Renal disease in acute medicine

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Management of acute renal presentations
- Stabilisation on admission
- Diagnosis in acute renal failure
- Acute on chronic renal failure
- The acutely unwell dialysis patient
- When to transfer to the renal unit

Approach to acute renal failure
- Is the patient in danger of imminent death?
- Is it pre-renal, renal or post renal?
- Is it really acute?

‘Danger’ in Renal Failure
- Hyperkalaemia
- Severe acidosis
- Pulmonary oedema
  - Clinical features from ‘uraemia per se are rarely seen in UK practice and usually indicate chronic renal disease
- Rapid arterial blood gas (or venous blood gas) is crucial
  - Also helps rule out haemolysis in lab sample

Hyperkalaemia: [K⁺] > 5.5mmol/l
- 98% body potassium intracellular
  - Maintained by 3Na/2K-ATPase
  - Passive movement to ECF responsible for ubiquitous negative resting membrane potential
- Reduction of this gradient causes decreased myocardial conduction velocity and instability
  - Exacerbated by acidosis, hypocalcaemia, hyponatraemia and hypomagnesaemia
- [K⁺] > 6.5mmol/l carries risk of life threatening arrhythmias

Hyperkalaemic ECG
- Peaked T waves
- Prolonged PR and QT intervals with widened QRS
- Flattened p waves
Treatment

- May be needed rapidly
- Three mechanisms
  - Stabilise cardiac myocyte membranes  **Seconds**
  - Drive K⁺ intracellularly  **Minutes**
  - Reduce total body K⁺  **Hours**

IV Calcium – Effective but high risk

- Infuse 10ml 10% calcium gluconate via large vein over 2-3 minutes
  - Stabilises myocardial membrane
  - ECG changes should improve within 1-3 minutes
  - Lasts 30-60 minutes
  - Can be repeated if ECG changes persist
  - Has no effect on plasma [K⁺]
- Carries significant risk of severe tissue injury if it extravasates
- Only used to buy time for additional therapy

Insulin-dextrose infusion

- Stimulates movement of K⁺ into cells
  - Insulin stimulates Na⁺/H⁺ exchange, increasing intracellular Na⁺ which drives K⁺ into cells
- Standard cocktail: 50ml 50% dextrose with 15u actrapid over 10 minutes
- Takes 15-45 minutes to reduce K⁺ by 0.5-1.5mmol/l
- Effects last 4-6h
- Risk of delayed hypoglycaemia

Intravenous Bicarbonate

- In acidosis Na⁺/H⁺ exchange impaired
  - Neutralising H⁺ with IV bicarbonate allows Na⁺ into cells allowing exchange with K⁺
- 500ml 1.26% bicarbonate over 15 minutes
  - 50ml 8.4% bicarbonate if cardiac arrest imminent
- Unlikely to be of benefit if not acidotic
- Risk of fluid overload and hypocalcaemia
- Calcium carbonate (chalk) will precipitate if bicarbonate administered via same line as calcium salts

Nebulised salbutamol

- Stimulates movement of K⁺ into cells
  - Catecholamines activate Na⁺/K⁺ ATPase directly
- 5-10mg nebulised salbutamol may reduce serum K⁺ by 0.5-1.5mmol/l
- No effect on total body K⁺
- Significant risk of tachyarrhythmia in unstable patients so NOT recommended

Reduce total body K⁺

- If UO maintained frusemide may aid K⁺ clearance
  - High doses (>250mg) needed if severe renal failure
  - Rarely effective in hyperkalaemia in ARF
- Urgent dialysis or haemofiltration needed if renal function not improving
- Little benefit of calcium resonium in the acute setting
Acidosis (pH<7.35)
- Contributes to risk of arrhythmia with hyperkalaemia
- Can also contribute to hyperkalaemia itself
- Disrupts other cellular processes
- Treatment is with bicarbonate
  - NB risk of fluid overload
- If cause of acidosis not reversed RRT will be necessary

Fluid overload
- Inability to excrete salt and water leads to increased circulating volume and pulmonary oedema and respiratory failure
- GTN infusion and CPAP often effective in preserving gas exchange in short term
  - Diuretics needed at high dose – can be effective if kidneys recovering
- If renal recovery not anticipated RRT will be needed

Diagnosis of acute renal failure
- Reduced glomerular filtration rate
  - *Estimated* by measuring serum creatinine
  - ARF: Elevation of creatinine from baseline
  - Creatinine remains ‘normal’ until GFR < 50%
  - A rise from 60 to 110μmol/l indicates a greater % change in GFR than 110 to 150μmol/l
- Acute implies *potentially reversible*
  - Usually less than 3 months
  - Often only a few days

Estimated GFR is IRRELEVANT in ARF
- Creatinine also depends on muscle mass
  - eGFR expressed as ml/min per 1.72m² BSA
  - Relies on steady state creatinine
  - If kidneys removed creatinine will continuously rise despite actual GFR = 0 ml/min
- eGFR is good for population screening for CKD
  - Corrects for ‘normal’ reduction in GFR and muscle bulk with age

