vascular access scar site: Radiocephalic ArterioVenous Fistula (AVF), Snuff box AVF, Brachiocephalic AVF, Transposed Basilic vein AVF, Transposed forearm cephalic vein AVF and Transposed forearm basilica vein AVF.
- ◊ Signs of infection and inflammation
- ◊ Hematoma
- ◊ Signs of ischemia (Steal syndrome); distal limb ischemia following dialysis access placement, the signs will include blue and cold fingers, hand pain at rest or during exercise, Paresthesia, ulcers and dry gangrene.
- ◊ Aneurysm:
  - » Stable aneurysm has intact overlying skin and no features of outflow obstruction.
  - » Unstable aneurysm has a thin, shiny skin, depigmentation, prolonged leaking, ulceration and

rapid enlargement.

» According to KDiGO guidelines an aneurysm 1.5 to 2 times the native vein should be referred for surgical evaluation.

◊ Central vein stenosis evidenced by a massively swollen upper arm due to occlusion of subclavian vein.

Perform an arm elevation test outflow for patency of the AVF.(see diagram I)

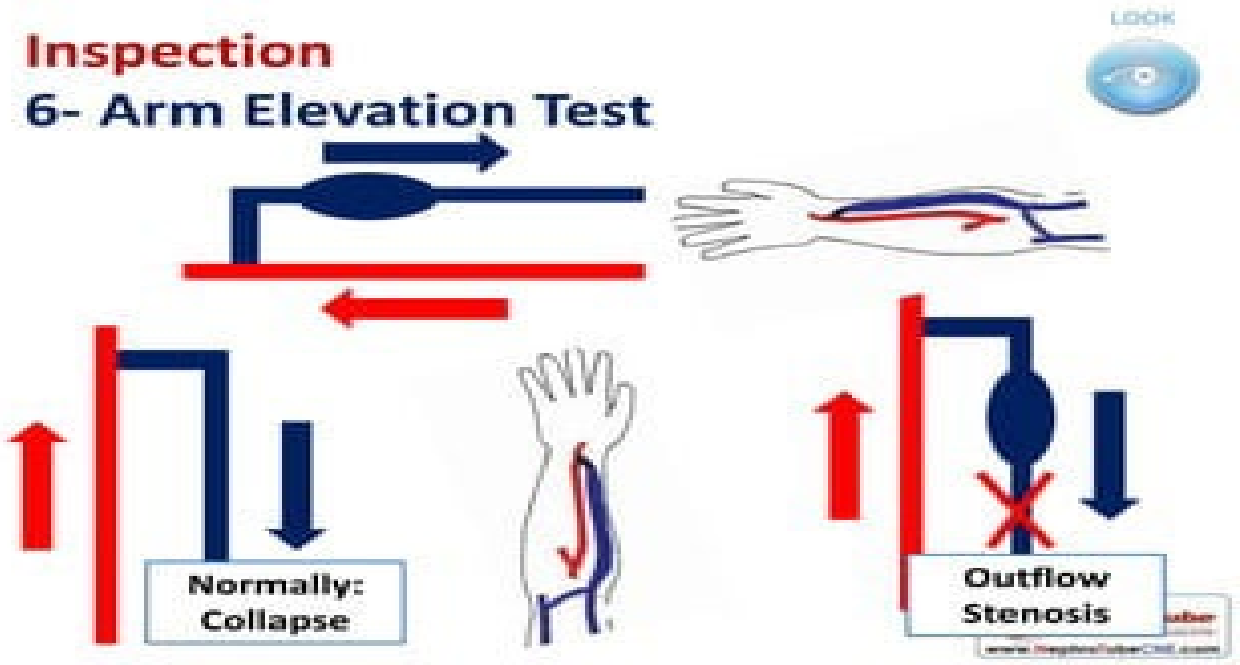


Diagram I: demonstrating an arm elevation test.

Feel to detect inflow and outflow problems

AVF pulse character: This should be soft and compressible, if there is a hyper- pulsatile (water hammer) suspect outflow stenosis and if the pulse is hypo- pulsatile, feeble and flat suspect inflow stenosis.
AVF thrill; examine for thrills from the anastomosis all the way to the chest wall.
Normally there is a continuous nature to the thrill (systolic and diastolic soft background) except at the arterial anastomosis where the thrill normally is discontinuous). Presence of a strong localized thrill will indicate outflow stenosis while a weak localized thrill will indicate inflow stenosis.

Listen

Bruit: Ascultate from the anastomosis all the way to the chest wall, normally there is a continuous murmur (low pitched systolic and diastolic background sound).
Abnormally discontinued murmurs and loud is due to outflow obstruction and low is inflow obstruction.

Treatment of Access Complications

Infection
Temporary catheter in the femoral vein should always be removed if suspected to be infected. Temporary catheters in the internal jugular vein may be retained for a 24 to 48 hour period while systemic antibiotics are administered, but should be removed if fever persists for longer than that and subsequently replaced at a fresh site.

Cuffed tunneled catheters may be retained for 72 hours or longer while antibiotic therapy according to culture reports is administered. Systemic antibiotics should be accompanied by local antibiotic lock solutions, the concentration of which can be several times higher than that of the Minimum Inhibitory Concentration (MIC) reported for blood cultures.

Example of antibiotic locks: antibiotic – heparin solution -antibiotic mixed with 4 ml of Heparin containing 1000 units/ml and up to 1.5 ml of the solution should be injected into each limb of the cannula. The final solution contains 800 units/ml of Heparin and antibiotic, which are physic-chemically compatible. Stronger concentrations should not be used and care should be taken not to exceed the volume of the cannula to avoid systemic toxicity

PRIMING, CONNECTING AND DISCONNECTING DIALYSER

The connection, starting stopping and disconnection of a patient from the extracorporeal circuit is an integral part of the treatment and one which can give rise to complications both early and delayed if improperly handled. The starting and stopping of dialysis also includes assessment of the patient as well as noting developments during dialysis and in the interdialytic period. Because this is the period when both patients and unit staff are under pressure to speed up the entire procedure strict adherence to a protocol and perhaps the use of checklists is necessary to prevent complications. The following guidelines provide a protocol for the procedure, designed to optimize treatment.

Rinsing & priming of dialyzer:

- Thorough rinsing of dialyzer is important since it will reduce the incidence or severit y of anaphylactic reactions by removal of leachable allergens.
- For new dialyzer rinse blood compartment with 1 litre of normal saline .This is done to eliminate all the air and residual sterilants from the dialyzer, blood lines and for priming of the circuit. Dialysate compartment of dialyzer is rinsed with dialysate for at least 5 minutes before initiating dialysis.

Check all alarms

1. Blood circuit

a) Inflow (pre- pump) pressure monitor

» Inflow pressure is 80 to -200 mm Hg. If there is poor blood flow from the vascular access the alarm will beep and the blood pump will stop. Once the pump stops the suction is relieved and the alarm is deactivated.

» Important causes for excessive suction could be either a thrombus or fibrin plug at catheter tip (venous catheter) or improperly placed arterial needle or clotting of arterial needle (AV fistula), drop in patient's BP, kinking of arterial line, use of a too small needle.

b) Outflow (venous) pressure monitor

» It is usually +50 to +250 mm Hg. Causes of high pressures could be clotting of the venous blood line filter or venous line/needle, high blood flow rates when using a small venous needle, kinked venous line, stenosis at the venous limb, improperly placed venous needle.

c) Air detector:

Important alarm to prevent air embolism which can be fatal. Common sites for air entry include the region

around the arterial needle, via leaky tubing connections, via broken blood tubings, via saline infusion tubing.

Blood line kinking & hemolysis alarm

Dialysis solution or Dialysing fluid

This is a solution of purified water and electrolytes in concentration similar to blood

Composition of Dialysis Fluid

1. Purified water
2. Bicarbonate solution
3. Acid concentrate

Purpose of Dialysis Fluid

- It carries away waste products and excess fluid removed from the patient's blood during dialysis treatment
- Prevents excess removal of essential electrolytes and avoids depletion during the dialysis process
- Monitors conductivity, temperature and blood leak
  - » **Conductivity:** Most common causes of reduced conductivity are empty concentrate container or defect in the proportioning pump.
  - » **Temperature:** Automatically limited between 35 & 39 degrees Celsius for most machines in the dialysis mode. A common cause of low temperature alarms could be either a loose heater cable, a tripping of the safety switch or a faulty sensor in some machines
  - » **Blood leak:** A blood leak alarm should be confirmed by testing the effluent dialysate with a test strip used for detecting hemoglobin in the urine. If leak is confirmed, the dialysate compartment pressure should be set to -50 mm Hg or lower to minimize entry of bacteria from the dialysis solution into the blood side of the extracorporeal circuit. The blood should be returned and dialysis should be discontinued. Use new dialyzer to restart dialysis.

Patient assessment pre-dialysis session

- Record weight of patient
- Measure Blood Pressure in lying and standing position
- Assess patient for any new symptoms and examine patient
- Assess dry weight of patient and Plan target UF
- Patient interview i.e. ask about appetite, sleeping pattern, medications taken on day of dialysis, how they have been after the last treatment and occurence of any unusual bleeding

PROCEDURE TO BE TAKEN WHEN HANDLING DIALYSIS CATHETERS

1. Aspirate residual heparin or clot from each catheter lumen
2. Check patency of catheter lumina by irrigating with normal saline (20 – 40mls)
3. During catheter connect and disconnect procedures, both dialysis staff and patient should wear surgical masks. Face shield should not be used without surgical mask.
4. The lumen and catheter tips should never remain open to air. A cap or syringe should always be placed on or in the catheter lumen while maintaining a clean field under the catheter connectors.

5. Caps should be soaked in povidone-iodine and kept wrapped in gauze soaked in povidone iodine for the entire length of the dialysis. Alternatively, the caps can be sterilised with ethylene oxide autoclaving during the dialysis and can be reused after the dialysis is completed.
6. Catheter lumens must be kept sterile. Inter dialytic infusions through the catheter are forbidden.
7. Always inspect the exit site for any evidence of infection (redness or purulent discharge)
8. If any evidence of exit site infection is seen a swab culture should be taken and sent to the laboratory
9. The exit site should be cleaned with betadine and then dried before it is dressed.
10. If there are infection appropriate systemic antibiotics either oral or parenteral can be started.
11. Local antiseptic ointment such as Mupirocin can be applied to the exit site
12. Exit site should never be immersed in bath water. Showering is best avoided but if the patient showers it should be done prior to coming for dialysis where a new dressing and antibacterial ointment can be promptly applied.
13. Encourage use of water proof plaster(Tegaderm)
14. Use any recommended cleaning solution for catheters such as chlorhexidine 0.5% and alcohol 70% solutions.

Arteriovenous fistula:

1. Check the fistula for patency and function after tying tourniquet
2. Both needles are placed in the vein downstream to the anastomosis
3. Arterial needle is placed distally as compared to the venous needle
4. If the patient has a poorly distended venous limb, briefly apply a tourniquet to define the location
5. A 16 or 15-gauge needle should be used in adults
6. Prepare the needle insertion site with povidone iodine for a full 10 minutes.
7. Arterial needle is inserted first 3 cm from the anastomosis site. The needle is inserted bevel up at25 degree angle for AVF and a 45-degree angle for AVG pointing either upstream or downstream
8. The venous needle is inserted at same angle pointing downstream (usually towards the heart)
9. The insertion point of the venous needle should be at least 3-5 cm downstream to the arterial needle to minimize recirculation

Arteriovenous graft:

1. Guidelines for placing the needles are similar to that of AV fistula

Initial heparin administration:

If heparin is used, heparin loading dose (diluted with normal saline) is administered into the venous port

After 3 minutes of administration of heparin the blood flow is started (Some centers administer the heparin into the arterial line leading to the dialyzer and start the blood flow immediately)

Initiating dialysis:

Blood flow rate is initially set at 100 ml/min until the entire blood circuit fills with blood depending on machine specification.
The priming fluid in the dialyzer should be disposed off to the drain
Ensure proper blood levels in the venous drip chamber
Promptly increase blood flow rate to close to 250-350 ml/min
Record the pressure levels at inflow and outflow monitor

Set the pressure limits slightly above and below (10-20 mm Hg) the operating pressure to ensure that the blood pump will stop in case of any change of operating pressure beyond the limits set
Dialysis solution flow is initiated

Monitoring of patient

- The patient's BP should be monitored and recorded as often as necessary. In an unstable patient the BP should be checked every 15 minutes. In a stable patient BP is checked every 30-60 minutes. Check that needles, lines and connections are securely taped
- Check the lines and connections during EACH AND EVERY patient and machine safety check Keep access uncovered at all times
- In diabetic patients attempts should be made to measure the capillary blood glucose levels to detect any episode of hypoglycemia.

Termination of dialysis:

1. Blood in the extracorporeal circuit is returned using saline
2. If saline is used patient receives 100-200 ml of this fluid during the rinse back procedure

Closure of vascular access:

1. AV fistula

a. Remove the needles from the AV fistula and apply gauze.
b. Tie the tourniquets at the sites of puncture over the gauze pieces
c. Patient advised to loosen the tourniquet straps after 4 to 6 hours and remove the tourniquets if there is no oozing from the puncture sites.

2. Venous catheters

a. After each dialysis session, the dead space of each lumen is filled with heparin through the injection ports using 1000-5000 units/ml. Do not use higher concentration of heparin than suggested since it may result in significant systemic anticoagulation.
b. The dead space of each catheter lumen varies among different manufacturers and also depends on the length of the catheter. The required volume of heparin is usually labelled on the catheter hub. It should be recorded on the patient's chart. Do not inject a volume of heparin solution than necessary as it may be hazardous in patients who are at risk for bleeding.
c. After each dialysis, catheter hubs or blood line connectors should be soaked in povidone- iodine for 3-5 minutes, then dried prior to separation.
d. The catheter should be covered with a sterile dry dressing. Non breathable or non porous transparent film dressings should be avoided since they pose a greater threat of exit site colonization than dry dressings.

Post dialysis monitoring:

a. Measure blood pressure
b. Record the UF done
c. Measure post dialysis weight
d. Ensure that dressings are clean and dry before allowing the patient to leave the treatment room
e. Ensure that dressins is not applied too toghtly

COAGULATION IN HAEMODIALYSIS

Clotting in the extracorporeal circuit is a major challenge in carrying out haemodialysis. The hemodialysis circuit represents a large extracorporeal surface area and the simple passage of blood through the circuit could potentially lead to the deposition and activation of plasma coagulation proteins thus initiating clotting. Therefore, dialysis and CKD patients are at increased risk for venous thromboembolism and access thrombosis.

- Risk factors for clotting in extracorporeal circuit:
- Polycythemia/high haematocrit
- Low blood flow rate
- High ultrafiltration rate
- Transfusion of blood or blood products during dialysis
- Lipid infusion during dialysis
- Dialysis access recirculation
- Use of drip chambers (air exposure, foam formation, turbulence)
- Inadequate priming

Rationale of anticoagulation use during dialysis

Anticoagulation is required to prevent clotting in the extracorporeal as well as prolong the life of the dialyser. Anticoagulation is also crucial to prevent undesirable blood loss and provide constant optimal solute clearance. However, appropriate anticoagulation for heamodialysis requires a subtle balance between under and over heparinization to prevent extracorporeal circuit (ECC) clotting and bleeding respectively

Choices of anticoagulants

- Unfractionated heparin(UFH) –

  Advantages
  - Inexpensive
  - Efficacious,
  - Safe unless the patient has bleeding tendency

  Disadvantages
  - Lack of routine/accurate monitoring of anticoagulant effect
  - Risk of Heparin Induced Thrombocytopenia is greater with UFH
  - Other side effects- hyperkalemia, osteoporosis, lipid disturbances, vascular smooth muscle proliferation and intimal hyperplasia, alopecia, Anaphylaxis, pruritus

- Low molecular weight heparin (LMWH)
- Others such as trisodium citrate, Fondaparinux and prostacyclin

FACTS ABOUT HEPARIN IN HAEMODIALYSIS

- Action is 2 to 4 hours before being broked down by the liver
- Patients differ in their sensitivity to Heparin and how they metabolise it. So Heparrin doses must be Individulised.

