

Acute Kidney Injury (AKI)

Epidemiology of acute kidney injury (AKI)

- Staging (Kidney Disease Improving Global Outcomes grouping) – uses both serum creatinine and urine output

STAGE	SERUM CREATININE	URINE OUTPUT
1	1.5–1.9 times baseline OR ≥0.3 mg/dl (≥26.5 mmol/l) increase	<0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥12 hours
3	3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dl (≥353.6 mmol/l) OR Initiation of renal replacement therapy OR, In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m ²	<0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours

- Grading of AKI directly is proportional to mortality
 - Chertow, Glenn M., et al. "Acute kidney injury, mortality, length of stay, and costs in hospitalized patients." *Journal of the American Society of Nephrology* 16.11 (2005): 3365-3370.
- Costs approximately 1.2 billion pounds per year in the UK, not including those who go on to CRF
 - http://www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO%20AKI%20Guideline.pdf

Causes of acute kidney injury (AKI)

- AKI is NOT a diagnosis – it is a reflection of an unwell patient
- Nearly always signifies a systemically unwell patient rather than a primary renal injury- the history is very important!
- Classically broken into pre-renal, renal and post-renal
 - There can often be a mixture of all aetiologies

Pre-renal causes of acute kidney injury (AKI)

- Renal blood flow is compromised causing a reduced glomerular filtration rate
- Fluid
 - Hypovolaemia (bleeding, dehydration, burns, pancreatitis)
 - Hypotension (e.g. septic shock)
 - Heart failure – low cardiac output
 - Liver cirrhosis causing low volume
- Vascular
 - Renal artery stenosis
- Impairment of renal blood flow autoregulation
 - Liver disease (hepatorenal syndrome - rare)
 - ACE inhibitors and NSAIDs

Renal causes of acute kidney injury (AKI)

- Acute tubular necrosis
 - Ischaemia
 - Hypovolaemia, CCF, renal artery stenosis, hepatorenal syndrome
 - Basically all the causes of pre-renal failure, hence why intrinsic and pre-renal disease overlap.
 - Nephrotoxic
 - Endogenous
 - Haemoglobinaemia
 - DIC and other causes of haemolysis
 - Myoglobinuria: Rhabdomyolysis (NB have low ca in these and very high cr>ur)
 - Myeloma kidney disease – light chain nephropathy and tubular cast damage
 - Tubular crystal formation
 - Exogenous
 - Imaging contrast
 - Nephrotoxic medication
 - Drugs
 - Aminoglycosides, Amphotericin
 - Contrast agents
 - NSAIDs, Platinum drugs
- Acute tubulointerstitial nephritis
 - Drugs: NSAIDs, penicillins, diuretics, antiretrovirals and many more
 - Infections: TB, legionella, leptospirosis
 - Autoimmune: Sarcoid and Sjogren's
- Acute glomerulonephritis
- Vascular
 - Thrombotic microangiopathies: Haemolytic Uraemic Syndrome, Thrombotic Thrombocytopenic Purpura, Pre-eclampsia, Malignant hypertension
 - Vasculitis

Post-renal causes of acute kidney injury (AKI)

- These are all some form of obstruction either intraluminal, intramural or extrinsic obstruction
 - Intraluminal
 - Nephrolithiasis
 - Tumours
 - Sloughed papilla (post ATN, DM, sickle, analgesic nephropathy, amyloid and acute pyelonephritis)
 - Clot retention
 - Intramural
 - Reflux
 - Adynamic urethral segments
 - Neurological disorder (MS, spinal cord injury, DM, PD, post-stroke)
 - Drugs: Anti-cholinergics and levodopa
 - Tumours
 - Strictures (worldwide post *Schistosomomosa haematobium*)
 - Extrinsic
 - Gravid uterus
 - Benign and malignant masses
 - Iatrogenic ureteric ligation
 - Benign prostatic hypertrophy/prostate cancer
 - Retroperitoneal pathology
 - Retroperitoneal fibrosis – idiopathic, post inflammatory AAA, drugs (beta blockers and ergots)

Treatment of acute kidney injury (AKI)

