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Diagnosis and Initial Management of Acute Kidney Injury

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DEFINITIONS

Acute kidney injury (AKI) is characterised by a rapid reduction in kidney function resulting in a failure to maintain fluid, electrolyte and acid-base homoeostasis. It is a spectrum disorder which can range from a transient, small decrement in renal function to established organ failure requiring renal replacement therapy (RRT). AKI is defined as a change in baseline of serum creatinine and/or urine output by the KDIGO AKI staging guideline which is now the globally accepted classification system for AKI. Most AKI is caused by acute tubular injury (often referred to as 'acute tubular necrosis') provoked by renal ischaemia, sepsis and or nephrotoxin exposure. A multifactorial aetiology is common. AKI is distinguished from chronic kidney disease (CKD) on the basis of previous serum creatinine or estimated glomerular filtration rate (eGFR).

KDIGO AKI staging		
	Serum creatinine: change from baseline	Urine Output
1	↑≥26 μmol/L or ↑1.5-1.9 fold	<0.5mg/kg/hr for>6 hours
2	↑2.0-2.9 fold	<0.5mg/kg/hr for>12 hours
3	≥354 μmol/L , ↑≥3.0 fold or needing RRT	<0.3mg/kg/hr for>24 hr or anuria ≥12hr

SIGNIFICANCE OF AKI

Acute kidney injury occurs outside of hospital (community acquired) but is most common in hospitalised patients and is associated with a mortality ranging from 10%-80%. Patients who present with uncomplicated AKI (single organ failure), have a mortality of ≤10%. In contrast, patients presenting with multiorgan failure involving AKI have mortality rates ≥50%. AKI requiring renal replacement therapy has the highest mortality. The causes of increased mortality amongst patients with stage 1 AKI are not fully explained but likely relate to inflammatory responses in other organs (including the gut and lungs) triggered by renal ischaemia. AKI is also associated with prolonged hospital admission and a substantial proportion of AKI survivors fail to completely recover baseline kidney function. Out-of-hospital mortality rates are elevated after discharge from an AKI episode. The 2009 NCEPOD report into AKI deaths in UK hospitals found that standards of care were good in only 50% of cases.

RAPID RECOGNITION OF SYMPTOMS AND DIAGNOSIS

AKI is recognised when creatinine is found to be abnormal and can be distinguished from CKD on the basis of previous creatinine measurements available to the assessing doctor.

- Baseline kidney function should be taken from a serum creatinine measurement performed within 1 week or within 3-12 months if no more recent measurement is available.
- Where multiple recent measurements of kidney function exist, lowest value within 3-12 months can be taken as the baseline function.
- Where records of prior kidney function are not available, the assessing doctor should usually regard abnormal kidney function in an acutely unwell patient as representing AKI rather than CKD. Serum creatinine should be repeated after 24 h to diagnose evolving AKI. Where kidney function subsequently recovers, AKI can be staged retrospectively based on the highest and final creatinine measurements.
- Coexistent features such as anaemia or hypocalcaemia can arise in AKI and do not reliably predict CKD. The only feature that clearly identifies CKD is the presence of small kidneys on imaging and even in this setting, there may be ‘acute-on-chronic’ kidney injury (i.e. rapid deterioration from impaired baseline function).

AKI can either be part of a life threatening illness such as severe sepsis or may directly result in life-threatening complications, specifically **pulmonary oedema, hyperkalemia and severe metabolic acidosis**.

Accurate recording of urine output in the inpatient setting may alert healthcare workers to the risk of the presence or development of AKI.

RISK FACTORS FOR AKI

AKI most frequently develops in the setting of recognizable risk factors. Typically, AKI is multifactorial with several contributory events. The major AKI risk factors are:

Risk factors for AKI
1. Old age (>75)
2. Acute illness or acute physiological disturbance in a patient with Chronic Kidney Disease (baseline eGFR < 60 mls/min/1.73m2)
3. Sepsis
4. Heart failure
5. Liver disease
6. Peripheral vascular disease
7. Major surgery
8. Intravenous iodinated radiocontrast media exposure
9. Exposure to or inappropriate continuation of medication interfering with renal blood flow (i.e. antihypertensive agents, especially those producing RAAS blockade; diuretics)
10. Exposure to or inappropriate continuation of medication with direct renal toxicity e.g. gentamicin
11. Diabetes mellitus

EMERGENCY MANAGEMENT OF AKI

Patients who are identified as having AKI should be managed with close medical supervision, senior doctors should be involved at an early stage and appropriate location and management of the patient should be an active decision. **Criteria for referral to the renal unit are included below, but most patients can be appropriately managed by general medical teams**. Early recognition of AKI and prompt intervention to correct or ameliorate reversible risk factors are essential in all cases. Some patients, particularly those with severe AKI and oliguria will require input from the renal team and may require transfer to the renal unit.