- Signs and symptoms of overdose include;

  ◊ Bruising
  ◊ Nose bleeding
  ◊ Bloody urine
  ◊ Voming or coughing up blood
  ◊ Prolonged menstrual beeding on women
  ◊ Black or tar-like stools
  ◊ Prolonged bleeding after dialysis treatment
  ◊ Protamine is an antidote for too much Heparine.
  ◊ It is used in cases of prolonged bleeding and also in regional haparinization
  ◊ It is administered slowly and in small doses
  ◊ Be sure to monitor vitor signs as severe allergic reactions may occur

Assessing coagulation during dialysis

The following are clues which help in assessing anticoagulation status during dialysis.

- Visual inspection
  - Extremely dark blood
  - Shadows or black streaks in the dialyser
  - Foaming with subsequent clot formation in the drip chambers and venous trap
  - Rapid filling of transducer monitors with blood
  - Tethering (blood in the post dialyser venous line segment that is unable to continue in the venous chamber but falls back into the line segment).
  - Presence of clot at the arterial header

- Extra corporeal circuit pressures.
- Measurement of residual dialyzer volume.

Precautions to minimize clotting in the extracorporeal circuit

1. Dialyser priming
   - Following the correct priming technique and adequate priming to prevent retained air in dialyser.

2. Heparin administration
   - Correct loading dose
   - Correct heparin pump setting for constant infusion
   - In time starting of heparin pump
   - Ensuring timely release of heparin line clamp
   - Adequate time interval after loading dose for systemic heparinisation to occur

3. Vascular access
   - Ensuring adequate blood flow by correct needle and catheter position.
   - Correct needle position to prevent recirculation.
   - Adequate uninterrupted blood flow by preventing repeated machine alarm situation

Heparin administration techniques

Decision should be made regarding the correct heparinisation schedule taking into consideration the risk of hemorrhage and other co- morbidities.

A)Routine anti coagulation with unfractionated heparin-

1. **Indication** –
   - Those patients who do not have increased risk of hemorrhage or co - morbidities like CNS bleed, GI hemorrhage, and uremic pericarditis are routinely treated with full dose heparinisation.
2. **Delivery techniques**
   - Intermittent bolus: Bolus loading dose 35-55units/kg, saline flush and wait for 3-5 minutes before initiating HD followed by intermittent maintenance dose of 10-20 IU/kg boluses hourly OR
   - Constant infusion: Bolus loading dose of 35- 55units/kg followed by constant infusion 1000U/hr from the arterial line
3. **Dose of unfractionated heparin**
   - Body weight between 50 – 90 kg: no change in dose
   - Body weight outside these limits: bolus dose 75-100 units per kg; Infusion dose 750-1000 units per hour
4. **Termination of heparin infusion**
   - AV fistula/graft -one hour before end of dialysis
   - Venous catheters- at the end of dialysis
5. **Reversal of over heparinisation**
   - Injection of antidote -protamine 1 mg for every 100 unit's heparin
6. **Target clotting times during dialysis**
   - Activate Partial Thromboplastin Time (APTT) or Whole blood Activated Clotting Time (ACT) test baseline value 120 -150 seconds
7. **Routine heparinisation:**
   - During dialysis desired range baseline +80% - (activated clotting time ACT 200-250 seconds); At the end of dialysis baseline +40 % (170- 190 seconds)

B) Tight heparinisation

1. **Indication –**
   - Patient at slight risk of bleeding
   - Heparin free dialysis unsuccessful due to frequent clotting
2. **Delivery technique**
   - Bolus dose followed by constant infusion 600U/hr from arterial line till termination of HD
   - Do not try intermittent boluses as it will lead to rising and falling clotting times
3. **Dose**
   - Initial bolus dose: 750 units
   - Heparin infusion rate: 600 units per hour
   - Monitor and keep Activated Clotting Time (ACT) at baseline +40
4. **Termination of heparin infusion**
   Continue till end of dialysis
5. **Target clotting times during dialysis**
   - Whole blood Activated Clotting Time (ACT) test baseline value 120 -150 seconds
   - Tight heparinisation: During dialysis desired range baseline +40% (170-190 seconds); At the end of dialysis baseline+40 % (170-190 seconds)

Heparin free dialysis

1. Indication

- Pericarditis
- Recent surgery with bleeding complications or risks
  - -Vascular and cardiac surgery
  - -Eye surgery (retinal and cataract)
  - -Renal transplant
  - -Brain surgery or recent head injury
- Coagulopathy
- Thrombocytopenia /HIT
- Intracerebral hemorrhage
- polytrauma
- Active bleeding /acute bleeding disorder
- In some units the blood lines and dialyzer are pretreated with 2000–5000 U of UFH and then flushed with 1 L of normal saline to coat the lines.

Disadvantage:

- Labour intensive and cost intensive
- Clotting still occurs in 20% of cases with complete clotting of lines or dialyzer, requiring line changes or conversion to low dose heparin in 7% of cases.
- Risk of clotting exacerbated by poor access blood flow, venous catheter, hypotension and concomitant blood transfusion.
- No significant clearance difference compared to full anticoagulation.

2. Technique

- Heparin rinse the extracorporeal circuit (avoid in case of thrombocytopenia) – rinse with saline containing 3000 units heparin per litre
- Drain out heparin containing saline out completely before filling extracorporeal circuit with patients blood or un-heparinised saline at the start of dialysis
- Keep blood flow to 400 ml per minute for a catheter and 350ml per minute for a fistula. In case high blood flow is not possible due to small patient size, very high pre-dialysis plasma urea level
- May use small surface area dialyser or reduce dialysate flow or shorten treatment session.
- Periodic saline rinse allows inspection of dialyser for evidence of clotting. In case clotting detected, consider changing dialyser or terminating dialysis. Remove amount of saline infused by adjusting ultra-filtration.

Anticoagulation in case Heparin use is contraindicated

In situations where the use of heparin is contraindicated and heparin free dialysis is not advisable, the following are alternative anticoagulants like:

A) Bicarbonate dialysis solution with low concentration citrate

This is indicated when heparinisation is contraindicated and heparin free dialysis not possible. The technique uses dialysis solution containing 0.8 millimoles per litre citrate

B) Regional citrate (high concentration anti coagulation)

1. Indication

- When systemic heparinisation is not desirable
- Restricts the anticoagulant effect to the dialysis circuit and prevent systemic anticoagulation

2. Technique

- Infuse tri sodium citrate in arterial blood line
- Use dialysate containing no calcium
- Infuse calcium chloride in venous blood line

Advantage over heparin free dialysis –

- Blood flow rate need not be kept high
- Clotting rarely occurs

Disadvantage of Citrate

- Possibility of metabolic alkalosis- used with caution in patients with liver disease
- chronic citrate used may result in aluminium overload

C) Low molecular weight heparin

1. Dose

- Loading dose: 125-250 IU/kg'
- No intermittent bolus or infusion required.

2. Reversal

- Protamine of no use
- Use plasma if needed

Advantages

- Less osteoporosis
- Better lipid profile
- Less hyperkalemia
- Monitoring not required

Complications

- Bleeding complications – seen in patients receiving clopidogrel and aspirin
- Anaphylactic reactions

Monitoring of Heparin complications

Heparin use may be associated with complications like heparin induced thrombocytopenia (HIT), drug interaction, bleeding events and osteopenia.

There are two types of HIT as shown below:

Heparin induced thrombocytopenia-Characteristics	HIT TYPE 1	HIT TYPE 2
Frequency	10-20%	2-3%
Timing	1-4 days	5-10 days
Platelet count	100 x 1012/L	30-50 x 1012/L
Antibody against heparin-PF4	No (non immune)	Yes
Thrombosis	No	Yes
Skin necrosis	No	Yes
Repeated circuit	No	Yes
Access thrombosis	No	Yes
Management	Observe , usually resolves sponta-neously	Withdraw or use alternative anticoag-ulation – Danap-aroid, Lepirudin or Argatroban

Potential hazardous Drug interactions of heparin

- Analgesics – Increased risk of bleeding with NSAID – avoid concomitant use with IV diclofenac.
- Anti-platelets such as clopidogrel increases the risk of bleeding
- Nitrates- Anti coagulant effect reduced by infusion of gyceryl-trinitrate
- Haemolysis ans Hyperkalemia.

DIALYSIS PRESCRIPTION

Dialysis modality	Haemodialysis		
Dialysis frequency and duration	Conventional hemodialyis:Day time,3-5 hours session and 3-4 sessions per week		
Dialysis dose	Minimum delivered Kt/V	of 1.2	(Sp KT/V)
	The determinants of urea clearance are; Blood Flow rate: standard =≥ 300 ml/min and Low flow=≤ 300 ml/min Dialysate Flow rate: standard = ≥ 500ml/min and low flow =≤500 ml/min Membrane dialyzer efficiency		
Dialysate	Sodium : 135-145 mmol/l Potassium: 2-4 mmol/l,better survival is associated with pre-dialysis potassium of 4.6 -5.3 mmol/l · Calcium : 1.25-1.50mmol/l(2.5-3 mEq/l) Bicarbonate: target pre-hemodialysis of ≥22 mEq/l and avoid severe post hemodialysis alkalosis Temperature: Cool dialystate of 35- 36 degrees celcius may reduce intra dialytic hypotension and increase Mean Arterial Pressure		
Ideal body weight	Shorter dialysis session and inter dialytic weight gain is associated with higher mortality		
Initial dialysis prescription	Reduce haemodialysis treatment if serum level of urea is high e.g. ≥240 mg/dl A 2 hours' session is recommended Aim at urea reduction rate of ≤ 40 % The subsequent dialysis sessions may be increased to standard 3-5 hours		

POSSIBLE INDICATIONS TO CONSIDER TO INCREASING DIALYSIS DOSE

- Ureamic encephalopathy
- Ureamic pericarditis
- Anaemia
- Unexplained nausea and vomiting
- Sleep disturbance
- Restless leg syndrome
- Uncontrolled hyperphosphotaemia
- Evidence of fluid overload
- Hyperkalaemia
- Pruritis
- Metabolic acidosis unresponsive to oral bicarbonate

HAEMODIALYSIS ADEQUENCY

Hemodialysis provides only a fraction of some of the numerous functions performed by the native kidneys. Patients receiving inadequate dialysis suffer from malnutrition, inflammation, and a poor quality of life. Additionally, these conditions may lead to accelerated atherosclerosis. Inadequate dialysis has been shown to adversely affect survival, while increasing frequency and duration of dialysis improves survival, quality of life and medication amounts. No single clinical or laboratory parameter can assess adequacy of dialysis and overt signs may develop very late, hence it is necessary to develop a comprehensive monitoring system of measuring adequacy of dialysis which is also easy to use and reproducible.

Recommendations

- Kt/v or urea reduction ratio (URR) should be used as a measure of dialysis prescription.
- URR is a simpler method to determine dialysis adequacy. URR should be targeted to > 65 %. Post dialysis sample should be taken 2 minutes after dialysis or during slowing pump speed to 100 ml/min and sample taken 15 seconds later.
- An assessment of the dialysis dose in stable hemodialysis patients should be performed once per month. More frequent measurements may be required in patients not doing well on dialysis.

If there is low Kt/V or URR look for the possible underlying causes by assessing the following:

- Fistula integrity
- Treatment duration
- Possible technical errors in the method of obtaining BUN samples
- Dialysis machine and patient specific variables such as:
    - ◊ Inadequate machine calibration
    - ◊ Low blood flow rates
    - ◊ Hypotensive episodes that require changes in treatment
    - ◊ Overestimation of dialyzer clearance.

Measures should be incorporated to improve effective hemodialysis treatment times, improve blood flows, correct errors in blood sampling, or improve dialyzer clearance.
Dialysis adequacy also needs to be assessed in addition by the clinical well-being of the patient assessed by a regular monthly clinical checkup of dialysis patients in the clinic along with monthly hematology and biochemistry

reports & other tests as required.

Basic criteria to be met by adequate dialysis

- Fluid removal permitting return to correctly evaluated 'dry weight' at end of dialysis.
- Predialysis blood pressure < 140/90 mmHg with or without antihypertensive drugs.
- Predialysis plasma concentrations :
    - ◊ Potassium: ≤5.5 mmol/l without adsorption of ion exchange resins.
    - ◊ Bicarbonate: ≥22 mmol/l.
    - ◊ Phosphate: ≤5.5 mg/dl without oral binding agents.
    - ◊ Urea: <35 mmol/l with daily protein-intake 1.2 g/kg/BW.
    - ◊ Albumin: ≥4 g/dl.

Technical requirements for delivery of adequate dialysis

- Vascular access: blood flow ≥ 300 ml/min.
- Dialysis fluid: bicarbonate buffered, sterile, pyrogen-free, QD: ≥500 ml/min.
- Dialyzer: Highly permeable, biocompatible membrane. Suitable surface area.
- Dose of dialysis: Minimum Kt/V of: 1.2–1.4 (single pool), Minimum urea reduction rate of 65–70% and a weekly dialysis time of ≥12 h (4–4.5 h × 3).
- Haemoglobin: 10–12 g/dl with or without rHu-EPO.

Criteria for Optimal Dialysis

- Normalized blood pressure with minimal antihypertensive medications.
- Normalized calcium-phosphate product with neither phosphate binders nor phosphate supplements.
- An absence of intradialytic symptoms such as hypotension, cramps, and nausea.
- An absence of interdialytic symptoms.
- No interference with ability to hold a job.
- Protein appetite under the patient's free will.
- Neither alkalotic nor acidotic.
- Hematocrit in the 35-to-38 range with the use of at least 50% or less of today's average erythropoietin dose.
- No dialysis-related or access-related hospitalizations.
- Normal triglyceride level.
- No evidence of amyloidosis.
- The longest preservation of residual kidney function.
- Life expectancy approximately that of living-related-donor transplants.
- No evidence of left ventricular hypertrophy

Explanation & Discussion

Note 1: BUN & creatinine are insufficient measures of dialysis adequacy because a low BUN & creatinine may reflect malnutrition & poor muscle mass rather than sufficient dialytic removal, also a common clinical state in our dialysis population. Protein catabolic rate (PCR) and timed average urea concentration have been shown to be important determinants of morbidity & mortality as shown by National cooperative dialysis study (NCDS).

Note 2: Urea clearance has been used mechanistically in a formula kt/v & shown to reflect the amount of dialysis prescribed & delivered.

Note 3: Kt/v is defined as dialyzer clearance of urea (k obtained from manufacturer of dialyzer & is available as ml/min), multiplied by duration of dialysis and divided by volume of distribution of urea in the body (v in ml), which is approximately equal to total body water. Individualizing dialysis prescription is a useful method to achieve a cost effective dialysis treatment. Dialysis dose can be measured by Kinetic urea modeling (kt/v) or by simple urea reduction ratios (URR).

Note 4: There is no universally accepted target value for the Kt/V. It is recommended that target single-pool Kt/V of approximately 1.4 to 1.6 be achieved. These levels are consistent with the 2006 K/DOQI guidelines for hemodialysis patients with minimal residual renal function (less than 2 mL/min per 1.73 m2).

Common complications of Haemodialysis and Prevention

1. Patient-related complications

- Hypotension
- Muscle cramps
- Nausea and vomiting
- Headache
- Itching
- Fever and chills
- Hypertension
- Chest pain
- Disequilibrium syndrome
- Pyrogenic reaction

2. Technical-related complications

- Blood clotting
- Blood leak
- Power failure
- Air in blood lines
- Dialyzer reaction
- Haemolysis

Hypotension management

- Position patient in Trendelenburg position
- Reduce ultrafiltration rate or stop ultrafiltration
- Give Intravenous Fluids 0.9% Normal saline (between 100mls and 500mls bolus)
- Reduce machine from 37 degrees Celsius to 35.5 degrees Celsius
- Reduce blood flow rate
- Check Blood Pressure every 5 minutes until stable Blood Pressure
- Change to colloids if blood pressure is still low (max 500mls bolus).
- Re-transfuse if still no response.

