Acute kidney injury

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James talks to Professor Steve Chadban about Acute kidney injury (AKI), which develops in 5 - 20% of hospitalised patients and a significant contributor to morbidity and mortality in the critically ill.

Steve Chadban studied Medicine at the University of Newcastle, Australia, where he received the University Medal in Medicine. He undertook physician training in Newcastle, before specialising in Nephrology, completing a PhD in macrophage biology at Monash University, then a post-doctoral study period in immunology at the University of Cambridge, UK. Steve returned to Monash University to run the Transplantation Program from 1999–2002 before moving to Royal Prince Alfred Hospital, where he is a full-time staff Nephrologist, Director of Kidney Transplantation, Professor of Medicine (Nephrology) at The University of Sydney and researcher in basic and clinical sciences. Steve is the President-elect of the TSANZ, Executive member of ANZDATA and a Lead Investigator in the AusDiab Kidney Study. He has spoken at numerous National and International meetings and produced over 170 papers published in leading International journals including The New England Journal of Medicine, JAMA and The Journal of Clinical Investigation. Steve's research interests include the molecular mechanisms of transplant rejection, with a focus on macrophages and innate immunity, clinical transplantation, diabetes and outcomes in CKD and transplantation.

Acute Kidney Injury

With Professor Steve Chadban, Renal Physician at Royal Prince Alfred Hospital, New South Wales, Australia

Introduction

Acute kidney injury (AKI) encompasses an entire spectrum from mild, temporary impairment to severe disease requiring renal replacement therapy. It develops in 5-20% of hospitalised patients and is a significant contributor to morbidity and mortality in the critically ill. Having an efficient and methodical approach to investigating AKI can turn a seemingly complex scenario into a fairly straight-forward problem for the junior doctor to manage. In this podcast we will deconstruct the myriad causes to outline a helpful, systemic approach to investigating and managing such patients.

Case 1 – As the junior doctor covering the orthopaedic ward on the weekend, a nurse calls you as he is worried about one of his patients, a 30 year old man who is 24 hours post intramedullary fixation of a fractured femur sustained in a high speed motorbike accident. It appears that his urine output has been trending steadily down, and he has only produced 70mLs of urine in the last 6 hours.

1. Are you concerned about this patient? What do you want to ask the nurse over the phone?

- He is oliguric, as he's making <30mLs of urine per hour
- Questions to ask the nurse
    - Is the blood pressure trending down?
    - Is the pulse trending up?
    - Is the patient febrile?
    - Is he conscious and well?
    - Is he eating & drinking?
    - Does he currently have a cannula?

2. Is there anything you should ask the nurse to do while you make your way to review the patient?

- If the patient does not have an indwelling urinary catheter in, you could ask the nurse to perform a bladder scan

3. Can you define AKI for us?

- AKI is characterised by an acute insult to the function of the kidney
- It is potentially reversible
- It can be the result of a number of varying causes
- AKI is different to chronic kidney disease (CKD), however take note that patients with CKD (10-15% of the general population) are more likely to develop AKI

4. Do you look at the creatinine or the estimated GFR?

- The rise in serum creatinine is delayed after an AKI. In an anephric patient, the serum creatinine will rise approximately 200mmol in a 24 hour period
- The serum creatinine is the key driver of the eGFR and also shows a delayed response, however eGFR is a more sensitive marker than creatinine alone
- I would say that the presence of oliguria is the most important measure

5. What are the common causes of AKI in hospitalised patients?

- Hypovolaemia
- Sepsis
- Drugs (radiocontrast or antibiotics such as gentamicin)

6. How do you classify AKI?

- Pre-renal vs. intra-renal vs. post-renal
    - This mechanism is valuable in helping to figure out what's going wrong, whilst providing a structured approach
- Start off by considering pre-renal factors such as hypovolaemia and sepsis
- Then post-renal factors can be excluded with a renal ultrasound scan
    - Renal ultrasound is the best way to exclude obstruction to a kidney (although this is less commonly a cause of AKI in a 30 year old)
    - Renal ultrasound demonstrates the size of the kidney (chronic renal disease will cause scarring and a reduced volume, inflammatory disease can cause enlargement)
- Following this, consider causes of intra-renal injury such as nephrotoxins, or an underlying kidney disorder
    - Don't forget to check the urine for red cells or white cells that can indicate bleeding or inflammation of the kidney