Initial assessment

Initial assessment should establish whether the patient has life threatening features associated with AKI:

Life threatening features associated AKI
1. Shock
2. Sepsis
3. Pulmonary oedema (hypoxia and/or tachypnoea with consistent radiological appearance)
4. Hyperkalaemia (K≥6.5mmol/L)
5. Uraemic pericarditis (pericardial rub +/or consistent ECG changes)
6. Uraemic encephalopathy (flapping tremor +/or reduced conscious level)

Immediate Management for life threatening features should be initiated:

Immediate Management of AKI
1. **Senior review**
2. **Critical care referral if appropriate (severe haemodynamic/respiratory compromise)**
3. **Escalate level of monitoring and nursing (minimum 6 hourly obs if AKI stage 2 or 3)**
4. **Hypoxia: High flow oxygen**
5. **Pulmonary oedema: furosemide (50mg iv bolus)**
6. **Hypotension (SBP<100 mmHg or SBP reduced by >20mmHg relative to baseline): High flow oxygen and crystalloid or colloid fluid bolus (250-500ml) unless in pulmonary oedemaΨ**
7. **Hyperkalaemia (K≥6.5mmol/L):** I. **Dextrose insulin infusion (10 units actrapid in 50ml of 50% dextrose over 10-30 minutes into a large vein) *DO NOT REPEAT without discussion with renal team*.** II. **Calcium gluconate or calcium chloride by slow IV injection or rapid infusion into a large vein if ECG rhythm change consistent with hyperkalaemia (not tented T waves alone)**
8. **Infection (suspected or proven): Broad spectrum antibiotics**
9. **Metabolic acidosis (pH <7.3/bicarb <20mmol/L): 500ml 1.26% sodium bicarbonate iv over 1 hour unless in pulmonary oedema**

- ΨCrystalloid should not be administered to patients in pulmonary oedema. Critical care input and pressor/inotrope support is likely to be required. Caution should be used in administration of crystalloid if the patient is oliguric/anuric and has already received significant quantities of iv fluid in the preceding 48 hours due to the risk of pulmonary oedema.

Patients may require immediate transfer to intensive care areas.

Clinical and biochemical assessment

After initial management, or in the absence of life threatening features, completion of clinical and biochemical assessment should take place. This will include:

Clinical and biochemical assessment of AKI	
1	**Biochemical profile (assessment of kidney function and electrolytes)**
2	**FBC (exclude anaemia, assess inflammatory response, identify eosinophilia and check for platelet consumption)**
3	**CRP for inflammatory response**
4	**Coagulation studies for DIC**
5	**Arterial or venous blood gas for assessment of acidosis**
6	**Blood cultures and MSU if infection suspected (any of fever, elevated CRP, blood leucocytosis or leucocyturia)**
7	**Serum and urine electrophoresis or serum free light chain ratioΣ**
8	**Urine dipstick: if blood and/or protein positive check:** **a. ANA (lupus nephritis)** **b. ANCA (ANCA associated vasculitis)** **c. anti GBM (Goodpasture's disease)** **d. complement (C3+C4) (consumption in lupus and infection)€**
9	**Urine albumin to creatinine ratio (ACR) if dipstick positive for protein to quantify proteinuria**
10	**Creatinine kinase (exclude rhabdomyolysis)**
11	**ECG (potassium causes conduction defects)**
12	**CXR to (exclude pneumonia/ pulmonary oedema/haemorrhage)**
13	**Urgent (same day) renal USS to assess for hydronephrosis if obstruction clinically suspected and in all AKI stages 2 or 3 Ψ**