If not responding refer to critical care management:

- Give oxygen as per requirement
- Give Adrenaline 1mg Intravenous push if blood pressure is still low.
- Consider sodium modelling

PREVENTION

- Avoid eating while on dialysis treatment
- Do not use antihypertensives prior dialysis
- Consider sodium modelling
- Increase dialysis treatment time OR
- Increase frequency of dialysis sessions

Hypoglycemia management

- Give 50mls of 50% Dextrose IV push in adults. Give 5ml/kg 10% Dextrose in children
- Let the patient eat if they can
- Use glucose based dialysate in patients who cannot eat,
- Check blood sugar after 15 minutes then after 30 minutes then hourly.

PREVENTION

- Avoid use of glucose free dialysate.
- Insulin therapy and oral hypoglycaemic agents should be used with caution in patients on dialysis.

Bleeding from catheter insertion site

- Stop dialysis and reinfuse the blood
- Apply continuous direct pressure on the catheter insertion site for 5 to 10 minutes
- Suture the catheter insertion site
- If bleeding continues, remove the catheter and apply direct pressure for 5 to 10 minutes
- Apply a pressure bandage

PREVENTION

- Avoid use of overdose heparin
- Use regular gauze and adhesive plaster for compression to prevent bleeding
- Use low molecular weight heparin than unfractionated heparin
- Secure the catheter with a suture

Clotting in the dialysis lines

- Flush lines with Normal Saline until the venous chamber is clear
- If no clots use same blood lines and increase heparin dose and pump speed
- If clots in the lines don't reinfuse, discard the lines and replace with new
- bloodlines.
- Transfuse blood if Hb is 6mg/dl or below
- Check FBC immediately
- Recheck after 48hours

PREVENTION

- Titrate dose of heparin according to patient needs
- Prime the extracorporeal circuit at low pump speed 100 to 200mL/min
- Expel all air contents from the circuit before commencing dialysis
- Avoid low pump speed of below 200mL/min in chronic patients with good access

Muscle cramps

- Stop ultrafiltration
- Give 100ml Normal saline intravenously as bolus
- Massage the affected area

PREVENTION

- Stretch and massage muscles
- Advise patients that salts should be within allowable limits potassium, sodium, calcium and magnesium.
- Prevent hypotension
- Avoid excessive fluid removal.

Dialysis disequilibrium syndrome

- Maintain airway
- Hyperventilate the patient
- Give oxygen 3 -5 liters/minute in adults., in paeds 0.5-3L/min
- Lower Blood Flow Rate between 150 to 200mls/minute
- Mannitol 0.5 t0 1g/kg
- Give glucose 50% dextrose, in paeds give 10% dextrose 5mL/kg
- Terminate session if no improvement
- Consider hemofiltration only if severe fluid overload followed by haemodialysis
    - Use small dialyzer
    - Give Antiemetic (promethazine 25mg /IM) if vomiting

- Give anticonvulsants (diazepam 10mg /IV) if convulsing

PREVENTION

- Recognize those at risk
- Choose small dialyzer
- Use Blood Flow Rate between 150 to 200mls/minute
- Consider performin5g ultrafitration only followed by haemodialysis or VICE VERSA in severe fluid overload
- Replace urea with another osmotically active substance during dialysis session such as mannitol 1g/kg
- Consider dialysate sodium chloride concentrations of 144-154 mmol/L
- Use a lower bicarbonate dialysate concentration to improve acidosis more gradually

Fistula rupture

- Call for help
- Colleague should call theatre team
- Apply tourniquet on the forearm
- Apply pressure with gauze on the anastomosis
- Rush patient to theatre while still applying pressure

PREVENTION

- Identify those at risk; thinning of vessel wall, exclude low flows associated with impending vascular access failures (look, touch and listen)
- Document on dialysis treatment record or progress notes
- Cannulation should not be continued along any type of aneurysm
- Avoid long standing high blood flow rates that result in shear forces causing damage to the AVF
- Avoid repeated needle punctures
- Manage infections of false aneurysm promptly
- Preserve the maximum area of cannulation
- Identify adverse trends and take action if indicated
- Use of antibacterial soap to clean the AVF site
- Perform early vascular surgical intervention before an episode of life threatening haemorrhage
- Reduce or avoid medications that increase the risk of bleeding complications such as aspirin

Dialyzer reaction

Type A (anaphylatic reaction)

- Occurs very shortly after dialysis is initiated typically within the first few minutes
- Stop dialysis
- Do not return blood. Discard the blood
- Provide cardiorespiratory support
- Administer IV antihistamines
- Administer adrenaline 1mcg/kg IV
- Give steroids; hydrocortisone slow IV

Type B

Signs and symptoms are more delayed and less severe.

- Continue dialysis as required
- Give oxygen as needed

PREVENTION

- Use a more biocompatible membrane and dialyzer not sterilized with ethylene oxide (ETO)
- Syringes, needles, and infusion sets made from ETO should not be used during dialysis
- Rinse the dialyzer and blood tubings with 3L normal saline without Heparin
- Give pre-medications: antihistamines & Methylpredinisolon (Urbason)20mg IV at the start of dialysis
- Change B-blockers to a-blockers
- Try using medium flux dialyzers
- Avoid combination of polyacrylonitrile (PAN) membrane and ACIs

Air embolism

Characterized by sudden onset of shortness of breath, dropping blood pressure and restlessness

- Stop hemodialysis
- Place patient on Left side or in trendelenburg position
- Administer 100% oxygen 3L/min or more
- Assess vital signs and pulse oximetry
- Attempt aspiration of air
- Document the event, actions taken and the client's response

PREVENTION

- Avoid high blood flow rates.
- Blood flow rate should collate with needle gauze
- Keep leur lock tightened
- Prime adequately the dialyzer and tubings before dialysis

Ensure good patency of the dialysis catheter

Peritoneal Dialysis (PD)

This is another type of a renal replacement therapy which filters blood through the lining of the abdomen called peritoneum, a semi permeable membrane where fluid and solutes are exchanged between the peritoneal cavity and blood capillaries

- Waste products and excess body fluids are removed from blood to dialysate in the peritoneal cavity through the peritoneum.
- Peritoneal cavity acts as a reservoir for the dialysate
- Abdominal cavity is lined by a vascular peritoneal membrane which acts as a semi- permeable membrane. Peritoneal membrane is large and porous across where excess body fluids and solutes including uremic toxins pass through
- PD occurs through diffusion and osmosis across semi-permeable membrane and capillaries.
- Allows uremic toxins (urea, creatinine.,) and water to move from an area of higher concentration, in blood to an area of lower concentration in the dialysing fluid (dialysate) contained in the abdominal cavity through the process of diffusion
- Allows removal of excess water (ultrafiltration) due to osmotic gradient generated by glucose in dialysate
- The process of PD is an intracorporeal method where the cleaning of blood happens inside the body. Blood is not seen outside the body circulating on the machine

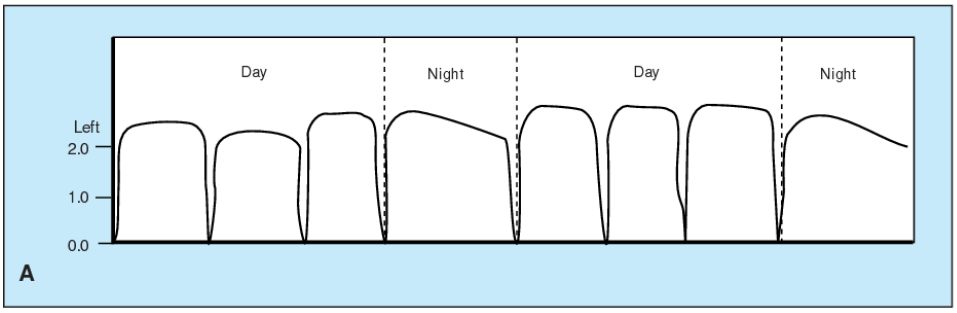
TYPES OF PERITONEAL DIALYSIS

- Continuous Ambulatory Peritoneal dialysis (CAPD)
- Continuous Cycling Perotoneal Dialysis (CCPD)
- Automated Peritoneal Dialysis (APD)
- Intermittent Peritoneal Dialysis (IPD)

CAPD

- It is done manually by patients or caregivers without a machine.
- considered dose is four exchanges/ day, each 4 hours with 2L of PD solution (Dianeal), 4 to 8 hours dwell
- Exchange occurs 7 days in a week
- carried out during day time
- Patients can continue activities while dialysate dwells in the abdomen
- CAPD: C – Continuous, carried on all the time
  A – Ambulatory, once exchange is performed, one can move around and carry out normal activities
  P – Peritoneal, uses the peritoneum as a filter
  D – Dialysis, the process of removing excess water and waste from the blood

Diagram showing PD exchanges in 24 hours



APD

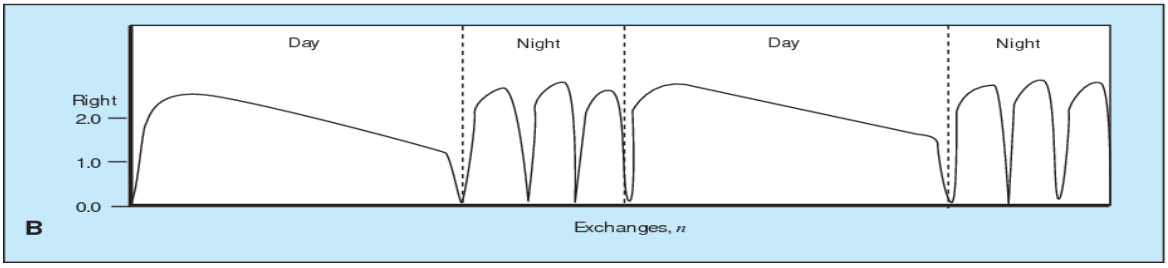
- Often used in acute setting,
- Uses a machine that cycles through multiple exchanges at night
- Machine fills abdomen with dialysate, allows to dwell before draining
- Considered dose is 8-10 hours night dialysis with day dwell
- Patient remain attached to machine for the prescribed hours

CCPD

is a form of automated dialysis that uses an automated cycling machine, multiple exchanges are performed at night when patient is asleep

The final exchange of the night is left to dwell through the day and is drained in the next evening as process is repeated

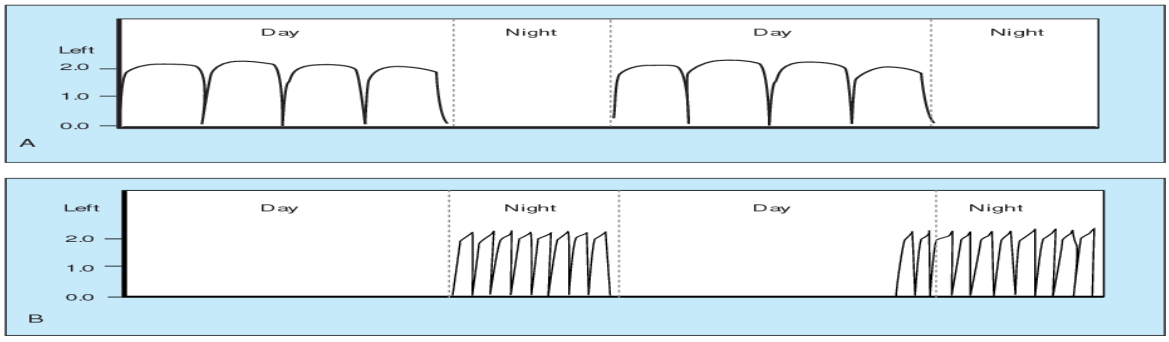
Dialysis occurs 24 hours a day, 7 days a week



I PD

Patient requires exchange of 2L dialysate to go for 60 minutes interval, then 20minutes drain, multiple exchanges (>20sessions) of 2L three times weekly are needed.

Intermittent can be automated or manual



Advantages of Peritoneal Dialysis

- Can be done at home
- Privacy, flexibility and comfort of home dialysis
- Helps to preserve residual renal function
- Client visit the hospital once a month for check-up
- Dialysis supplies are portable
- No needle pricks to perform the procedure
- No travelling to dialysis centre to perform treatment

- No blood thinning medication is required i.e. heparin
- It is ambulatory, no machine required
- Less dietary and fluid restriction
- Better BP control
- Freedom in scheduling, can be done at a convenient time based on work schedules, can be performed anywhere hence more control over on life

Disadvantages of Peritoneal dialysis

- It is done every day, there's is no day off
- Client does own care
- Time consuming
- Risk of peritonitis
- Sterile technique is required
- Permanent catheter
- Contraindicated in abdominal surgeries, chronic backpain or hernia

INDICATIONS FOR PD

- Unstable patients who cannot tolerate anticoagulation
- Vascular access problems
- Older adults
- Patients with a "virgin abdomen" or minor prior abdominal surgery
- Reasonable abdominal musculature, i.e., exclude those with significant obesity and/or hernias
- Physically capable of doing bag changes in particular, good visual acuity and limb dexterity
- Who can perform repetitive bag changes per sterile protocol
- Suitable storage space for dialysate
- Adequate hand washing facilities
- PD requires patient training and dedication

CONTRAINDICATIONS FOR PD

- Peritoneal adhesions
- Extensive intra abdominal surgery
- Morbid Obesity
- Recurrent episodes of peritonitis
- Abdominal malignancies
- Severe respiratory diseases
- Hernias
- Chronic backpain
-

PERITONEAL DIALYSIS PRESCRIPTION

- Type of modality
- Type of PD solutions
- Number of infusion volume
- Duration of dwells

TYPE OF PD SOLUTIONS

- Conventional solution glucose based (dextrose):
  - ◊ PD fluids come in concentration of 1.5%, 2.5% and 4.25%,
    - » 1.5% Dianeal, - hypotensive, normal levels of electrolytes
    - » 2.5% Dianeal, - maintenance
    - » 4.25% Dianeal - fluid overload
  - ◊ Two litres each bag.
  - ◊ The PD fluid contains dextrose and minerals
- Non - glucose based, Amino acid dialysate (Nutrineal)
- Icodextrin based solution (Extraneal) which has a long dwelling time, and more ultra- filtration in high transporters, low glucose load and better glycaemic control. May be good for diabetic patients

NUMBER OF INFUSION VOLUME

- Volumes of 2 to 2.5 Litres with each exchange for adults
- Paediatrics 20 to 30mL/Kg/exchange maintenance dose
- DAY 1 and 2 (PAEDS)
- ◊ The higher levels of the urea the smaller the fill volume at initiation to prevent abrupt drop of urea which can lead to dialysis disequilibrium syndrome (DDS).

UREA >400mg/dL	Urea 200 – 400mg/dL	Urea <200mg/dL
Fill volume: 5mL/kg Fill time: 1-10 min. Dwell: 2-3 hours Drain time: 10-20 min. # Exchanges: 2	Fill volume: 10mL/kg Fill time: 1-10 min. Dwell: 2-3 hours Drain time: 10-20 min. # Exchanges: 2	Fill volume: 10mL/kg Fill time: 1-10 min. Dwell: 2-3 hours Drain time: 10-20 min. # Exchanges: 3
GOAL Urea decrease	Urea decrease <40/day	Urea decrease <50/day

<20/day	
Day 3 increase gradually 10 to 20mL/Kg , exchanges 3 to 4/day Maintenance # Exchanges: 4-6 Shorter dwell times for life-threatening emergencies: Hyperkalemia - K>7, Severe fluid overload (requiring >1L oxygen) and Severe acidosis	

THE PD ACCESS INSERTION TEAM

- All renal physicians should be familiar with the percutaneous technique of insertion of the Tenckhoff catheter
- Each centre should have a dedicated surgical/urology team involved in the implantation and care of peritoneal catheters•
- The access team should comprise nurses, nephrologists, urologists, and surgeons who have experience in peritoneal dialysis and understand the importance of successful access placement and the need for attention to detail in the reduction of complications.

