# Acute Kidney Injury

## *Executive summary*

## Introduction

Acute kidney injury (AKI) is an abrupt decline in renal function usually taking place over hours to days. It is defined by a reduction in urine output and / or a laboratory diagnosis of a decline in renal function i.e. increased serum creatinine (Cr) or reduced glomerular filtration rate (GFR). Acute kidney is a significant risk factor for morbidity and mortality – a moderate elevation of serum creatinine is an independent risk factor for mortality. The causes of AKI in adults are not significantly different from the causes in children, however, different criteria are used to determine the severity of AKI in each age group. AKI may progress to chronic kidney disease(CKD) and may occur in the presence of CKD. It is important to distinguish between the two conditions as interventions and prognosis are very different. The difference is usually temporal.

### Target User

* Doctors
* Nurses

### Target area of use

* Ward
* Outpatient department

### Key areas of focus

The main focus of this guideline is to stimulate doctors to recognize and manage AKI promptly. It is necessary to follow up the patients with AKI and those at risk of AKI to reduce morbidity. Furosemide use in the management of AKI is restricted to patients with signs of fluid overload.

### Limitations

## Management of stage 3 AKI (an indication for renal replacement therapy) is not available at CSD. These patients must be referred to EFSTH. Patients at Keneba who are suspected to have AKI should be referred for adequate management.

## Common causes:

The following are common causes of AKI in this region.

* Gastroenteritis
* Sepsis
* Malaria
* Haemorrhage
* Glomerulonephritis, especially in children
* Use of nephrotoxins – NSAIDs, aminoglycosides, ACEi / ARBs, diuretics overuse, metformin, herbal medications, contrast dyes
* Heart failure
* Liver failure
* Urinary tract obstruction - stones, tumours

## Risk factors for AKI

* Patients older than 65 years of age
* CKD
* Heart failure
* Liver disease
* Diabetes
* Haematological malignancy
* History of AKI
* Neurological/cognitive impairment
* Reliance on carers for fluids

## Presenting symptoms and signs

* Reduced urine volume or absent urine
* Features suggestive of uraemia including epigastric pain, haematemesis, haematochezia, chest pain, confusion, obtundation, loss of consciousness, increased somnolence, dyspnea, abnormal bleeding and bruising, generalised body weakness
* Features of the underlying causes: fever, vomiting, diarrhoea, haematuria

## Examination findings

This includes features of AKI (oliguria or anuria) as well as those of the underlying cause or complications.

* Oliguria or anuria
* Fever or hypothermia
* Pallor
* Dehydration
* Jaundice
* Oedema

## Investigations

* Urinalysis
* Urine microscopy
* Serum electrolytes, urea, creatinine, calcium and phosphate
* FBC and blood film
* Renal ultrasound

**Diagnosis of AKI**

The diagnosis of AKI is made if any ONE of the following criteria is fulfilled:

* Increase in Cr by 26.5 µmol/l within 48 hours; or
* Increase in Cr ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
* Urine volume of ˂ 0.5 ml/kg/h for 6 hours (adults) or 8 hours (children); or
* 25% of greater fall in eGFR in children within past 7 days.

The staging of AKI severity is determined by creatinine levels in adults and by creatinine clearance in children as well as urine output in both age groups

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| **Stage** | **Children** | | **Adults** | |
| **eGFR criteria** | **Urine output criteria** | **Creatinine criteria** | **Urine output criteria** |
| 1 | eGFR decreased by 25% | < 0.5ml/kg/hr x 8 hours | 1.5 – 1.9 times base line Cr OR  Cr ≥ 26.5µmol from base line | < 0.5 ml/kg/hr x 6 hours |
| 2 | eGFR decreased by 50% | <0.5ml/kg/hr x16 hours | 2.0-2.9 times baseline Cr | <0.5 ml/kg/hr x12 hours |
| 3 | eGFR decreased by 75% OR  eGFR < 35 ml/min/1.73m2 | <0.3 ml/kg/hr x 24 hours OR anuria (≤100ml) for 12 hours | ≥ 3.0 times baseline Cr  OR  Cr ≥ 353.6µmol/L  OR  Initiation of renal replacement therapy | <0.3 ml/kg/hr x 24 hours or anuria (≤100 ml) for 12 hours |

For children, the eGFR is calculated using the Bedside Schwartz formula which can be found on this link <http://nephron.com/bedside_peds_nic.cgi>.

## Management

Management of AKI is similar for both adults and children

### Patients at risk:

Serum creatinine levels should be done at baseline and this should be repeated periodically. While on admission, creatinine levels should be done at least on a weekly basis (more often depending on the patient’s clinical status). Urine output should be monitored throughout admission.

### Conservative management:

* Treat underlying cause e.g. antibiotics for sepsis, antimalarials for malaria etc.
* Replace fluid losses (insensible water loss plus fluid loss from vomiting or diarrhea) with colloids, preferably containing saline.
* Monitor input and output. Use urethral catheter only if it will be difficult to estimate urine volume or if a urethral obstruction is suspected.
* Ensure patient is feeding well: High caloric intake with low to normal protein.
* Manage electrolyte disturbances such as hyperkalaemia and metabolic acidosis using the guidelines on the management of electrolyte disturbances.
* Note: The use of diuretics is restricted to conditions in which there is fluid overload e.g. peripheral and pulmonary oedema. They should not be used routinely.

### Indications for renal replacement therapy (referral to EFSTH):

**Biochemical**

* Refractory hyperkalaemia of ≥ 6.5 mmol/L or 6 mmol/L with ECG changes or a daily rise of 1 mmol/L/ day
* Refractory metabolic acidosis : Bicarbonate levels ≤ 12 mmol/L
* Urea level of > 25 mmol/L or increase of >10 mmol/L/day
* Stage 3 AKI (Serum creatinine ≥ 353.6 µmol/L) or daily rise of ≥ 100 mmol/L/ day

**Clinical**

* Refractory pulmonary oedema
* Uraemic gastritis with persistent symptoms
* Uraemic pericarditis
* Uraemic encephalopathy
* Bleeding diathesis

### Follow up:

* Counsel patient on the prognosis of AKI and the need for follow up care.
* Serum creatinine levels should be measured at least 90 days after recovery from AKI. Monitoring can be as frequent as monthly
* Follow up in clinic may be extended to the next 2-3 years to check for progression of the disease, even if creatinine levels are normal and stabilised.

## Key Issues for Nursing care

* Strict monitoring of patient’s input and output on the fluid balance chart with escalation of care where necessary.
* Ensure adherence to the management protocol**.**
* Restrict potassium-rich foods e.g. bananas, oranges, cooked spinach and potatoes.

## References

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