- Ψ Ultrasonography should also be performed without delay (usually within 48 hours) in all AKI cases were kidney function fails to improve despite initial management. In addition, ultrasound will identify renal size and other significant renal pathology (e.g. stones, complex cysts).
- Σ Multiple myeloma is the paraproteinaemic disease that most often causes AKI. Renal injury may result from hypercalcaemia associated dehydration, sepsis or direct nephrotoxicity of free light chains (cast nephropathy). There is typically minimal urine sediment (unless there is coexistent amyloidosis). Paraproteinaemic renal disease should be strongly suspected in any patient with AKI and hypercalcaemia and actively considered in patients with AKI complicated by anaemia, leucopenia or thrombocytopenia and in all patients with unexplained AKI. Conventionally, myeloma diagnosis utilizes serum and urine electrophoresis. Testing for serum free light chain ratio is appropriate in selected cases (discuss with renal team or haematology). Serum FLC testing and highly sensitive and can be performed urgently (same day result). Cast nephropathy typically occurs with serum free kappa or lambda LC concentrations >500mg/l.

- € The presence of significant isolated proteinuria or coexistent dipstick haematuria and proteinuria suggests glomerular disease. AKI may arise in patients with background glomerular disease (e.g. diabetic nephropathy) through unrelated, superimposed tubular injury (e.g. ischaemia, sepsis or nephrotoxin exposure). However, it is essential to identify patients with rapidly progressive glomerulonephritis (RPGN) as a cause of AKI in order to instigate specific treatment as quickly as possible. For this is the reason, patients with significant isolated proteinuria or coexistent dipstick haematuria and proteinuria should be *urgently tested* for autoantibodies (ANCA, anti-GBM and ANA). ANCA positive systemic vasculitis is the most common cause of RPGN. Rapid (same day) autoantibody testing is available via Clinical Immunology upon request. Albuminuria should be quantified by ACR. RPGN is often accompanied by an acute phase response (elevated CRP) in the absence of an infective cause. There may be other systemic features such as cutaneous vasculitis and pulmonary haemorrhage but RPGN may also occur in isolation.

- Patients with acute tubular injury alone (i.e. AKI resulting from ischaemia, sepsis or nephrotoxin exposure) will have minimal/no dipstick haematuria or proteinuria.

- Patients with acute tubulo-interstitial nephritis (AIN) also typically have minimal/no dipstick haematuria or proteinuria. Alternatively, sterile leucocyturia may occur. AIN has multiple causes (including drug induced) and should be considered in all patient, particularly those without significant risk factors for ischaemic or nephrotoxic acute tubular injury. Peripheral blood eosinophilia is detectable in a minority of cases.

Criteria for urgent referral to renal unit (via PICS referral and call mobile 07920846700 or BB 16157).

Some patients will require referral the renal unit. Renal medical involvement in the management of patients with AKI primarily supports those with more severe AKI in whom generic measures have failed to restore kidney function. This will include but is not exclusive to patients requiring RRT. Renal referral also facilitates diagnosis of less common but important causes of AKI where specialist investigations (including renal biopsy) are appropriate and early intervention will improve outcome (e.g. RPGN and multiple myeloma). The following people should be referred for discussion:

Criteria for urgent referral to renal unit
1. All AKI stage 3 (see table 1)
2. All AKI complicated by **a. Pulmonary oedema refractory to diuretics** **b. Hyperkalaemia (K≥6.5mmol/L) recurring after one dextrose-insulin infusion** **c. Significant metabolic acidosis (pH ≤7.2)** **d. Active urine sediment (blood +/or protein)** **e. Paraprotein or abnormal SFLC ratio**
3. All AKI resulting in eGFR <15 ml/min

Referral may not be appropriate for selected patients in whom aggressive intervention is judged to be unlikely to be of benefit based on extensive co-morbidity (e.g. in terminal illness). Treatment is usually appropriate for patients with AKI as a single organ failure and age alone **should not** be used as a criterion to limit referral or treatment.

Patients with AKI as a component of multi-organ failure will usually require referral to Critical Care which should not be delayed. The Renal Team will be able to advise but patients with significant cardiovascular and/or respiratory instability are unlikely to appropriate for transfer to the renal unit. Patients of this type are more likely to be stepped down to the renal unit after an ICU admission.