DURATION OF DWELLS:

- 1 to 8 hours depending on type of PD fluid
- Typically, performed 4 times in a day with exchange times at 7:00 am, 12:00 noon, 5:00 pm and10:00 pm.

EQUIPMENT AND RESOURCES

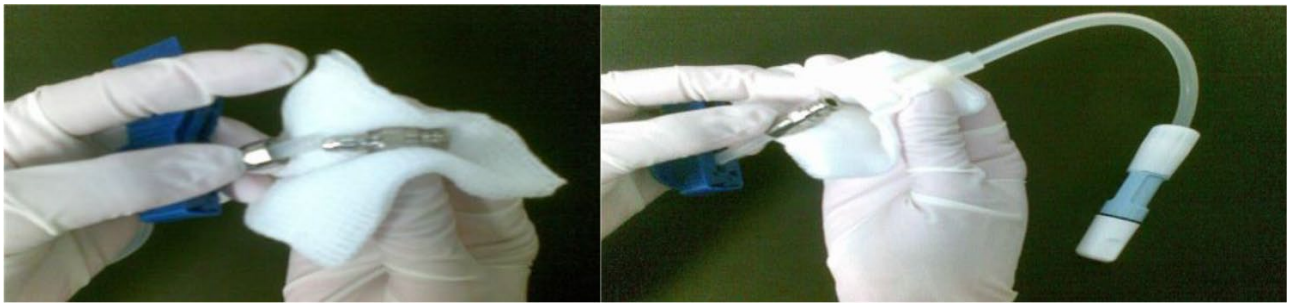
- CAPD and APD require back up of haemodialysis services.
- Dedicated PD nursing staff should be part of the multidisciplinary team.
- Hand washing facilities i.e. buckets with lever tap, soap, hand sanitizer
- Face masks, plaster, antiseptic solutions, gauze and cotton swabs
- PD consumables
- Weighing scale
- Glucometer

INSERTION PROTOCOL

- Treatment units should have clear protocols for catheter insertion and care, including the use of antibiotic prophylaxis
- Perform catheter insertion at least 2 weeks before starting PD.
- Consult surgeons for PD catheter placement
- Obtain informed consent before catheter insertion
- PD nurse will train the patient and family on catheter care and how to perform PD at home.
- Insertion should not be delegated to inexperienced, unsupervised operators

Preoperative:

- check for hernias
- identify a catheter of suitable length;
- mark the exit site with the patient sitting or standing;
- screen for nasal carriage of methicillin- resistant Staphylococcus aureus (MRSA) and Staphylococcus aureus
- Pre-implantation: prepare the bowel with laxatives; ensure bladder emptying and prepare the surgical site;
- Use antibiotic prophylaxis Ceftriaxone 2G intraperitoneally upon insertion
- Titanium and transfer set SHOULD BE connected right in theatre and every six months



Post-procedure:

- flush catheter and cap off;
- immobilise the catheter;
- cover the exit site with a non-occlusive dressing
- If possible, do not disturb the catheter for 5-10 days post insertion;
- Supply patient with laxatives
- Give Ceftriaxone 2g intraperitoneally as prophylaxis and apply topical antibiotic for exit site care
- Provide patients with hand sprays sanitizers upon discharge when going home

INITIATING PD TREATMENT

- Small dialysate volumes in the supine position can be used if dialysis is required earlier.
- If paeds move the child to HDU for daily weights, BPs and other vital signs monitoring
- Labs (FBC, U&Es, HIV, Hep B, Hep C, urine dipstick, urine culture if febrile)
- Renal ultrasound
- Consult Renal Team
- Obtain informed consent (from guardian if a child)

PROCEDURE

- Each procedure consists of 3 phases: fill, dwell and drain
- PD catheter is surgically placed into the abdominal cavity for infusion of PD fluids
- Usually 1 to 2 litres per session (fill)
- Fluid stays (dwell) in the peritoneal cavity for prescribed duration
- Fluid then flows out of the cavity (drain) by gravity into the drainage bag called PD effluent which contains the dialysate and excess waste, electrolytes
- Inspect each bag of solution for signs of contamination, expiry date, volume, concentration before use

PRE-DIALYSIS CARE

- Document vital signs to assess fluid volume status and tolerance of dialysis treatment
- Daily weight check
- Check biochemistries to assess efficiency of treatment
- Measure abdominal girth, increased may indicate retained dialysate, excess fluid volume or early peritonitis
- Maintain fluid and dietary restriction to prevent hypervolaemia and azotaemia
- Have client empty bladder prior to catheter insertion to prevent punctures

INTRA-DIALYSIS CARE

- Use strict aseptic technique during the procedure.
- Peritonitis is a common complication of peritoneal dialysis; sterile technique reduces these INFECTIONS.
- Add prescribed medication to the dialysate
- Prime tubings with PD solution and connect to the PD catheter, avoid kinks
- Instill dialysate into abdominal cavity within 10 minutes and follow the rest
- Clamp tubing and allow the dialysate to remain in the abdomen for the prescribed dwell time.
- Keep drainage tubing clamped at all times during instillation and dwell time. Dialysate
- should flow freely into the abdomen if the peritoneal catheter is patent.
- Dialysis, the exchange of solutes and water between the blood and dialysate, occurs across the peritoneal membrane during the dwell time.
- During instillation and dwell time, observe for signs of respiratory distress, such as
- dyspnea, tachypnea, or crackles. If develops, place in Fowler's or semi-Fowler's position and slow the rate of instillation slightly to relieve respiratory distress. This may result from overly rapid filling or overfilling of the abdomen
- After dwell time, allow to drain by gravity into a sterile bag.
- Observe the clarity, color, and odor of the PD effluent.
- Blood or faeces in the dialysate may indicate organ or bowel perforation;
- Cloudy or malodorous dialysate may indicate an infection.
- Accurately record fill volume and type of dialysate including medications added, dwell time, and amount

and character of the drainage.
- Monitor BUN, serum electrolyte, and creatinine levels to assess the effectiveness of dialysis

PD COMPLICATIONS AND MANAGEMENT

- Infections: exit site infection, tunnel infection and Peritonitis
- Blockage of catheter
- Dialysate leakage due to high pressure in the abdomen
- Bleeding
- Abdominal hernias
- Catheter blockage by fibrin
- Genital oedema
- Hyperglycaemia due to glucose in the dialysate
- Lower back pain
- Excessive fluid removal
- Hypoalbuminemia
- Bladder perforation
- Bowel perforation

EXIT SITE INFECTION

- Suggested by pain, swelling, crusting, erythema and serous discharge; purulent discharge
- Swabs should be taken for culture and initial empiric therapy should be with oral antibiotics that will cover Staphylococcus aureus and P. aeruginosa
- Clean exit site daily with anti-bacterial soap and water then dry
- Put on a clean dressing
- Secure the catheter with tape
- Apply a waterproof dressing over the top of the dressing when showering.
- Sterile packs should be used when dressing and provide Chlorhexidine solution for cleaning exit site
- Between exchanges, catheter and transfer set should be hidden inside pouch

PERITONITIS

- Peritonitis is the infection of the peritoneum.
- Peritonitis should be treated as quickly as possible to maintain effective treatment and prevent removal of PD catheter.
- PD units should undertake regular audit of their peritonitis and exit-site infection rates, including causative organisms, treatment and outcomes.
- The flush-before-fill dialysis delivery systems should be used
- Intraperitoneal administration of antibiotics is superior to Intravenous dosing for treating peritonitis;
- Intermittent and continuous dosing of antibiotics are equally efficacious
- Once culture results and sensitivities are known, antibiotic therapy should be adjusted to narrow spectrum agents as appropriate.
- For patients with substantial residual renal function the dose of antibiotics may need to be adjusted.

Signs and Symptoms of Peritonitis

- Cloudy effluent
- Abdominal pains (cramping)
- Fevers
- Nausea and vomiting

Management

- Send the whole bag or hang the bag of the PD effluent for about 30 minutes and collect sample from the most dependent part to Lab to check for culture and sensitivity
- Treat peritonitis promptly with Intraperitoneally antibiotics according to culture sensitivities.

PREVENTION OF PERITONITIS

- PD catheter placements, usage, and access should be done in as sterile a fashion Catheter should be tunneled during placement.
- Hand washing and use of hand gel should be emphasized
- Keep the exchange area clean and tidy
- Do not touch the connections
- Do not use a bag that is leaking
- Clean exit site daily
- Treat promptly if an exit site infection is suspected
- During exchanges,
    - ◊ keep small children and pets away when performing PD
    - ◊ Keep windows closed and fans switched off when doing PD
    - ◊ All staff members and individuals nearby should wear masks
    - ◊ Place a screen between patients and other staff not wearing masks.
    - ◊ Sterile gloves, gown, masks should be worn by all staff.
    - ◊ catheter and transfer set should be hidden inside pouch
    - ◊ Inspect each bag of solution for signs of contamination, expiry date, volume, concentration before use
- Give a prophylactic dose of ceftriaxone
- Apply topical antibiotic when catheter is accessed for an exchange.
- Patients who experience abdominal pains due to PD infections should be given lignocaine 1mL of 1% intraperitoneally
- Store supplies in a cool, clean, dry place to prevent damage

BLOCKAGE OF CATHETER

Slow dialysate instillation.

- ◊ Increase the height of the bag and reposition the client.
- ◊ Check tubing for kinks

Poor dialysate drainage or flow.

- ◊ Poor flow may be related to a partially obstructed tube or catheter.
- ◊ Lower the drainage bag, reposition, check for tubing kinks.
- ◊ Add Heparin 500u to each litre of PD fluid until problem is resolved if cause is clots or fibrin
- ◊ Catheter migration – consider plain abdominal Xray and reposition the catheter

LEAKAGE AROUND THE CATHETER.

- ◊ Slow dialysate

EXCESS DWELL TIME.

- ◊ Prolonged dwell time may lead to water depletion or hyperglycemia.

DIETARY ADVICE FOR PATIENTS ON PD

Limit;

- Sodium
- Phosphorus
- Calories due to glucose in PD fluid
- Add protein to your diet because PD removes protein
- Advise patients to choose foods with the right amount of potassium

HOME VISITS

- Home visits will be intensified to ensure standard procedures for PD are followed.
- 1st visit – prior to insertion of the PD catheter to verify suitability
- 2nd visit – upon discharge from the hospital
- Subsequent visits – Every quarter

PD ADEQUACY TARGET

Kt/V

- Dialysis adequacy is measured by Kt/V
- Kt/V measures the clearance of waste products in urine and PD effluent
- The test is done at one month then every six months
- Target Kt/Vurea of ≥2.0/week OR a creatinine clearance of 60L/week/1.73m2 is the minimum treatment dose
- Weekly Kt/V for PD

K = Volume cleared of urea/time (litres/day) T = time in days
Vdurea = total body water in litres
Kt/V in over 1 day X 7 = adequacy over 1 week

For example: 70kg patient is infusing 4 exchanges per day each exchange 2L of 2.5% Dianeal and drains out a total of 10L/day, in a week drains 10L X 7days = 70L/Week/1.73m2. If estimated total body water volume is 35L, Kt/V=70L/35L. Kt/V = 2.0

PERITONEAL EQUILIBRATION TEST (PET)

- Measures the performance/functionality of the peritoneal membrane
- PET is performed at 6 weeks after commencing treatment and at least biannually/annually
- The PET process starts with a long overnight dwell of 8 -12 hours. The overnight dwell is drained in the morning. Then, 2L of 2.5% dianeal is infused over 10mins, every 2minutes patient should roll from side to side to mix the dialysate, once the infusion is complete , immediately drain 200Ml OF DIALYSATE. Mix the drained dialysate by inverting the bag several times then obtain a 10mL sample USING ASEPTIC TECHNIQUE, reinfuse back the remaining, the 4 – hour dwell patient should be ambulatory . At 2 – hour timepoint, repeat collection steps (drain 200mL, mix, aseptically obtain 10mL sample THEN REINFUSE) AND TAKE A BLOOD SAMPLE for serum measurements. After 4 hours completely drain the patient for 20 minutes, measure drain volume then collect a 10mL sample for analysis of urea, creatinine and glucose
- A 4- hour Dialysate/plasma ratio of 0.59 and D/DO glucose ratio is 0.47 patient would be classified low average transporter

PET calculations

cr /P cr = dialysate concentration of creatinine at 0, 2, and 4h, divided by serum concentration of corrected creatinine.
D/D 0 = dialysate glucose concentration at 2h and 4h, divided by dialysate glucose concentration at 0h.

- ◊ High (>0.8) and high-average (0.65-0.8) transporters equilibrate rapidly, their transport for urea and creatinine is fast which also means they lose the glucose gradient rapidly so ultrafiltration is limited. Hence need shorter dwells and frequent sessions to avoid reabsorption. Icodextrin should be considered for daytime dwells. Dry days may be possible.
- ◊ Considered for APD
- ◊ While LOW (<0.5.) and LOW- AVERAGE (0.5-0.64) transporters equilibrate slowly, transport of urea and creatinine is slower which result in good ultrafiltration with minimal reabsorption even for a long day dwell. May require longer dwells with higher volume exchanges to get adequate clearance. Dry days may not be possible. Considered for CAPD/CCPD
- ◊ During PET ensure, overnight long dwell is performed, complete drain is done, fill of 2L of 2.5% glucose, filling and draining in specified time 0hour, 2hours and 4 hours, taking samples at right time, complete mixing of samples, ambulation during 4 hour-dwell and label samples correctly.

WHERE THERE IS NO PD FLUID

Ringer's lactate (RL) can be used where there's no commercially available PD fluid. Trained staff in the most sterile environment should prepare this due to HIGH risk of iatrogenic infections/peritonitis.

PD Fluid Concentrations	1.5%	2.5%	4.25%	
	RL	1L	1L	1L
	D50%	30mL	50mL	80mL

INDICATIONS FOR SWITCHING FROM PERITONEAL DIALYSIS TO HAEMODIALYSIS

- Inadequate solute transport or fluid removal
- Unacceptably frequent peritonitis or persistent peritonitis
- Other PD-related complications
- Development of technical/mechanical problems
- Severe malnutrition resistant to aggressive management (relative).

PREVENTION OF INFECTIONS IN HEMODIALYSIS UNIT

The hemodialysis patient is particularly susceptible to several infections both bacterial occasioned by the decreased immunity and blood borne viral infections. Studies have shown that bacterial infections in addition to carrying a higher short term mortality also increase the risk of long term cardiovascular complications. Viral infections like Hepatitis B and C progress to liver cirrhosis and increase the morbidity and mortality on hemodialysis. In addition, the staff of a dialysis unit are uniquely at risk of contracting these viral infections from contaminated blood and dialysate. Preventing the transmission of infections involves several links in the chain involving the patients, the dialysis procedure and ancillary care, the staff of the unit and various administrative and waste disposal protocols. Malawi Infection prevention protocols should be used as reference material for IP. Comprehensive infection preventive protocol includes hygiene measures, vaccination, dialyzer reprocessing and disposal of biohazardous materials as set out in the following guideline.

The number of patients on maintenance hemodialysis is increasing rapidly in Malawi. Chronic hemodialysis patients have an increased infection risk.
HD facility is very conducive for transmission of infection since multiple patients receive dialysis concurrently.

Transmission can occur directly or indirectly via contaminated devices, equipment and supplies, environmental surfaces, or hands of personnel.

The important infections that develop in these patients include viral infections such as hepatitis B and C, HIV and bacterial infections, especially those involving vascular access.

