SUMMARY ACUTE KIDNEY INJURY GUIDELINES

Reference No:CG-T/2013/123

Acute kidney injury (AKI) is a clinical syndrome characterised by a rapid reduction in renal excretory function, and is associated with poor clinical outcomes. AKI is not a single condition, but has a variety of different causes.

Lorenzo and Meditech will automatically issue reports on all patients who sustain AKI by creatinine criteria. These reports only take account of changes in creatinine and it is up to you to consider changes in urine output. Further information in staging AKI is contained in the full guideline.

INVESTIGATIONS

History & Clinical Examination:

• Patients with AKI often present with other acute illness. AKI should be managed alongside other conditions, in particular sepsis, but it indicates that the patient is at higher risk of deterioration.

The most important factors relating to AKI that should be determined are:

- Clinical diagnosis of cause of AKI
- Assessment of volume status.

Urinalysis: main purpose is to help differentiate causes of AKI. If blood and or protein are present, consider intrinsic renal disease and send for protein creatinine ratio.

Ultrasound scan: of renal tract and bladder in selected patients, (suspicion of obstruction, pyonephrosis, or in non-resolving/persistent AKI).

Other investigations: should be guided by clinical findings for examples see full guideline

MANAGEMENT OF AKI

Treat the underlying cause of AKI and co-existing medical problems

Additionally: think **PUMP**

PERFUSION: Think volume status, withhold BP lowering agents, and consider vasopressors in shock.

UNDERLYING CAUSE: Sepsis – complete sepsis six care bundle and identify source of sepsis.

Obstruction – Catheterise if bladder outflow obstruction is suspected. Medicines -

Medicines optimisation in AKI		
Withhold	Review dose	Monitor
NSAIDs (Ibuprofen, Naproxen, Diclofenac) Bendrofluazide Spironolactone ACEI & ARB ('-prils' & '-sartans') Metformin Methotrexate	Opioids, Tramadol Benzodiazepines Gabapentin & pregabalin Loop diuretics (e.g. frusemide) Aciclovir, Gentamicin, Penicillins Teicoplanin, Trimethoprim Vancomycin Oral diabetes meds, Insulin Levatiracetam (Keppra) LMWHs (enoxyparin) & NOACs (rivaroxaban, apixaban, dabigatran)	Ciclosporin (e.g. Neoral®, Capimune®) Tacrolimus (e.g. Prograf®, Adoport®, Advagraf®) Digoxin Lithium Phenytoin Warfarin

MONITOR: Daily UEs, Volume status and fluid balance

PREVENT & TREAT COMPLICATIONS:

Complications of AKI	Management	
Hyperkalaemia	 Severe K > 6.5 or if > 5.5 with ECG changes Refer to Trust Hyperkalaemia Guidelines for detailed guidance Resistant Hyperkalaemia > 7 or ECG changes requires urgent referral for consideration of dialysis 	
Acidaemia:	 pH< 7.25 Senior Advice should be sought. Resistant metabolic acidosis due to AKI is an indication for dialysis 	
Pulmonary oedema	 Priority should be to avoid precipitating pulmonary oedema in AKI with accurate fluid assessments. Dialysis may be indicated if oliguric or diuretic resistance. 	
Uraemic encephalopathy /pericarditis	 This is a rare but serious complication. Referral to nephrology for consideration of renal replacement therapy should be considered in all cases 	
Dialysis is useful in a number of drug toxicities	Be Guided by Toxbase but early discussion is advised even without AKI	

HOW TO REFER

- Urgent referrals should be discussed with the Renal SpR on call via switchboard or Bleep 8121 (9am to 10pm Monday Friday, (9am 9pm on weekends). Out of hours the renal consultant on call can be contacted via switchboard for both RDH and QHB
- Routine C2C Referral via ExtraMed and will be picked up the next working day
- QHB inpatients, the Renal SPR covering QHB can be contacted via switch
- The AKI specialist nurses will review all appropriate AKI 2 and 3 inpatients, at RDH and QHB. They will also arrange appropriate follow-up for these patients on discharge.