Causes of acute renal failure
- Pre-renal
  - Impaired perfusion of kidneys due to general or local circulatory compromise
- Renal
  - Host of disease processes directly affecting kidneys
  - Often multisystem
- Postrenal
  - Obstruction of urinary flow

Diagnosing the cause of ARF in the MAU
- Assessment of volume and cardiac status
  - Clinical examination or CVP monitoring
  - JVP, BP, peripheries, CXR
- Urine dipstix
  - Significant proteinuria suggests glomerular disease
  - Haematuria and ARF suggests intrinsic renal disease
- Ultrasound scan of urinary tract
  - >80% negative predictive value for obstruction
  - Yields additional useful information eg size of kidneys
- Acute renal screen blood tests
  - Blood film, CK, CRP particularly useful acutely
**Frusemide in ARF**

- The higher the creatinine the bigger dose needed
  - Ototoxicity if large doses given (usually >1g/day)
- Theoretically beneficial in 3 situations:
  - If off-loading patient improves haemodynamic status and cardiac output, renal injury may be improved
  - If patient fluid overloaded may delay need for RRT usually need high doses (>250mg/4h) for this
  - Low dose infusion (5-10mg/h) may decrease renal O$_2$ consumption and have some protective effects in pre-renal failure (highly controversial)
- No evidence that frusemide cures renal failure

**Dopamine in ARF**

- No evidence that dopamine improves outcome in ARF
- May have a role as an inotrope in supporting blood pressure (especially in sepsis)
  - Usually need >5mcg/kg/min for this (ie ‘pressor’ dose not ‘renal’ dose)
  - Noradrenaline almost certainly more effective for this
- Also has some diuretic effect

**Chronic Renal Failure in MAU**

- Old blood test results most useful
- Common features suggesting undiagnosed underlying CKD:
  - PMH of renal disease, DM or HT
  - <8cm kidneys on USS
  - Anaemia (normocytic)
  - High phosphate
    - High parathyroid hormone levels

**Acute on chronic renal failure**

- Common for patients with CKD to develop severe renal impairment in context of intercurrent illness/procedures
- Baseline renal function is key piece of information
- If fluid balance/K+ /H+ manageable with medical therapy no indication for emergency RRT even if creatinine very high
- Renal input and follow-up usually helpful

**The sick dialysis patient**

- When very sick often brought to nearest hospital (not renal unit)
- **Always check the potassium immediately**
- Commonest presentations are sepsis and fluid overload (over-drinking or loss of flesh weight due to underlying subacute illness)
- NB post dialysis coagulopathy
  - Platelet function impaired by renal failure
  - Significant doses of heparin given on dialysis
  - Treatment is with FFP

**IV access in HD patients**

- Avoid tourniquets/BP cuffs on functioning fistulae
- Backs of hands and upper arms best for cannulae
- Never cannulate a foot/leg vein
- Dialysis catheter (and fistulae) can be used for central access in life-threatening emergencies
  - NB Heparin linelock
- Central access is precious – central vein stenosis is common so neck lines are contentious
  - Femoral line often easiest if central access needed
Fluid overload in a HD patient

- HD takes place on either MWF or TTS
- Most likely to present night before HD
  - Sunday and Monday nights are busiest
- SOB, pulmonary oedema and raised BP
- Oxygen and GTN often sufficient to stabilise patient enough for transfer for dialysis
  - Frusemide unlikely to be effective
- If CPAP needed ITU should be involved early
- Early renal referral usually helpful

Sepsis in a dialysis patient

- Line and chest sources most common
  - NB pyocystis in anuric patients
- Usually need aggressive intravenous antibiotic therapy
  - Immunosuppression and exposure to HD and other dialysis patients means resistant organisms more likely than in the community
  - Local prescribing probably influences local flora

Septic dialysis patient

- Be cautious with fluid resuscitation
  - 250-500ml rapid bolus followed by review
  - No maintenance fluids(!)
  - Pulmonary oedema occurs very readily in septic HD patients
- Often respond very well to inotropes – institute early
- Consider hydrocortisone if previous steroid use (eg previous transplant or immunosuppression for 1ary disease)
- Generally do better than expected in HDU/ITU

Who to transfer to renal unit

- Dialysis patients not fit for discharge
- ARF requiring urgent biopsy and/or specialist treatment
  - Eg vasculitis, HUS, RPGN
- Deteriorating renal function despite therapy
- If emergency treatment was needed and no recovery or reversible cause for ARF identified
  - Likely need for RRT
  - Most nephrologists will only be interested if the ultrasound scan result is known
- If small kidneys (ie likely/confirmed CKD) and no recovery of function may be RRT-dependent for life

When not to transfer

- Untreated/refractory hyperkalaemia or acidosis
  - Significant risk if K>5.5 following treatment for hyperkalaemia (rebound)
- Respiratory failure (ie need for high-flow oxygen)
- Haemodynamic instability
- Decision to transfer also depends on how far/quick transfer is and ITU availability

Take-home messages

- Always know the potassium (especially in HD patients)
  - Medical treatment of hyperkalaemia can only buy time for transfer or definitive treatment of ARF
  - Treated hyperkalaemia can rebound while awaiting transfer
- Do not overfill HD patients
- If in doubt, filter on ITU for 12h before transfer