Environmental Issues including Equipment and Consumables

- Storage of equipment close to dialysis machines and patients should be minimized.
- Where possible, regularly used equipment such as adhesive tapes, tourniquets, blood pressure cuffs and clamps should be designated to each patient.
- Consumables taken to the patient's station should be used only for that patient and should not be returned to a common clean area or used on other patients.

Cleaning of dialysis machines and chairs/beds

- Dialysis machines should be internally disinfected, externally cleaned (and disinfected if indicated), and dried after each patient.
- The exterior of the machine should be effectively cleaned using protocols following manufacturer's instructions.
- Special attention should be given to cleaning control panels on the dialysis machines and other surfaces that are frequently touched and potentially contaminated with patients' blood.
- Cleaning of non-critical surfaces (e.g. dialysis bed or chair, countertops, external surfaces of dialysis machines and equipment) should be done with neutral detergent and warm water.
- The following procedure should be adopted for any surface/item that is visibly contaminated with blood OR following dialysis of a patient infected with blood borne virus:
  -Clean with neutral detergent and water, and then
  -Disinfect with sodium hypochlorite 1% (1,000 ppm available chlorine; 1:10 dilution).
  -Remove chlorine residues from metallic surfaces with water as sodium hypochlorite in high concentrations (>500 ppm) is corrosive to metals.
- The machine should be decommissioned if spillage occurs at inaccessible locations, such as behind the blood pump until proper cleaning and disinfection are done.

The following practices should be avoided

- -Blood tubing draped or clipped to waste containers,
- -Use of attached waste containers during priming of dialyzers
- -Placing items on tops of machines for convenience (e.g., dialyzer caps and medication vials

Due to the instability of chlorine compounds all diluted solutions should be discarded at the end of the day.

Disinfection of Haemodialysis Machines

- Dialysis units must follow the manufacturer's recommendations in relation to management of haemodialysis machines
- Manufacturers producing dialysis machines each recommend a different procedure for decontamination, but they concentrate only on bacterial kill. It is recommended that efficacy of decontamination procedure should additionally take into account level of biofilm and endotoxin removal.
- The development of bacterial biofilms in the hydraulic circuit of haemodialysis machines can be prevented by frequent use of chemical and heat disinfection strategies.
- Disinfection should include the following
    - ◊ Heat disinfection (80°C to 90°C) after each dialysis
    - ◊ Citric acid and heat disinfection at the end of the day o Bleaching (5% chlorine) once a month.
    - ◊ Frequent bleaching is not recommended because of possible damage to the machine.

Dialysates

- Liquid bicarbonate dialysate concentrate can support rapid bacterial proliferation, and hence it not be used more than 24 hours after opening.
- Bottles containing unused dialysate should be immediately capped and the exterior of the bottle wiped over with detergent and water as part of the overall procedure of cleaning the haemodialysis machine.
- The date and time of opening should be recorded on the bottle using an indelible pen.
- Opened bottles containing unused fluid should be discarded after 24 hours.
- Unfinished bottles used for infected patients must be discarded immediately after the dialysis session

Medications

- Bags or bottles of intravenous solution should not be used as a common source of supply for multiple patients
- When multiple dose medication vials (e.g., heparin, vials containing diluents) or solution bags are used for multiple patients, individual patient doses should be prepared in a clean, centralised area away from dialysis stations and delivered separately to each patient.

Blood Borne Virus Screening and Management

- All patients should be tested for HBV, HCV and HIV on admission to the dialysis unit including after transfer from another unit
- All maintenance dialysis patients should be retested at regular every 6 months for HBV, HCV and HIV infection.
- All HBsAg-negative patients must be vaccinated against hepatitis B using approved protocol.
- Anti-HBs titers should be checked 4 weeks after the last dose and at 6 monthly intervals thereafter.
- Non-responders (anti-HBs titers < 10 IU/ml) should receive 3 more doses of the vaccine.
- All staff members should be vaccinated against hepatitis B, have their anti-HBs titer tested and be aware of their serostatus, i.e., whether or not they have titers >10 U/ml
- Testing of staff and carers for HCV or HIV is only recommended following a needle stick injury or body fluid exposure

- Patients with different blood borne virus infections should be managed separately.
- HBsAg, HBeAg and HBV DNA positive patients should be dialysed in a separate room.
- Where there are no isolation facilities, positive patients should be separated from susceptible patients (negative for HBsAg, anti-HBs, anti-HBc, anti-HCV, or anti-HIV), and undergo dialysis on dedicated machines last in the loop
- When a room/area/machine has been used for dialyzing infected patients, it should be used for uninfected patients only after cleaning and disinfection.
- Dialysis staff members caring for positive patients should not care for susceptible patients at the same time (e.g. during the same shift or during patient change-over), but may change in different shifts.
- If staff members must care for both positive and negative patients during the same shift, they must change their gown and gloves, and clean their hands in between patients.
- Close contacts of positive patients should be tested for HBsAg and anti-HBs testing and if necessary, vaccination.
- If a staff member or carer experiences a needlestick injury or exposure to blood or potentially blood-contaminated secretions from an infected patient, specialist opinion should be sought for management

Vaccinations

- All patients over 5 years old should receive pneumococcal vaccine (23vPPV).
- Hepatitis B Vaccine
- Covid-19 Vaccine

Optional

Influenza vaccine should be given annually before the beginning of the influenza season
Non-immune future transplant candidates should receive varicella vaccine.

Multi-Resistant Organism (MRO) Screening

Dialysis units should institute measures to prevent transmission of MROs. These include

- ◊ Access to good clinical microbiology laboratory to ensure prompt detection of MROs including antimicrobial susceptibility.
- ◊ Appropriate antimicrobial stewardship (optimal selection, dose, and duration of treatment)
- ◊ Active surveillance cultures (screening) to identify patients colonised or infected with MROs (quartery or as need arises) decolonisation therapy where appropriate

Preparing the Access for Cannulation

- Wash hands.
- Wash (or ask the patient to wash) the access site with antimicrobial or plain soap and water.
- Apply clean gloves.
- Cleanse the skin by applying any one of the following:
- 0.5 - 2% chlorhexidine gluconate in 70% ethyl or isopropyl alcohol
- alcoholic chlorhexidine (0.5% - 2% chlorhexidine gluconate in 70% ethyl or isopropyl alcohol 70% isopropyl alcohol using sterile swabs
- Cleanse in a circular, rubbing motion from the centre outwards, for 1 minute immediately
- prior to cannulation. Do not use a backward and forward movement.
- Wear sterile gloves for cannulation if the skin needs to be re-palpated.
- Gloves should be changed if contaminated. In patients being dialyzed using central venous catheters, topical antimicrobial ointments (e.g. povidone-iodine and 2% mupirocin) should be applied to the exit site

Staff Training

- All staff in dialysis units should be trained in infection prevention and control practices including;
  -Proper hand hygiene technique
  -Appropriate use of personal protection equipment
  -Infection Control Precautions for Dialysis Units
  -Rationale for segregating patients
  -Correct techniques for initiation, care, and maintenance of dialysis access sites.
- New and inexperienced staff should be supervised until they are considered competent to practice safely on their own.

Surveillance

- All units should develop methods to monitor, review and evaluate all infection data including
  -Rates of infection with blood borne viruses and bacterial infections overall and individually
  -Results of serological testing for blood borne viruses.
  -They should calculate incidence and conversion rates for blood borne viruses.
- Unit in charge should regularly review adherence to infection control practices annually and more frequently if there is significant staff turnover.

EMERGENCY SERVICES

Rationale: The process of hemodialysis is akin to a major surgical operation. At any given time, a fixed amount of blood is in the extracorporeal circuit which is not under the physiological control and the normal feedback mechanisms of the patient. Although most dialysis machines are equipped with a fail safe mode, a self-test, alarms and a safety profile of less than 1 event per 100 million treatments, emergencies related to personal error, and patients intrinsic condition ranging from minor discomfort to cardiac arrests on hemodialysis have been reported in dialysis units. The following guideline elaborates the personnel protocols and equipment required for managing emergencies in the dialysis unit

Description

Hemodialysis unit may be located in the premises of a hospital or it may be a standalone HD unit. In either case emergency equipment, personnel and medicines are to be kept ready in the unit for urgent use before the patient is shifted to ICU.

The common haemodialysis emergencies are:

- Hypotension
- Dialyzer reactions
  -Type A (anaphylactic reaction)
  -Type B (non-specific reaction)
-
- Haemolysis
- Air embolism
- Disequilibrium syndrome
- Chest pain, MI
- Arrhythmias
- Sudden cardiac arrest
- Hyperkalemia

All clinical and nursing staff should be trained in handling emergencies.

Equipment required to prevent and treat these emergencies

- Accurate weighing scale to exactly measure weight.
- Dialysis machines with ultrafiltration controller and sodium modelling to prevent hypotension. Spare HD machine is advisable
- Micro-haematocrit tube for manual measurement
- Activated clotting time machine.
- Glucometer.
- Multichannel cardiac monitor, Signal-averaged ECG (SAECG) and defibrillator.
- Laryngoscopes, Endotracheal tubes, Suction apparatus or wall mounted suction, Central oxygen supply & suction tubes, mouth gag and Ambu Bag
- Ryles tube.
- Arterial blood gas analysis machine.
- 24-hour emergency power generator to ensure uninterrupted power supply.

Equipment not required for an emergency, but useful in preventing an emergency

Ambulatory blood pressure monitor.
Portable ultrasound for abdominal emergencies.
Hand held doppler device for vascular access assessment.

These are optional equipments and may be made available depending on the size of the unit.

- Medicines to be available for emergency use
- Ionotropes: Injections: Dopamine, Dobutamine, Nor-adrenaline, vasopressin
- Solutions: 25% dextrose; 3% saline; 5% dextrose
- Injection Protamine
- Injections: Lignocaine, amiodarone
- Injection Hydrocortisone
- Dexamethasone
- Injection Adrenaline
- Injection Atropine
- Injection and tablet Pheniramine maleate
- Capsule and tablet Nifedepine
- Tablets: Clonidine, paracetamol, sorbitrate
- Injection Nitroglycerine
- Injections: Ondansetron, metoclopramide, pantoprazole, ranitidine
- Injection vitamin K
- Anti-convulsants Midazolam, Dilantin
- Salbutamol

All medicines are to be stacked in Crash Carts in adequate quantities depending on patient load. Expiry dates of medicines to be verified periodically.

Stocks are to be verified every morning and replaced.

An Intensive Care and a Respiratory Care Unit are to be within the reach so that a critically ill patient may be shifted there, without delay.

LABORATORY AND IMAGING BACKUP

Assessment of adequacy of dialysis, nutritional status, bone mineral disorders, anemia and monitoring for infections all require frequent laboratory investigations. Since various biochemical and serological parameters are dependent on the methodology used and the standardization and calibration of equipment widespread inter laboratory variation may be observed. It is therefore necessary for a unit performing hemodialysis to have access to a laboratory with reliable and reproducible results and to establish protocols of investigations for patients dialyzing with them. Some of the basic protocols and requirements of the laboratory for a hemodialysis unit are laid out below.

Investigations recommended for patients with acute kidney injury who are to be initiated on short-term haemodialysis

Full blood count

Blood urea

Serum creatinine

Serum potassium

Serum sodium

Hepatitis B surface antigen

Hepatitis C antibodies

HIV test

Urine analysis

Renal USS

CXR

ECHO

ECG

Other tests will depend on the clinical presentation of the patient

Investigations recommended for patients on maintenance haemodialysis:

Patients who are stable on maintenance haemodialysis may not require frequent laboratory monitoring. The following tests should be done at least once every 3 months (may be done more frequent if clinically indicated)

Blood urea

Serum creatinine

Serum sodium

Serum potassium

Kt/v

Full blood count

Serum calcium

Serum phosphate

Parathyroid hormone

Liver function tests

Iron studies

ECG

Renal USS

Cardiac ECHO

HIV test must be done before initiation of maintenance haemodialysis

Hepatitis B surface antigen, Hepatitis C antibodies must be checked before initiation of maintenance haemodialysis and thereafter every 6 months.

Staffing of chronic haemodialysis units

Medical staff should be readily available to attend to any emergencies. The overall care of patients on dialysis should be under the supervision of nephrologists.

Other staff: The staff to patient ratio for chronic dialysis should be 1:2 (including nurses and clinical technologists). A registered nurse with experience in haemodialysis should be present in the dialysis unit at all times.

We recommend that in each and every heamodialysis unit a specialized nurse and clinical technologists should be allocated to the department

Renal Transplant
Ensure that exit strategy for patients who are on dialysis and kidney transplant is the viable option.

We recommend that 5 patients accepted in the renal replacement programme go for renal transplant every year

Ensure that medications are available and affordable post transplant

Ensure that there is a programme where either patient are sent outside for transplant or the country should

develop a renal transplant centre.

Establishment of a laboratory services to aid tissue typing, immunosuppression drug levels and other relevant tests.

Ensure that anti rejection drugs are available

There should be a national waiting list for patients who need transplant.

Work up for kidney transplant for recipient and donor

Recepient

Full blood count, U &Es, Hep B and C, LFTs, Blood group, HIV

Imaging, Chest Xray, ECHO, ECG, Abdominal USS

Tissue typing- HLA type, T-Cell cross match

BMI

Active Malignancy

Donor

Age – above 18 yrs

Same as recipient

Factors that can make one disqualify as a recipient or donor

Recipient

Ejection fraction of less that 40%

Active Malignancy

Active infection such as TB, Malaria

Severe arthrescerosis

Mental disorders and substance abuse

Morbid obesity

Non compliance to medication

Donor

Evidence of renal disease, Hypertension, Diabetes

Same as recipient

All patients who have undergone kidney transplant should be monitored locally.

Follow up care for kidney donors annually

Lobby for availability of antirejection drugs

Strengthen post transplant care

Set up monthly clinics

For drug level monitoring

Monitor patient creatinine, blood sugar, blood pressure To monitor early signs of kidney rejection

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APPENDICES

Appendix 1: Leaflets on Kidney Disease

What are kidneys?

Kidneys are two bean shaped organs and reddish/brown in colour

Rarely people are born with one kidney

Lies behind posterior abdominal wall, in your lower back

Right kidney is slightly lower than the left owing to the presence of the liver on that side

Equivalent to size of a fist

Weighs about 150g

Each kidney is connected to your bladder by ureters

Functions of Kidneys

Major excretory organ

Removes body waste products (urea, phosphates, organic acids)

Removes excess water which forms urine

Regulates fluids and electrolytes

Controls blood pressure

4. Secretes erythropoietin to produce red blood cells

Produces Vitamin D for calcium absorption

Bean shaped organs

Position of kidneys,
Nurse in Male medical ward

Impsyo ndi chani?

Impsyo ndi ziwalo zimene zimakhala ziwiri, mmaonekedwe onga nyemba, mtundu wake wofiirira
Nthawi zina munthu amatha kubadwa ndi impsyo imodzi

zimapezeka kumbuyo munsi mwa msana, munsi mwa nthiti zathu

impsyo ya kumanja ili munsi kusiyana ndiya kumazere chifukwa cha kupezeka kwa chiwindi mbali imeneyo ndiyofanana ndi kukula kwa chibakera chathu

Imalemera pafupifupi 150g

Impsyo iliyonse imalumikizidwa ndi chikhodzodzo ndi tinjira todutsa mkodzo

Ntchito za impsyo

Ndi Sefa yayikulu ya thupi lathu

Imachotsa nyasi kapena zoyipa za thupi

Zimatchotsa madzi osafunikira a mthupi kupanga mkodzo

Imayika madzi ndi timichere ta mthupi mumlingo woyenera

Impsyo zimathandiza kathamangidwe kamagazi kuti kakhale mu mlingo woyenerera

Zimapanga timichere tothandiza kupanga magazi (erythropoietin) kuti akhale okwanira mthupi
Zimathandiza thupi kuti tikhale ndi mchere wa calcium wolimbitsa mafupa

Appendix 2: Acute Kidney Injury

AKI is defined as an abrupt reduction in kidney function occurs over a few hours or days and it is reversible.