Monitoring and reassessment

All patients require close follow up and monitoring. We recommend the following:

- Rapid reassessment of response to immediate management (as above). Hypotension, pulmonary oedema, hyperkalaemia and acidosis require frequent and regular monitoring until corrected with appropriate repeated investigations e.g. serial venous or arterial blood gases (usually beginning within one hour of immediate intervention). Frequency of observations and further clinical input should be guided by hospital SEWS guidelines
- Daily biochemical profile, FBC and fluid balance assessment until AKI episode resolved
- Repeat urine dipstick with MSU and/or ACR as appropriate
- Review need for referral to nephrology on a daily basis

All AKI stage 2 with baseline eGFR <30 ml/min should be referred to the renal team via PICS if not resolving within 72 hours.

SPECIFIC ADVICE AROUND MANAGEMENT OF AKI

Fluid balance

Fluid balance is critical in the management of patients with AKI. Dehydration (total body water deficit) or intravascular volume depletion (variably associated with total body water deficit or total body water overload) can both cause and worsen kidney injury, but conversely in patient where kidney injury exists, the excretion of salt and water is impaired and fluid can accumulate dangerously resulting in rapid fluid overload, morbidity and death. It is therefore crucial that fluid state is assessed properly and repeatedly and that fluid or diuretics are correctly administered. An expansion in volume status of as little as 10% above baseline (based on body weight) is associated with increased mortality in patients with oliguric AKI.

Fluid balance assessment in AKI
1. Daily weight (serial weights are very useful in assessing fluid accumulation)
2. BP relative to baseline
3. Heart rate
4. SaO2
5. Monitoring urine output
6. Clinical examination (JVP, lung fields, peripheral oedema)
6. Daily UE
7. CXR

Conventionally, ‘pre-renal’ and ‘renal’ causes of AKI have often been considered as distinct. In practice it is more useful to consider a continuum from ‘**volume responsive AKI**’ where restoration of renal blood flow (by volume resuscitation or

blood pressure support) restores renal function through to established '**volume unresponsive AKI**' with oliguria that does not respond to further volume expansion. Patient are then at risk of pulmonary oedema and other complications from further injudicious fluid administration.

Volume depletion with hypotension should be managed **serial, rapid fluid bolus** administration (250-500ml crystalloid or colloid over ≤30 minutes) until BP recovers (SBP>100) unless the patient develops pulmonary oedema. An increase in urine output is typically the first sign of improved renal function. Larger volumes of crystalloid administered more slowly (e.g. '1 in 8') should **not** be used to correct hypotension. Rates and volumes of fluid administered to patients who are volume deplete but normotensive should be judged by an appropriately experienced clinician and reviewed regularly.

Fluid restriction should be undertaken once the patient is volume replete and oliguria is established (urine output is less than 500ml in 24 hours). Fluid restricted patients should usually receive no more than 750ml fluid per day (including oral intake, NG feed, IV fluids and IV drugs). Appropriate allowance should be made for other losses e.g. GI loss.

Assessment should then continue with

- Daily weights
- Fluid balance charts

It is important to avoid accumulating positive balance
Routine administration of diuretics to patients with AKI has not been proven improve outcome and is not recommended. Bolus furosemide may be used in an attempt to produce a diuresis in patients with pulmonary oedema. If there is no increase in urine output, additional diuretics are unlikely to be of benefit.

The administration of dopamine does not improve outcomes in AKI, may be injurious and is not recommended.

Selection of fluids

There is no ideal intravenous fluid for use in AKI. Crystalloid (saline, dextrose saline and dextrose) and simple colloid (gelofusine) are appropriate. 5% dextrose should not be used to treat hypotensive patients since it has a short intravascular half life. Albumin is not routinely recommended since it confers no additional benefit and is expensive. High MW starches can potentiate AKI and should be avoided. Packed red cells may be appropriate for anaemic patients (Hb <8g/dl) but should be administered cautiously due to the risk of hyperkalaemia, particularly in oliguric patients.

Daily sodium intake in health is between 70 and 100 mmol/day. Following acute physiological stress, sodium and water retention are a normal response. Excessive volumes of 0.9% sodium chloride (Na 154mmol/l, Cl 154mmol/l) can provoke hyperchloraemic acidosis and sodium, chloride and water overload. Meanwhile, excessive fluid replacement with 5% dextrose increases the risk of hyponatraemia. Fluid replacement prescriptions should be tailored to the patient. Potassium containing solutions (**Hartmann's and Ringer's Lactate**) are recommended for

routine post operative fluid replacement but **should be used cautiously in patients with AKI** due to their potassium content and the risk of exacerbating hyperkalaemia.