- Avoidance of AKI
 - Identify high risk patients: Elderly, CKD, cardiac failure, liver disease, diabetes, vascular disease, on nephrotoxic medications
 - Monitor patients appropriately: fluid balance, bloods tests
 - Maintain circulation: Hydration, resuscitation and oxygenation
 - Minimise renal insult: nephrotoxics, contrast hospital acquired infection
 - Manage acute illness
- Optimise volume status
 - BP, CRT, pulse, oedema, sats, urine output
 - Should have iv fluid protocol
 - If fluid deplete: Crystalloid (500ml) stat bolus THEN REASSESS
 - Maintenance: Hartmanns
 - Need 25-30ml/kg water, 1mmol salt and 50-100g glucose per day
 - Evidence of crystalloid or colloid
 - Volume needed to resuscitate someone is comparable between crystalloid and colloid - SAFE study (2007) showed only 1.3x more crystalloid than colloid needed
 - No evidence from RCTs that colloid is better at all and *may* cause harm. They are also more expensive.
 - NB. CRISTAL study actually found that colloids (inc HES) improved mortality at 90 days post-ITU admission relative to crystalloid. So a confusing area.
 - Saline or Hartmanns
 - Plasma-lyte better outcomes than saline
- Stop nephrotoxics
 - ACE-I, diuretics, metformin, allopurinol etc.
 - Also consider stopping antihypertensives
 - Avoid contrast if possible
- Dialysis or haemofiltration
 - Renal ward
- Further treatment
 - Treat sepsis – antibiotics should be given within 1 hour of suspicion of sepsis
 - Steroids (in AIN)
 - Cyclophosphamide (vasculitis)
 - Plasma exchange (HUS/TTP)

Complications of acute kidney injury (AKI)

- Hyperkalaemia
 - Calcium gluconate 10mls of 10% if ECG changes
 - Insulin and dextrose 10units of actarapid in 50ml of 50% dextrose over 15 minutes (if potassium >6.5 mmol/L or ECG changes)
 - Salbutamol 5mg nebulised (caution in tachycardia or heart disease)
 - Insulin and salbutamol work for roughly 4 hours or less
 - Furosemide is useful if the patient is passing good volumes of urine but ONLY IF THE PATIENT IS FLUID OVERLOADED
 - Cation exchange resins are overused – moderate effect with high rates of constipation which might paradoxically make the situation worse – rectal route is preferable if must be used.
 - Renal replacement therapy if refractory
 - Longer term review diet, avoid potassium sparing diuretics/ACE-I, ARBS, NSAIDs.
 - Be wary of the potassium load in blood transfusions
- Pulmonary oedema
 - Sit patient up
 - High-flow oxygen unless contraindicated – consider CPAP
 - Opiates e.g. IV diamorphine 1.25-2.5 mg or morphine 2.5mg-5mg as both an anxiolytic and a venodilator – don't give too often due to accumulation in renal failure
 - If haemodynamically stable give 80mg furosemide IV and consider further boluses or infusion of 10mg/hour
 - If haemodynamically stable GTN infusion titrating up from 1mg/hour as tolerated by blood pressure (systolic above 100mmHg)
 - If unstable will need transfer to high dependency setting for urgent filtering
 - If hyperkalaemic with bicarbonate <22mmol/L and not fluid overloaded then can consider 1.26% sodium bicarbonate over 1 hour (can be given via peripheral cannula but avoid in cannula that calcium gluconate was given through)
- Acidosis
 - Bicarbonate use should be reserved for hyperkalaemia pending specialist help
 - pH<7.15 will need immediate critical care input for filtration
- Uraemic encephalopathy
 - Acute setting presents as coma
 - Chronically: fatigue, weakness, anorexia, nausea, metallic taste, pruritis, impotence
 - Will need emergency renal replacement therapy in the acute setting
- Uraemic pericarditis
 - Will need RRT
- Hypertension (more of a problem in CRF – but acute renal failure can present with hypertensive emergency and needs aggressive treatment, e.g. nitroprusside, beta blockers, ACE-I slowly over 24-48h)
- Sepsis – avoid or adjust doses of nephrotoxic drugs (e.g. vanc and gent)
- Electrolyte derangement – fluid, sodium and potassium restriction, and RRT if it is still there.

Questions concerning acute kidney injury (AKI)

- A 50 year old alcoholic male presents with sepsis secondary to klebsiella pneumonia. His background includes IHD, previous pneumonia, hypercholesterolaemia and hypertension. Medications include: furosemide, enalapril, aspirin, clopidogrel, co-amoxiclav (current) and simvastatin
- He is treated with IV antibiotics and is managed on an ITU setting for 1 week
- On step down to a medical ward routine bloods reveal:
 - Sodium 132
 - Potassium 5.0
 - Urea 24 (from 8)
 - Creatinine 390 (from 60)
- Clinically he is mildly dry, with a BP 135/83, HR 90, he is catheterised with a U/O 35ml/hr
- His management plan should include which of the following?
 1. Switch to high dose IV furosemide, stop enalapril, give IV fluids to maintain urine output, daily bloods
 2. Stop furosemide, stop enalapril, add in dopamine and maintain adequate hydration to maintain urine output, daily bloods
 3. Stop furosemide, stop enalapril, adequate fluids to maintain urine output, daily bloods
 4. Continue furosemide, stop enalapril, high dose corticosteroids and continue adequate fluids to maintain urine output, daily bloods
 5. None of the above
- Answer – 3