Risk Factors

Fluid loss i.e. severe dehydration, severe burns

Diseases such as liver failure, heart disease, malaria, anaemia

Pregnancy complications such as abortion, Post-Partum Heamorrhage (PPH), Pre- eclampsia
Severe bleeding from trauma

Medications such as water pills, Bp pills, brufen, Asa, Bumalo, indocid and antibiotic

Herbs such as local or Chinese herbs.

Sepsis/Infections such as HIV/AIDS, glomerulonephritis

Alcohol intoxication

Drug and substance abuse such as cocaine, marijuana

Cancers such as cervical, bladder, prostate

Conditions that block urine flow such as kidney stones, enlarged prostate, urethral strictures, bladder cancer, and prostate cancer.
Clinical presentation of AKI
Acute changes in urinary habits

Decreased urinary output although occasionally urine output remains normal

.

Unexplained weight gain

Flank pain

Oedema change in urine colour foamy urine
Nausea, vomiting

Weakness

Drowsiness

Confusion, at times seizure

Chest pains

Shortness of breath

White crystals of salt on the Skin

Unexplained bleeding

Management of Acute Kidney Injury

Manage underlying factors such as infections, heart failure, high blood pressure, Malaria,

HIV/AIDs

Urine dipstick

Full blood counts, urea, Creatinine and Electrolytes

Ultrasound scan to check for urinary bladder.

If bladder is distended a catheter may be inserted

Stop medications that may damage kidneys (Gentamycin, brufen, indocid, diclofenac and others)
Medical nutritional therapy

Adequate nutritional support is important

Optimal protein intake

If potassium is high try to eat foods such as apples, tangerines, green beans, cabbage, cucumbers, mgaiwa porridge, tea, use little sugar, apple juice, cranberry juice. Strawberry mahewu, vegetables
Prevention of Acute Kidney Injury
Minimize damage to the kidneys i.e. avoid use of herbal medicine

Pay attention to labels, avoid over the counter medications such as bumulo

Increase water intake to improve hydration

If diabetic or/and hypertensive, follow doctors' recommendations

Make a healthy life style a priority. Be active, eat balanced diet

Appendix 3: Kuonongeka Kwa Impsyo Mwakanthawi Kochepa

Kuonongeka kwa impsyo mwakanthawi kochepa kochitika mmaola kapena mmasiku ochepa pamene zimalephera kusefa zinyalala m'magazi athu.

Zoyambitsa

Kutha madzi mthupi/ kupsya kwambiri.

Matenda onga okhudza chiwindi, mtima, malungo ndi ena otero.

Mavuto odza chifukwa cha uchembere monga kuchoka mimba, kutaya magazi kwambiri nthawi pobereka/ kuthamanga kwa magazi.
Kutaya magazi kwambiri

Kugwiritsa ntchito mankhwala mosayenera monga awa; burufen, Asipirin, Bumulo, indocid ndi ma antibiotic.

Kugwiritsa ntchito mankhwala adzitsamba.

Matenda aakulu (sepsis), komanso HIV/EDZI, kutupa kwa mitsempha yaying'ono yamagazi ya impso

Kumwa mowa mwa uchidakwa.

Kugwiritsa ntchito mankhwala ozunguza ubongo

Khansa ya khomo la chiberekelo / khansa ya mchinkhodzodzo/ya prostate

Matenda omwe amatseka njira ya mkodzo monga miyala mu impso, mchikhodzodzo, prostate wokula
Zizindikiro
Kusintha kwa kakodzedwe

Kuchepa kwa mikodzo ngakhale nthawi zina mkodzo umachuluka mulingo

Kuwonjezekera kwa sikelo mosadziwika bwino

Kupweteka kwa msana makamaka mmbalimbali

Kutupa kwa miyendo ndi mapazi

Mkodzo wamaokedwe athovu komanso nthawi zina ofiira

Nseru mosalekeza ndi kusanza.

Kufooka.

Kumva tulo

Mutu umasokonekera pena kukomoka kumene.

Kupweteka kapena kupanikizika mchifuwa.

Kupuma movutikira

Thandizo La Matenda A Impsyo
Thandizo limatengera chomwe chayambitsa vutoli

Madotolo amaunika chikhodzodzo

Ngati chikhodzodzo ndichozaza amalowetsa ka paipi kuchotsa mkodzo

Pewani kumwa makhwala omwe angathe kuwononga impsyo monga bumulo, brufen, kubayitsa majekitseni osadziwika ngati simunalembeledwe ndi adokotala.
Pewani makhwala azitsamba

Pezani thandizo mwachangu pa matenda omwe angapangitse vuto la impsyo kupitirira monga a shuga, a mtima, kuthamanga kwa magazi, malungo, ndi matenda ena
Zakudya zovomerezeka ndi a zaumoyo

Zakudya zakasinthasintha ndi zofunika kwambiri

Tidye zakudya zomanga thupi monga mazira, nyama yochokera ku mbalame monga nkhuku, zinziri, nkhunda Idyani zipatso monga apozi, manachesi, zikhakha, zakudya monga zitheba, kabichi, phala la mgaiwa, tiyi wothira sugar pang'ono, mahewu a strawberry, ndiwo za masamba, juwisi wa apozi, wa cranberry.

Kapewedwe Kake

Tichepetse mkhalidwe omwe ungapangitse vuto la impsyo kupitirira monga kugwiritsa ntchito mankhwala azitsamba
Tisagule ndi kumwa makhwala mwachisawawa

Kupanga masewero olimbitsa thupi

Kudya zakudya zakasinthasintha.

Appendix 4: Chronic Kidney Disease

Chronic kidney disease is an irreversible or permanent loss of kidney function, damage occurs slowly over a period of time three months or more

Cannot be cured or reversed

Categorized into five stages (one to five)

Risk of developing Chronic kidney disease

Family History of the following diseases

Diabetes

High blood pressure.

Heart diseases

Human immunodeficiency virus (HIV) or hepatitis C

Malignancy

Autoimmune diseases

Recurrent urinary tract infection

Renal diseases/ kidney disease

Smoking:

Obesity (excessive weight gain)

Abnormal kidney structure

Old age (above 60)

Kidney failure that occurs within a short period of time.

Low birth weight.

KEY POINT being given all those things that put a person at risk of developing chronic kidney disease, in most cases are mainly caused by hypertension and diabetes in developing countries

SIGNS AND SYMPTOMS OF CHRONIC KIDNEY DISEASE

Usually silent in its early stages

Weakness and tiring easily as a result of impurities in blood

Trouble sleeping due to presence of toxins in blood.

Dry and persistent itchy skin due to increased wastes in the body

Puffiness around eyes due to leakage of large amount of proteins in urine usually exacerbated in the morning and less during the day.
Swollen ankles and feet due to water retention

Blood in the urine as a result of damaged cells that leak out.

Foamy urine due to the presence of protein in urine.

Increased or decreased need to urinate

Muscle cramps

Headache

Nausea and vomiting

Loss of appetite

Convulsions (seizures)

Loss of interest

Depression

Confusion

Sudden sharp pains in back or side

High blood pressure

Weight loss

Irregular heart beats

Protein in urine

Reduced libido

Chest pain, if fluid builds up around the lining of the heart

Shortness of breath if fluid builds up in the lungs

Foamy urine

Paleness from anemia due to lack of erythropoietin which produces blood

Management of Chronic Kidney Disease

Early on CKD has less symptoms hence there's need to maintain a healthy lifestyle by eating a healthy diet (six food groups), promote physical exercises, no smoking.

Adequate control of blood pressure (BP)

Follow all treatment protocols prescribed by your health worker

Tell your health worker all medicines and herbal medicines that you are taking

Reduce dietary salt intake

Adequate blood sugar control

Have annual blood sugar checks if you have been found to have sugar disease

Follow all treatment protocols prescribed by your health worker

Avoid fizzy drinks

Your health worker may prescribe medications to reduce CKD symptom burden such as:

Erythropoetin to manage anaemia

Calcium to keep bones strong

Statins to reduce fats

Water pills to reduce oedema

Dietary Management of Chronic Kidney Disease

If you have kidney disease, you may need to control the following;

Potassium; found in fruits, vegetables, dairy

Phosphorus; found in processed/packaged foods, dairy, beans, whole grains

Protein; found in meat, meat substitutes, beans, dairy

Sodium; found in table salt, processed/packaged foods, snack foods, sauces, condiments

Fluid; water and fluid found in drinks and other foods

FOOD GROUP CHOICES AVOID

Staples 1 fist size mtanda of nsima

½ cup cooked rice 2 slices white bread 1 small muffin Potatoes*

*Potatoes: Peel, cut small pieces, soak in large amount of water for 8 hours, change water and boil

Animal sources Lean meat:

Pork Lamb
Chicken (small e.g., drumstick)

egg or 2 egg whites Boiled fresh fish Salted dried fish*
*Wash dried salted fish in warm water before cooking

Legumes ½ cup cooked beans*

*Dried beans; soak the dry beans in large amount of water for 8 hours, change water and boil

Noodles Packaged meals Chips
Plantains (green bananas) Pizza
Bran cereals Cereals with nuts Nut bread
Salted crackers Sausage
Hot dogs

Bacon Pizza
Canned meat Offal
Meat tenderizer Canned fish

Nuts

Fruits 1 small apple or ½ cup apple juice

medium slices pineapple or
½ cup pineapple juice,

5 medium slices of peeled

Bananas Guava, avocado Mango,
Oranges/orange juice

FOOD GROUP CHOICES AVOID

cucumber (nkhaka) 1 small lemon
1 small wedge water melon 1 small tangerine
15 small grapes

½ cup grape juice

Vegetables 2 palm full leafy vegetables*

½ cup cooked green beans (zitheba)

½ cup green peas, eggplant

small Onion, green pepper, carrot, vinegar, ginger, garlic

*Soak leafy vegetable in large amount of water for 2 hours, change water, boil and discard the broth

Fats and Oils 1 teaspoon margarine 1 teaspoon cooking oil
teaspoons mayonnaise

Papaya

Tamarind (bwemba) Orange

Tomato Tomato sauce Ketchup Mushrooms

Nuts

Salty salad dressings Bacon fat
Salted butter

Milk ½ cup milk

½ cup yogurt

½ cup chambiko

Chocolate milk

Fluids water according to urine output

Lemonade

Coconut water Cola beverages

FOOD GROUP	CHOICES	AVOID
	1 small cup coffee, tea	
Miscellaneous	2 cups popcorn (unsalted) 1 teaspoon sugar 1 small doughnuts 1 small samoosa	Cola soft drinks Chocolate Crisps Kamba puffs

PREVENTION OF CHRONIC KIDNEY DISEASE
Perform exercises regularly at least 30 minutes per day.

Reduce excessive weight if obese

Reduce alcohol intake

Take diet low in salt (at least less than a teaspoon per day)

Take a lot fruits and vegetables

Prevent high protein intake

Control high blood pressure

Patients with diabetes should stay in target blood sugar levels. Follow diet and adhere to medications
Take a lot of water at least 2Litres per day (weight times 30mls in an hour)

Stop smoking

Avoid traditional herbs and abuse of drugs

Go for screening at least once a year to assess if at risk or have kidney health problems

Guide to keep patient's daily phosphorus intake between 800 – 1200mg.

LOW PHOSPHORUS FOODS	Mg	MEDIUM PHOSPHORUS FOODS	Mg	HIGH PHOSPHORUS FOODS	Mg
Fruits All fruits ½ cup	40	Orange Tang 1 cup	150		
Drinks Tea 1 cup (250ml) Soda pops 360ml	5 50				
Dairy Products	30	Ice cream ½ cup (125ml)	80	Buttermilk 1 cup	220
		Frozen yoghurt ½ cup	100	Cocoa 1 cup	270
Cream cheese 30g		(125ml)		Whole milk 1 cup	225
(1oz)				Yoghurt fruit	
				flavoured 1 cup	270
				Chocolate milk 1 cup	255
Bread and Cereals	10	Oatmeal 1 cup	160	All bran 1 oz	245
Crackers (2)					
Cream of rice 1 cup	65	Waffles, homemade 1	130	Waffles, from mix 1	255
Cream of wheat 1 C	40				
Puffed rice 1 cup	30				
Pasta ½ cup	45				
White bread 1 slice	20				
Whole grain bread 1	50				
slice					
Snacks and	65	Custard ½ cup	150	Nuts ½ cup	260
Desserts					
Cake 1 slice	60				
Chocolates 1 oz	10	Peanut butter 2 teaspoon	120		
(30g)	40	Pudding ½ cup	120		
Cookies 1					
Popcorn 1 cup					
Vegetables		Lentils ½ cup	130		
		Mushroom ½ cup	120		
		Green peas ½ cup	75		
Meat, Fish and	60	Chicken 30g (1 oz)	70	Pizza 1 slice	220

Poultry					
Beef, Ham, Lamb					
1oz		Egg 1	100		
White turkey 1oz	55	Beef 30g (1oz)	155		
White fish 1oz	60				

Appendix 5: Matenda a Impsyo Okhalisa

Matenda a impsyo okhalitsa ndi pamene impsyo zawonongekeratu kUti sizingabwereretso kapena zasiyiratu kugwira ntchito zomwe zimachitika pang'onopang'ono kwa nthawi yayitali kapena kupotsera miyezi itatu
Palibe thandizo lochiritsiratu impsyo zowonongekeratu kapena kubwezeretsedwa kagwiridwe ka ntchito zake
Kuwonongeka kosathaku kuli mu magawo asanu (Gawo 1 -5)

Zinthu zimene zingamuike munthu pa chiopyezo cha kuonongeka kwa impsyo kwa nthawi yaitali.

Anthu amene alinawo kapena ku mtundu kwawo kuli matenda monga awa:

Nthenda ya Shuga,

Kuthamanga kwa magazi (Bp)

Nthenda ya mtima

Matenda a HIV kapena Edzi kapenanso kuonongeka kwa Chiwindi

Matenda a Khansa

Kusokonekera kwa chitetezo chamuthupi

Kudwala pafupipafupi matenda okhudza chikhodzodzo

Kuonongeka kwa impsyo

Zinthu zina ndi monga izi;

Kusuta fodya

Kunenepa kupyola muyezo

Amene anabadwa ndi chilema cha impsyo

Anthu amene adutsa zaka 60 zakubadwa.

Kuonongeka kwa ipsyo kwa kanthawi kochepa.

Amene anabadwa ndi sikelo yochepa

Chidziwitso

Mwa zinthu zonse zafotokozedwazi, odwala ambiri amene amapezeka ndi matendawa chimayambitsa ndi nthenda ya shunga ndi kunthamanga kwa magazi makamaka ku maiko oti angoyamba kutukuka kumene monga kwathu kuno

Zizindikiro za matenda a impsyo okhalitsa
Koyambirira munthu sawonetsa zizindikiro zili zonse

Pakuntha kwa nthawi munthu atha kuyamba kuwonetsa zizindikiro monga;

Kufooka ndi kutopa kosadziwika pafupipafupi chifukwa cha nyasi zomwe zili mthupi
Kukanika kugona kapena kusowa tulo

Khungu limakhakhala komanso kuyabwa

Kutupa kwa khope makamaka mmamawa, masana kumapwera

Kutupa mapazi chifukwa impsyo zikulephera kuchotsa madzi osafunikira mthupi mokwanira
Mu mkodzo pena mumapezeka magazi

Mkodzo pena wamawonekedwe a thovu chifukwa chakupezeka zomanga thupi

Pena kukodza pafupipafupi chifukwa chakuwonongeka kwa sefa

Kukodza mkodzo wochuluka kapena wochepa

Kukokana mmakatumba

Kupwetekeka mutu

Nseru ndi kusanza

Kusowa khumbo khumbo lakudya

Kukomoka

Kusowa chilakolako chopanga zithu

Kukhumudwa

Kusokonezeka mmutu

Kupweteka msana mbalimbali

Kuthamanga kwa magazi (BP)

Mikozo yathovu

Kutsika sikelo

Mtima kugunda mosalongosoka

Kusowa chilakolako m'banja

Kupweteka pa mthiti (muchifuwa) chifukwa cha madzi ku mapapu

Kubanika (phuma) chifukwa cha madzi akafika mmapapu

Kuchepa kwamagazi chifukwa chakuperewedwa mphamvu zimene zimapanga magazi za erythropoetin
Thandizo la matenda a impyo

kukhala ndi moyo wathanzi pakuchita izi; kudya zakudya zopatsa thanzi zamagulu onse 6, kuchita masewera olimbitsa thupi, osasuta fodya.