It is not usually necessary to catheterize patients during AKI management.
Bladder catheterization should ordinarily be restricted to patients with suspected urinary tract obstruction and those where catheterization is a part of standard care e.g. post operatively or in the treatment of severe sepsis.

It is not usually necessary to monitor central venous pressure during AKI management.
CVP line insertion should ordinarily be restricted to patients where it is a part of standard care e.g. post operatively or in the treatment of severe sepsis. Central venous access may also be required for patients with inadequate peripheral venous access or after discussion with renal team. Patients requiring CVP monitoring will require transfer to a level 2 or level 3 bed.

Prescribing

Drug prescribing in AKI is important as drug excretion is often impaired and drugs or their active metabolites can therefore accumulate resulting in toxicity. Some drugs directly cause renal injury and must be stopped.

Prescribing in AKI	
1	**Review all drugs & adjust doses for renal function**
2	**Treat infection urgently**
3	**Pause all anti-hypertensive medications unless BP>180/90**
4	**Pause diuretics unless volume overloaded**
5	**Avoid (pause immediately and do not start) ACEI/ARBs**
6	**Avoid (pause immediately and do not start) NSAID**
7	**Avoid gentamicin unless agreed with renal team and microbiology**
8	**Avoid metformin if eGFR <30ml/min (metformin is not nephrotoxic but can precipitate lactic acidosis)**

Radiology

Intravenous iodinated contrast is nephrotoxic, particularly in patients with impaired kidney function (risk greatest if eGFR <30ml/min). Contrast nephropathy typically occurs within 72 hours of receiving the contrast media and usually recovers over the following five days

Therefore we recommend:

Intravenous iodinated radio-contrast in AKI	
1	**Avoid IV iodinated contrast in patients with or at risk from AKI unless essential**

2	**Pause metformin in any patient with AKI requiring iodinated contrast (increased risk of lactic acidosis)**
3	**If IV contrast is required give IV Saline 1ml/kg/hr for 24h unless oliguric (commence 12h pre- contrast if possible)**

Interventional radiology

In obstructive nephropathy, early relief is important for correction and preservation of renal function. Bladder outflow obstruction is most common, particularly in elderly males. Ureteric obstruction may result from extrinsic compression (commonly in patients with malignancy and retroperitoneal nodes) or from luminal obstruction.

Dilatation of the urinary tract (i.e. hydronephrosis on ultrasound) may evolve slowly, particularly in the presence of concurrent AKI due to tubular injury. **Repeat ultrasound scanning should be considered in patients with AKI that does not recover promptly if obstruction is a clinical likelihood.**

- If hydronephrotic, catheterize and refer urgently to urology
- Nephrostomy/ies should be inserted urgently in upper tract obstruction (discuss with urology)
- Discuss with renal team as appropriate (AKI referral guidelines)
- **Relief of obstruction may lead to rapid (within the first few hours) and significant diuresis with electrolyte loss requiring regular thorough clinical assessment, frequent electrolyte measurements and appropriate fluid prescription. Discussion with the renal team may be required.**

Nutrition

Nutritional requirements of patients with AKI should be specifically considered. Many patients will be catabolic requiring additional nutritional support. Appropriate potassium and volume restriction should also be considered where appropriate. Enteral nutrition recommended for patients with AKI. If oral feeding is not possible NG feeding should be initiated

DISCHARGE AND FOLLOW UP

AKI with persistent renal dysfunction at discharge usually requires follow up.

Following an AKI episode where the patient has not already been referred to the renal team, a renal referral should be made (via PICS) before discharge in the following circumstances:

- eGFR<30ml/min
- eGFR 30-60mls/min after recovery from AKI if <65 years

The patient's GP should be informed of all patients who have had AKI.

FURTHER READING

Acute kidney injury: adding insult to injury (2009):
http://www.ncepod.org.uk/2009aki.htm

KDIGO Clinical Practice Guideline for Acute Kidney Injury:
http://www.kdigo.org/clinical_practice_guidelines/AKI.php

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Long-term Risk of Mortality and Other Adverse Outcomes After Acute Kidney Injury: A Systematic Review and Meta-analysis. Steven G. Coca, DO, Bushra Yusuf, MD, Michael G. Shlipak, MD, MPH, Amit X. Garg, MD,PhD, and Chirag R. Parikh, MD, PhD. Am J Kidney Dis. 2009 June ; 53(6): 961–973.