7. How is the severity of injury graded?

- There are multiple grading systems, including 'RIFLE'
- It is best to use a combination of oliguria and a rise in creatinine, as they're the two most readily available clinical tools
    - Note that if someone is anuric, then the creatinine level is less important, as the typical maximum rise in 24 hours is 200mmol
- The amount of rise in the creatinine level over 24 hours is also important

You arrive to the ward and note the bladder scan result of 10mLs. You notice that his routine blood tests from earlier this morning show that his creatinine had

increased to 180mmol/L.

8. What is your initial approach to this patient in regards to history and examination?

- A rise in creatinine such as this over just 6 hours suggests there is virtually zero kidney function
- Begin by thoroughly assessing possible pre-renal causes by assessing volume status, BP, pulse, JVP
- Promptly begin intravenous fluid resuscitation
- Then consider:
    - Is the patient septic?
    - Have they been treated with nephrotoxins?
    - Do they need imaging?

9. How do you differentiate between acute and chronic kidney disease when you don't have previous blood tests to compare to?

- The accompaniment of electrolyte disturbance, hyperkalaemia or acidosis suggests AKI
- Kidney size on ultrasound is preserved in AKI (no scarring or loss of volume)
- Chronic kidney disease may be accompanied by some of the typical complications, e.g. anaemia, hyperphosphataemia, hypocalcaemia, hyperparathyroidism, acidosis and hyperkalaemia

10. What investigations will you order and why?

- The urinary sediment should be tested to rule out glomerulonephritis or other internal cause – the sediment should be bland (no blood or cells in the urine) in normal patients
    - Note that the urine sediment is different to urinalysis – it involves looking under the microscope for cells and casts or other abnormalities present
- Renal ultrasound scan
- Clinical investigations looking for bleeding, hypovolaemia, etc.

11. What are the common complications of AKI?

- Hyperkalaemia, which can be life threatening
    - Measure the serum potassium urgently
    - If there is any suspicion for hyperkalaemia, record an ECG and look for peaking of T waves
- Acidosis
    - This can compromise cardiac function
- Excess volume and pulmonary oedema following vigorous intravenous fluid administration

12. What is your approach to initial management?

- Look for potential causes
- Resuscitate with intravenous fluids
- Remove any nephrotoxins and make sure to avoid further nephrotoxins
- Supportive care whilst the kidney takes time to recover (sometimes this involves dialysis)

13. What are some of the common nephrotoxic drugs that junior doctors should look out for?

- Non-steroidal anti-inflammatories (NSAIDs)
- Diuretics
- The combination of NSAIDs, diuretics +/- ACE inhibitor or angiotensin receptor blocker
- Protein pump inhibitors

14. What are the best clinical or biochemical markers used to monitor for progression of renal disease (urine output, plasma creatinine, creatinine clearance, novel renal biomarkers)?

- Creatinine or eGFR are the most sensitive measures and are the best day to day means of monitoring renal function

- Check for serum potassium and bicarbonate levels, as consequences of AKI
- Monitor urine output
- Note that as the injury recovers, the urine output typically increases first, which is then followed by a drop in the creatinine on the next day

15. Whenis it appropriate to ask for expert help? When is it appropriate to consider commencing dialysis?

- In the case of pre-renal causes, if the causes have been reversed without any resolution of the AKI, request an urgent renal consult by the end of the day
- Indications for dialysis
    - Life-threatening hyperkalaemia
    - Life-threatening volume overload
    - Life-threatening acidosis
    - Delirium, if believed to be driven by the AKI
    - In chronic kidney disease, it would usually be triggered by the development of complications, e.g. pericarditis or peripheral neuropathy
    - Note that pure rise in creatinine is not used as a trigger for commencing dialysis, as different levels are tolerated differently between patients

Take home messages

- Think of it!
- Think in terms of pre-renal, intra-renal and post-renal
- Don’t forget to look at what’s in the urine!

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- Approach to a dialysis patient
- Oliguria

Tags: #acute kidney failure,#nephrology,#renal

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