Onesetsani kuti kuthamanga kwa magazi anu ndikosamalidwa
Satirani ndondomeko yakamwedwe ka mankhwala momwe a zaumoyo akufotokozerani
Amasukireni a zaumoyo pa mankhwala onse amene mukumwa kuphatikizirapo mankhwala a zitsamba
Onesetsani kuti shuga wamumagazi anu ali mumulingu ovomerezeka ndi a zaumoyo
Ngati mwapezeka ndi matenda a shuga, dziwani mulingo wa shuga mumagazi anu chaka chili chonse
Satirani ndondomeko yakamwedwe ka mankhwala momwe a zaumoyo akufotokozerani

Adokotala amathanso kukupatsani mankhwala othandizira kuchepetsa mavuto obwera chifukwa cha vuto la impsyo, monga:

Erythropoeitin othandizira kuperewera kwa magazi m'thupi

Calcium wothandizira mafupa anu

Mankhwala ochepetsa mafuta m'thupi

Othandiza kukodza pakakhala vuto lotupa thupi

Appendix 6: Kadyedwe Koenera Kwa Munthu amene ali ndi Matenda a Impyo

Impsyo zili ndi udindo wochotsa zinyalala ndi kuika michere mu mulingo woyenera, chotero kudya zakudya zambiri zolakwika kumatha kukulitsa vuto la impsyo. Ngati muli ndi nthenda ya impsyo, mukuyenera kusamala pa kadyedwe ka zakudya izi; Zipatso, masamba, mkaka, nyemba zowuma, ndiwo za nyama, mazila, nsomba, zakumwa zoziziritsa kukhosi (monga fanta, coca kola, frozzy, nyika)

CHAKUDYA ZAKUDYA ZOVOMELEZEKA

Zakudya zokhutitsa Mtandaumodziwa nsima monga chibangera chako

Mpunga wokwana theka la kapu wa 250 ml

Mapisi awiri a buledi

Mbatata ya kholowa*

*Sendani mbatata ya kholowa, iduleni mapisi ang'ono ang'ono, kuyiviyika m'madzi kwa maola 8, kenako tayani madzi musanayiphike

Zanyama Nyama yopanda mafuta Nkhuku mulingo wokwana chikhatho chimodzi

Chiwalo chochepa cha nkhuku monga ntchafu

Dzila limodzi

Nsomba zaziwisi zongobwatitsa

Nsomba zowuma za mchere*

*Tsukani m'madzi ofunda

Zanyemba Nyemba zokwana theka la kapu*

*Nyemba ziviyikidwe

PEWANI ZAKUDYA IZI

Zakudya zopangidwiratu ku fakitole

Chipisi

Nthochi zophika Mabisiketi

Soseji Pizza
Nyama ya m'chitini

Za mkati mwanyama iliyonse

Zofewetsa nyama Nsomba za m'chitini

Mtedza

CHAKUDYA ZAKUDYA ZOVOMELEZEKA

m'madzi kwa maola osachepela 8 ndipo tayani madziwo musanaphike

PEWANI ZAKUDYA IZI

Zipatso Apozi m'modzi kapena juwisi wa apozi wokwana theka la kapu

Mapisi awiri ocheperako a chinanazi kapena juwisi wa nananzi wokwana theka la kapu

Mapisi asanu ocheperapo a nkhaka
Ndimu limodzi locheperako Pisi imodzi yocheperako la mavwende

Nachesi imodzi
Za masamba Zitheba zokwana theka la kapu Nsawawa zowuma zokwana theka la kapu

Anyezi wam'ng'ono m'modzi, Kaloti wam'ng'ono, girini pepa wam'ng'ono, jinja, galiki

Masamba oduldula zikhatu ziwiri*

*Masamba aziviyikidwe m'madzi kwa maola osachepela awiri ndipo tayani madziwo musanaphike

Nthochi Magwafa Mapeyala Mango
Lalanje kapena juwisi wa lalanje

Papaya Bwemba

Tomato Tomato sauce Ketchup Mushrooms

CHAKUDYA ZAKUDYA ZOVOMELEZEKA

Zamafuta Sipuni imodzi yaying'ono ya margarine

Sipuni imodzi yaying'ono ya mafuna ophikira

Masipuni awiri a ang'ono a mayonizi

PEWANI ZAKUDYA IZI

Mtedza Zamchere

Mkaka

Mkaka wochuluka theka la kapu
Yogati wakwana theka la kapu Chambiko chokwana theka la kapu

Mkaka wa chokoleti

Za madzi Kapu m'modzi wa madzi Juwisi wamandimu
Kapu m'modzi wamung'ono watiyi kapena khofi

Ayisi

Madzi akokonati

Zakumwa zangati kokakola

Mahewu achokoleti

Zakudya zina Makapu awiri achimanga popusi chosathira mchere

Sipuni imodzi yaying'on ya shuga

Donasi imodzi yaying'ono Samusa m'modzi wam'ng'ono

Zakumwa zangati kokakola

Chokoleti Khirisipi Kamba

Appendix 7: Kapewedwe ka Matenda a Impsyo Okhalitsa

Tikuyenera kuchita masewera olimbitsa thupi pafupipafupi. Khalani ndi mphindi zosachepera 30 zolimbitsa thupi patsiku masiku asanu mu sabata.
Sikelo yathu ikhale mu mlingo woyenera ngati ndife wonenepa kwambiri

Amene timamwa mowa tichepetse kamwedwe kamowa komaso kusiya kumene

Tidye zakudya zamchere wocheperako

Tidye zipatso ndi ndio zamasamba zambiri

Wodwala apewe kudya zakudya zomanga thupi zambiri.

Wodwala matenda a shuga awonetsetse kuti shuga akukhala pamulingo ovomelezeka ndi achipatala. Tisatire kadyedye koyenera komanso ndondomeko ya makhwala
Timwe madzi ambiri 2 litres pa tsiku

Tipewe kusuta

Tipewe makhwala azisamba komanso kugula makhwala mwachisawawa

Tikaunikitse kuchipatala ngati tili pachiwopsezo kapena tili ndi vuto la impsyo.

Renal screening

Urine dipstick

Appendix 8: Renal diet for in- Patients

Meal	Food Item	Preparation Method	Amount
Breakfast (6 AM)	Milk	Ready	½ cup
	Sugar Margarine Peanut	Ready	1 teaspoon
	Porridge	With minimal salt	1 cup
	Bread	Ready	x2 2 slices of bread
Mid-morning (9 AM)	Egg Porridge	Boiled Cooked	x 1 1 cup
Lunch (12 PM) / Supper (6 PM)	Nsima Rice	Grandmill	1 fist (mutanda umoza)
	Beef Chicken Fresh fish	Boiled Serve chicken without skin	small drumstick size 3 match-size beef medium size fish
	Dried beans	Soak in water for 8 hours, change water and boil	½ cup
	Leafy vegetables Green beans	Soak the leafy vegetables for at least 2 hours, boil in large amount of water, discard the water and serve	½ cup for green beans 2 palms for leafy veggies
Mid-afternoon fruit (3 PM)	Apple Pineapple Cucumber NO Banana	Ready	x 1

Cooking methods

The preferred cooking methods include boiling, baking, grilling or stewing.

Boiled meat, poultry and fish

Potatoes; peel, soak for 2-4 hours, change water and boil

Dried beans; Soak the beans for 2 - 8 hours, change water and boil

Green leafy vegetables; soak for at least 2 hours, boil in large amount of water and discard the broth
Use minimal salt in all food preparation

1 cup is equivalent to250 ml

ACUTE KIDNEY INJURY (AKI) RISK ASSESSMENT
Hypovolaemia from acute blood loss, GI loss, burns, diuretics
Hypotension – systolic ≤ 100mmHg; Sepsis; Age ≥65 years, post-operative period
Nephrotoxic medicines i.e. Use of NSAIDs, aminoglycosides, radio contrast
Co-morbidities: diabetes, hypertension, CCF, COPD, CVA, TIA,

U&Es, Creat-s STAT
Urine dipstick
Strict urine output record
Strict fluid balance
Cardiac monitoring
Manage acute illness promptly

ESTABLISHED ACUTE KIDNEY INJURY
Labs: Urine, FBC, LFTs, daily U&Es
Renal scan within 24hours.
Insert urinary catheter. Monitor U/O hourly
Daily fluid balance and weight check
Minimize further kidney insults. Avoid tenofovir, NSAIDs, amphotericin, radio contrast, Gentamycin, Tazobactum, COXII, herbal remedies, traditional medicine

PULMONARY OEDEMA
Put patient in Sit up position
Give high flow oxygen by face mask
Give Furosemide 80 -

HYPOVOLAEMIA/HYPOTENSION
Restore kidney perfusion – maintain hydration and oxygenation
Correct volume depletion with IV fluids or blood based on cause
Administer bolus 250-500ml crystalloids- N/Saline.
Maximum fluids 2 Litres in 2hours. Target SBP ≥100mmHg
After each bolus check for signs of fluid overload i.e. raised JVP, positive fluid balance, peripheral and pulmonary oedema
(clinical & radiological).
If hypotension persists SBP <100mmHg +/- MAP <70mmHg
Consult seniors for review
Temporary cessation of ACEi, ARBs. Do not restart until renal function is stable
Avoid antihypertensives and diuretics if SBP
<120mmHg
If euvolaemic (absence signs of dehydration; absence of volume overload and haemodynamically stable) give maintenance fluids: previous hours urine output + 30mL/hour
Restrict fluids to a match daily output

120mg within 1sthour
Administer venous dilator
e.g. morphine 2.5 mg iv
Give Metochropromide 10mg IV
Restrict fluids.
Consider CPAP
If no response urgent dialysis

INDICATIONS FOR DIALYSIS
Hyperkalaemia (K+≥ 6.5mmol/l) refractory to drugs
Pulmonary oedema refractory to diuretics
Severe metabolic acidosis (HCO3 <15mmol/L)
Uremic encephalopathy, significant bleeding, severe nausea and vomiting). Progress AKI (Scr>3.4mg/dL +rise >1.2mg/dL in 24 hours
Anuria non-responsive to non-dialytic measures

ESTABLISHED ACUTE KIDNEY INJURY

UREMIC BLEEDING
Ecchymoses, Purpura, Epistaxis, haemoptysis,
vomiting blood, black or coffee-like stools, Bleeding from venipuncture sites
Gastrointestinal and Intracranial Bleeding might be evident

Treatment Options Include
Tranexamic acid IV 0.5-1g BD or TDS
Cryoprecipitate 10 units IV given over 30 minutes. Fresh frozen plasma and platelets
Desmopressin (DDAVP) 0.3 µg/kg to 0.4 µg/kg IV or SC as a single injection
Blood transfusion to increase haemoglobin
If epistaxis:
-Place Ice pack on the bridge of the nose,
-Cotton with adrenaline in the nostril,
-Ballooning Foleys catheter placed at the back of nostril (should not be in for >30 minutes). Prednisolone 40mg OD for 5 days
Conjugated estrogens 0.6 mg/kg intravenously over 30–40 min OD for 5 consecutive days.
Erythropoietin 40–150 U/kg intravenously/SC three times a week
Anticoagulant–free dialysis should be considered IF INITIATED
Above methods may be used alone or in combination.

HYPERKALAEMIA (POTASSIUM ≥6MMOL/L)
ECG monitoring (P-wave, QRS complex, ST segment, T-waves and rhythm). Stabilize the cell, Shift potassium and Remove K+

Stabilize the cell
Give 10ml of 10% Calcium Gluconate over 10 minutes or
Calcium Chloride 10ml over 10 minutes. If digitalis toxicity slower infusion of calcium and consider Magnessium.

Shift the potassium into the cell
Administer Soluble insulin 10 units and 50mL of 50% dextrose
same time. Give dextrose if RBS is <250mg/dl. Insulin drops K by 1mmol/L in 1 hour..
Give Sodium bicarbonate 8.4%, (50mEq) 1 ampoule slow IV push over 5 minutes – only if in acidosis.
Administer Salbutamol Albuterol 10-20mg in 4mL normal saline Nebulized over 10 minutes. Lowers K by 1mmol/L in 30minutes and maintain it for up to 2 hours. Very effective for patients with fluid overloaded.

Remove the potassium
Give Furosemide 40-80mg IV depending on hydration status.
Administer Sodium polystyrene Sulfonate (Kayexelate) 15- 30g in 15-30mL (70% sorbitol) PO or Rectally. Avoid rectal sorbitol if bowel necrosis risk

MNEMONIC - C BIG K Drop: C- Calcium gluconate, B- Beta - 2 agonist (salbutamol), IG- Insulin + Glucose, K- Kayexelate, D- Diuretics or Dialysis, ROP- Renal unit for dialysis of patient

MANAGEMENT OF COMPLICATIONS IN CHRONIC KIDNEY DISEASE

Renal Osteodystrophy/Bone Disease
Assess Calcium, Phosp, PTH
Give Calcitriol 0.25-0.50 mcg OD or Doxercalciferol 1.0 mcg OD
Phosphate binders such as Calcium acetate 1.0-1.5g OD or Calcium carbonate 1.0-1.5g OD
Vitamin D 250u daily or 50,000u weekly
Seek specialist advice if renal bone disease
suspected

Metabolic Acidosis
(venous pH < 7.3 and/or
bicarbonate < 15mmo/L)
Treat with oral alkali supplementation
Sodium bicarbonate supplements 0.5-
2.0mEq/Kg OD 1 tsp in a
glass of water
Alternatively baking soda can be used, 1 tsp (2g) (23mEq Na and HCO3) in a glass of water.