GLOSSARY

AKI: Acute kidney injury
KDIGO: Kidney Disease: Improving global outcomes
CKD: Chronic Kidney Disease
ACR: albumin to creatinine ratio
eGFR: estimated glomerular filtration rate
ACEI: angiotensin concerting enzyme inhibitor
ARB: angiotensin II receptor blocker

Appendix 2

Acute Kidney Injury (AKI) Care Bundle

Abnormal kidney function tests or oliguria identified. AKI distinguished from CKD based on previous serum creatinine or eGFR.

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Stage AKI 1, 2 or 3 using KIDGO definition

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Urgent clinical and laboratory assessment for life-threatening illness

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AKI with Life threatening features:
1. Severe sepsis
2. Severe pulmonary oedema (hypoxic on O2)
3. Severe hyperkalaemia (K≥6.5 + ECG change)
4. Severe metabolic acidosis (pH ≤ 7.2)

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Initiate immediate management for life threatening features

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- Treatment of severe sepsis
- Dextrose-insulin for severe hyperkalaemia (K≥6.5 + ECG change)
- IV fluid bolus (500ml colloid) if hypotensive
- IV furosemide 50mg IV bolus for pulmonary oedema
- Summon senior help

↓

Complete clinical and laboratory assessment. Identify criteria for urgent referral to Nephrology

<u>Refer urgently to Nephrology (except patients managed palliatively or admitted to Critical Care):</u>

1. AKI Stage 3
2. All AKI complicated by
   - Pulmonary oedema refractory to diuretics
   - Hyperkalaemia (K≥6.5mmol/L) recurring after dextrose-insulin infusion x1
   - Significant metabolic acidosis (pH ≤7.2)
   - Active urine sediment (blood +/or protein) or paraprotein
3. All AKI resulting in eGFR <15 ml/min

Refer to nephrology via PICS <u>and</u> call Renal Acute SpR (07920846700) if life-threatening features. Make contact immediately criteria for urgent referral identified

Acute Kidney Injury (AKI) Care Bundle

Initial investigations for AKI

- Biochemical profile; FBC ; CRP; coagulation studies; blood gas (A or V); blood cultures if septic,CK
- Urine dipstick: if blood &/or protein +ve, check ANA, ANCA, anti GBM, complement (C3+C4)
- ECG; CXR
- Urgent (same day) renal USS to assess for obstruction and identify renal size
- MSU if dipstick positive (blood, protein or leucocytes)
- ACR if dipstick positive for protein
- Light chain paraprotein screen (serum electrophoresis & free light chains) if any clinical suspicion of myeloma or amyloidosis

Fluid balance

- Check BP relative to baseline, heart rate, SaO2, weight and CXR
- Monitor urine output
- IV fluid (250-500ml crystalloid) challenge unless overloaded
- Fluid restrict once volume replete & oliguria established (Urine <500ml, >24h)
- Daily weights & fluid balance charts
- Avoid increasing positive balance

Prescribing

- Review all drugs & adjust doses for renal function
- Treat infection urgently
- Pause anti-hypertensive medications unless BP>180/90
- Pause diuretics unless volume overloaded
- Avoid ACEI/ARBs
- Avoid NSAID/metformin
- Avoid gentamicin

Radiology

- Avoid IV iodinated contrast unless essential
- If IV contrast required give IV Saline 1ml/kg/hr for 24h unless oliguric

Intervention

- If hydronephrotic, catheterize and refer urgently to urology
- Nephrostomy/ies urgently if upper tract obstruction

Monitoring and reassessment

- Daily biochemical profile, FBC and fluid balance assessment until AKI episode resolved
- Repeat urine dipstick with MSU and/or ACR as appropriate
- Review need for referral nephrology

In patient referral to nephrology if not improving (PICS referral):

- AKI stage 2 with baseline eGFR <30 ml/min

At discharge, refer for renal out-patient review (PICS referral):

- eGFR<30ml/min
- eGFR 30-60mls/min if <65 years