Management of Hyperlipidemia
Lower lipids if triglycerides
>200mg/dl and/or
LDL>100mg/dl.
Administer: Artovastatin 20mg nocte or Simvastatin 20mg nocte (can be increased to 80mg)

MANAGEMENT OF COMPLICATIONS IN CHRONIC KIDNEY DISEASE

ANAEMIA PROTOCOL

Assess Hb status – target Hb 9 to 12 g/dl. If Hb below target;
Normochromic normocytic anaemia is commonly seen in CKD
Add Erythropoietin (EPO) 4000IU weekly (maximum dose 12000IU per week) IV or SC until target Hb is achieved within 2 months. If Hb <8g/dl/Hct <25% give EPO 2x weekly. Monitor Blood pressure

If Hb exceeds 11g/dl bring EPO dose down to 2000units per week until stable
Recheck FBC every 4 weeks. No earlier than 2 weeks after a dose of EPO
If at target Hb and stable – NO EPO therapy required
Assess Iron status. If Ferritin < 200mcg/mL, Transferrin saturation (TSAT) <20% – STOP Iron PO and Start IV Iron 100mg every week times 10 doses (1g in 10 weeks). Dilute in 100ml of N/S run over 30minutes.
Reassess after 10 week course of IV Iron is complete.
Ferritin>800 STOP; TSAT >40% STOP
Monitor Saturation index and ferritin monthly
If still depleted, then should get weekly maintenance IRON doses 100mg every 2 weeks
Patient should not exceed 4000mg of IRON during one calendar year
IV IRON may be held if patient is receiving antibiotics
– consult nephrologist/renal doctors
Transfuse blood when necessary. Routine administration of blood should be avoided

Investigate other possible causes of anaemia such as
Infections – treat infections
Aluminium toxicity – if on aluminium containing phosphate binders discontinue therapy.
B12 and folate deficiencies – add Vit B12 injection and folic acid.
Rule out Occult blood in stools, Poor nutrition, The stop EPO start syndrome, Chronic blood loss (haemorrhoids, menorrhagia), Immunosuppressive drugs, Auto immune diseases, Active liver disease, Dysplastic bone marrow, Hemoglobinopathies, Pregnancy, Osteitis fibrosa, Myeloma, Malignancy

Hypertensive Emergencies in Kidney Disease

Life threatening elevation in BP -treatment within one hour to prevent severe organ damage and death. Systolic BP >180mmHg or diastolic BP
>120mm Hg
Symptoms may include blurred vision, confusion, decreased alertness, chest pain, headache, nausea and vomiting, numbness, reduced urine
output, SOBs, seizure, weakness of arms, leg face or other areas
On exam:- high BPs, oedema of lower legs and feet, abnormal heart sounds and fluid in lungs, changes in thinking sensation and reflexes; Eye
exam may reveal bleeding of the retina
Tests include ABG, U&Es, creatinine, kidney USS and Urine dipstick
Chest X-Ray may show congestion in the lungs and an enlarged heart

Management of patient care
Admit patient in HDU/ICU until BP is under control.
Create a calm, quiet atmosphere, conducive to ample rest
BP should be measured in both arms.
Target BP in CKD <130/80 to improve clinical outcomes
Reduce SBP to a maximum of 25% within the first hour, if clinically stable, lower BP to 160/100 – 110 mm Hg over the next 2-6 hours and then cautiously to normal over the following 24-48 hours.
Certain pharmacological therapies are renoprotective and/or cardioprotective must be considered when instituting therapy i.e. Labetolol
40-80mg/hour IV, Hydralazine 5 - 20 mg IV.
Keep patient on NPO initially, later a sodium restricted diet
Obtain accurate intake and output, along with daily weights and strict U/O
Monitor U&Es,
If BP remain uncontrolled, evaluate for separate secondary causes of HTN.

IV drugs
Labetalol 10mg-20mg IV bolus, repeated after 10 minutes. Use with caution if COPD/Asthma or bradycardia
Hydralazine 5mg IV bolus, recheck BP every 15minutes if diastolic BP
>120mmHg repeat hydralazine 5mg IV
If pulmonary oedema give loop diuretic. Furosemide 40mg - 80mg IV. Watch for volume depletion
Close monitoring of BP every 15minutes until diastolic BP<120mmHg,
Watch for side effects of medications, observe ECG for the T-wave inversions that occur with rapid BP reduction

When stable change to Oral drugs
ACE inhibitor i.e. Enalapril 5mg OD or captopril 12.5mg BD. In pregnancy, give methyldopa, if no response, add hydralazine. Don't give
ACEs/ARBs
Calcium channel blocker i.e. Nifedipine long acting 20mg BD
Thiazide diuretic – HCTZ 25mg OD. If pulmonary oedema give.
Furosemide
Beta Blocker such as carvedilol 12.5mg BD increasing to 25mg OD increase to 50mg, alternatively use Atenolol 25mg OD

Appendix 9: Management of CKD

At risk patients

Asymptomatic

Diabetics, Hypertension, Heart diseases, Autoimmune disease (i.e. SLE), Recurrent UTIs, Malignancy, Age>60 years, Previous acute renal failure, Obesity, Heavy Smoking, HIV, Family history of kidney disease

Symptomatic

Edema, nausea and vomiting, itching, SOBs, anemia, loss of appetite, decreased or increased urine output, puffiness around eyes, confusion

Urine dipstick

Check for protein and blood in urine

Biochemistry (baseline urea, creatinine, electrolytes blood)

FBC

CKD stage 1 and 2 (GFR ≥60ml/min/1.73m2): adequate control of blood pressure and blood sugars, healthy diet, exercises, no smoking, limit alcohol. Investigations; labs, blood, urine, renal scan

Proteinuria

Prot

Raised creatinine, urea and electrolytes

CKD stages 3 and 4 (GFR ≥15 to 59 ml/min/1.73m2): intensify renal diet and manage anemia, renal osteodystrophy, metabolic acidosis, hyperlipidemia, hyperkalemia, pruritis (refer to protocols). Investigations; labs, blood, urine, renal scan

Does the patient have Nephrotic syndrome?
Yes

Yes

Does the patient have High BPs?

Yes

No

Investigate other causes

CKD stage 5 (GFR<15ml/min/1.73m2): intensify renal diet and manage anemia, renal osteodystrophy, metabolic acidosis, hyperlipidemia, hyperkalemia, pruritis (refer to protocols), if no response, start renal replacement therapy. Investigations; labs, blood, urine, renal scan. Plan for AVF construction

No

Low Na diet, protein diet, furosemide, spironolactone, weight monitoring, monitoring kidney function, fluid restriction, consider administration of albumin

Start ACE inhibitors (enalapril and captopril) or ARBs (losartan).
If not controlled, add Calcium channel blockers eg nifedipine, amlodipine.
If edema present, add Diuretics (furosemide).
Mineral corticoids receptor blockers such as spironolactone (use with caution in CKD due to risk of hyperkalemia).
Beta blockers (Lobetolol)
If pregnancy induced hypertension, treat with methylodopa,

Appendix 10: Management of Comorbidities in CKD

1. Reducing risk of Cardiovascular Disease
Patients aged ≥50 years with eGFR of ≤60ml/min per 1.73m but not treated with chronic dialysis and those between 18-49 years with no other factors for CVD be treated with low to moderate dose of statin regardless of cholesterol level
Smoking cessation should be encouraged
Management of hypertension
Antiplatelet therapy using low dose Aspirin, Low molecular weight heparin
Lipid lowering using statins such as Atorvastatin
Bone and mineral management which include treatment of hypophosphatemia using phosphate binders and dietary counselling
Management of anaemia. The target hemoglobin level should be within the range of 10- 12g/dl
Diuretics may be indicated to reduce the risk for decompensation.

2. Management of Hypertension in patients with CKD
Control of hypertension to prevent CKD progression, end organ damage and CVD risk
Hospitalize until BP is under control
Create a calm, quiet atmosphere, conducive to ample rest
BP should be measured in both arms
Target BP in CKD ≤130/80 to improve clinical outcomes
Reduce SBP to a maximum of 25% within the first hour
If clinically stable, lower BP to 160/100-110mmHg over the next 2-6 hours and then cautiously to normal over the following 24-48 hours
NPO initially, later a sodium restricted diet
Obtain accurate intake and output, along with daily weights
Use ACEI or ARB if Urine albumin excretion > 30mg/24 h
If BP not being optimally controlled, add thiazide diuretic
If edema present, add CCB or beta blockers
Use CCB as second line anti-proteinuric drugs when ACEI or ARB is contraindicated
Stop potassium sparing antihypertensives

3. Management of Diabetes Mellitus

Diabetic Nephropathy presents with persistent albuminuria (>300mg/day) confirmed on at least two occasions 3-6 months apart accompanied by consistent decline in GFR and high BPs
Workup – urine for proteins, U&Es, Creatinine, Calculate GFR, renal USS for kidney size
Glycaemic monitoring and control : HbA1c should be <6.5% to <8% in diabetic nephropathy
Treat patients with Type 2 diabetes, CKD, and an eGFR ≥30 ml/min per 1.73 m2 with metformin.
Adjust the dose of metformin to be halved when the eGFR is between 30 to 45 ml/min per 1.73 m2, and for some patients when the eGFR is 45–59 ml/min per 1.73 m2 depending on condition
Monitor patients for vitamin B12 deficiency when they are treated with metformin for more than 4 years.
Oral hypoglycaemics largely cleared by kidneys such as glyburide e.g. glibenclamide should be avoided
Whereas drugs metabolized by liver or partially excreted by kidneys such as Biguanides e.g. Metformin and some dipeptidyl peptidase 4 (DPP-4) e.g. sitagliptin and sodium –glucose co- transporter -2 inhibitors require dose reduction or discontinuation especially when GFR is <30mL/min/1.73m2
Use of SGLT-2 inhibitors in those with severely increased albuminuria should be considered e.g. Canagliflozin. Limit return of glucose to the bloodstream leading to increased glucose excretion in the urine.
In patients with Type 2 diabetes and CKD who have not achieved individualized glycaemic targets despite use of metformin and SGLT2 treatment, or who are unable to use those medications, use a long-acting glucagon-like peptide – 1 Receptor Agonist (GLP-1 RA). E.g. Dulaglutide 0.75mg and 1.5mg once a week – no dose adjustment, use with GFR of <15mL/min/1.73m2. To minimize gastrointestinal side effects, start with a low dose of GLP-1 RA, and titrate up slowly.
Statins i.e. Atorvastatin 10mg OD can be used to lower cholesterol and reduce proteinuria
Decrease insulin use as much as possible – with decreased GFR insulin is not metabolized leading a risk of hypoglycaemia
Treatment can be discontinued if patient has been initiated on dialysis due severe reduction in kidney function
Control hypertension
Reduction of dietary salt intake to <5g per day
Maintaining a protein intake of 0.8 g protein/kg/day those on conservative. Patients on dialysis, and particularly peritoneal dialysis, should consume between 1.0 and 1.2 g protein/kg (weight)/d.
Restrict high phosphorus and potassium diet
Moderate-intensity physical activity for a cumulative duration of at least 150 minutes per week
Smoking cessation
Reduce excessive weight

4. Nephrotoxins and CKD

Counsel all patients with CKD to avoid nephrotoxins
Routine administration of NSAIDS in CKD is not recommended
Avoid use of NSAIDS in individuals taking ACEi and/or ARBs therapy
Counsel patients to avoid use of herbal remedies as some cause kidney abnormalities including acute tubular necrosis, interstitial nephritis, nephrolithiasis, rhabdomyolysis, hypokalaemia and fanconi syndrome
Indications for use of proton pump inhibitors e.g. omeprazole in CKD should be discussed

Most antibiotics, oral anticoagulants, Gabapentin, oral hypoglycaemics, insulin, chemotherapy, opiates require dose reduction
Use of Gadolinium-based contrast agents are contraindicated due to risk of nephrogenic systemic fibrosis

5. Dietary management
Lower dietary acid loads;
Eat more fruits and vegetable and less meat
Low sodium diet. Less than 2g/day is recommended for patients with HTN, proteinuria or fluid overload
Vitamin D supplementation
Adequate elemental calcium intake

6. Chronic Kidney Disease Associated Pruritus Possible causes:

uremic toxins, electrolyte imbalances, immune dysregulation of uremia, calcium-phosphate deposition, high phosphate levels, abnormal serum calcium, parathyroid and imbalance in opioids
Xerosis (dry skin),
Uremic frost due to severe uremia.
Other factors: heat, stress, cold, physical activity, showering and dialysis
Clinical features

Itching, worse at night
Most common affected areas: back, arms and face
Restless throughout day and night
Scratching
Non-Pharmacological Measures

Aggressive skin hydration with aqueous cream emollient two to four times daily
Apply soothing skin lotions i.e. baby oil, creams with lanolin or camphor 2 to 4 times daily.
Advise patient to apply a moisturizing, high water content gel, lotion or cream to the body right after bathing, while the skin is damp and other times to relieve itching
Patient should avoid alcohol –based creams or lotions
Advise patients to limit food high in phosphorus such as nuts, cola drinks, offals
Getting sunlight or ultraviolet from sun may lessen itching this changes the chemicals on skin
Advise patients to take morning baths to relieve itching and prevent secondary infections.
Avoid long, hot showers or hot baths as they can make skin dry
Advise patients to use soaps with natural, pure ingredients without harsh perfumes and chemicals.
For sensitive dry, itchy skin; patient can use bath products made with oatmeal
Educate patient to minimize scratching
Rule out other causes of itching in patients who are refractory to a reasonable treatments
Pharmacological Measures

Patients should use itch-relieving topical creams
Topical 0.025% to 0.03% Capsaicin /ointment can be applied
1% Pramoxine hydrochloride lotion two times daily for 4 weeks
Tacrolimus cream 0.1% or 0.03% twice daily for seven days may be used in severe cases – use with caution
Ergocalciferol
Systemic therapies: use Gabapentin 100mg alternate days or Pregabalin 25 to 75mg Nocte
Type B Ultraviolet light phototherapy (UVB) is a good option for treating uremic pruritus inhibits T-helper-1
Mast cell stabilizers e.g. ketotifen for eight weeks

Antihistamines such as Piriton when necessary
Phosphate binders such as Calcium Carbonate to take with meals
Opioid antagonists e.g. Naltrexone 50gm OD orally or Naloxone
Optimize dialysis adequacy, calcium and phosphorus levels, skin hydration and nutrition

7. MANAGEMENT OF CVA IN CKD

Reducing risk of CVA-life style modification
Smoking cessation
healthy diet
weight restriction
regular exercise should all actively encouraged

Acute blood pressure targets
The presence of CKD should not modify these targets. Target BP 130/80 mmHg Consider Lipid-lowering therapy-statins
Consider CCB i.e. Diltiazem
Careful attention to blood pressure and volume control when a patient is first about to start dialysis. Maintain hemoglobin values

High-dose aspirin therapy
This should be given if hemorrhage has been excluded,
If the patient is not eligible for thrombolysis
If there is no other contraindication present
81mg to 325mg per day
IV thrombolysis
For thrombolytic therapy such as streptokinase

Antiplatelet therapy
Antiplatelet therapy for secondary prevention is uniformly recommended
warfarin, Heparin, clopidogrel

Physiotherapy

Positioning patients
Early Mobilization
Pressure Area care
Chest physiotherapy
Range of motion

NORMAL BIOCHEMICAL RANGES FOR ADULTS AND SAMPLING RECOMMENDATIONS

Tests	Sampling recommendations
Amylase	Keep sample at room temperature
Albumin	Keep sample closed, avoid hemolysis and test samples held at room temperature within 4 hours.
Acid phosphatase	
Alkaline phosphatase	

Fasting sample preferred. Morning collection is recommended.

Fasting sample preferred. Keep in covered sample container with minimal amount of air. Test samples held at room temperature within 4 hours. Keep sample covered.

Bilirubin Total Fasting sample. Protect sample from light. Keep protected from daylight and fluorescent light with foil over tube. Avoid hemolysis. Keep sample covered.

Bilirubin Direct

Protect sample from light.

Calcium Fasting sample preferred. Morning collection is recommended. Venous stasis in sampling causes misleading results. Avoid hemolysis and prolonged use of tourniquet.

Cholesterol Fasting sample. Postural variation may be a significant factor. Emotional and physical stress may influence levels. Increases can be caused by extended application of tourniquet.

Creatinine Certain cephalosporins may cause mis-leading (high) results. Avoid hemolysis. Keep sample covered.

GGT Fasting sample preferred. Phenytoin and Phenobarbital therapy may cause increase

Glucose Sample at appropriate time for test (e.g. fasting/random etc). Separate blood cells from serum or plasma within 1 hour. Keep sample covered.

Total protein Venous stasis during venipuncture can lead to increased values. Triglycerides Fasting sample.

Urea Keep sample closed and test samples held at room temperature within 4 hours.

Uric acid Fasting sample.

ALT Avoid hemolysis. Keep sample covered and do not freeze.

AST	Avoid hemolysis. Test samples held at room temperature within 4 hours. Keep sample covered.
Potassium	Avoid hemolysis, prolonged use of tourniquet or fist clenching during venipuncture or delay in separation from cells. Keep sample covered.
Sodium	Keep in covered sample container with minimal amount of air.
Chloride	Keep in covered sample container with minimal amount of air.