Revise nephrology MCQs 2023

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Hyponatraemia (1)

- What is the most likely cause of hyponatraemia in a 40 year old man with a long psychiatric history stable on an antipsychotic for years (more than one answer)

a. Thiazide-induced

b. Loop diuretics

c. Psychogenic polydipsia

d. SIADH

e. Diabetes insipidus

f. Salt wasting

g. ACTH deficiency

h. Nephrotic syndrome

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Hyponatremia (2)

- Most likely cause of hyponatraemia in an elderly lady with ischaemic heart disease and hypertension taking aspirin, combined anti-hypertensive and beta blocker? (More than one answer)

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Hyponatraemia (3)

- An 80 yo female is admitted to ED following a fall. She is mildly confused, afebrile, and euvolaemic. She takes sertraline 50mg daily for the past 6 months, with a dose increase 14 days ago. She takes no other medications. Other than cease her sertraline, what is the next best intervention for her electrolyte abnormality? Electrolytes shown: Na 124 ; Urea 8.4, K 3.9, Creat 89, Serum osmolality 240, urine osmolality 500.

a. IV hypertonic saline

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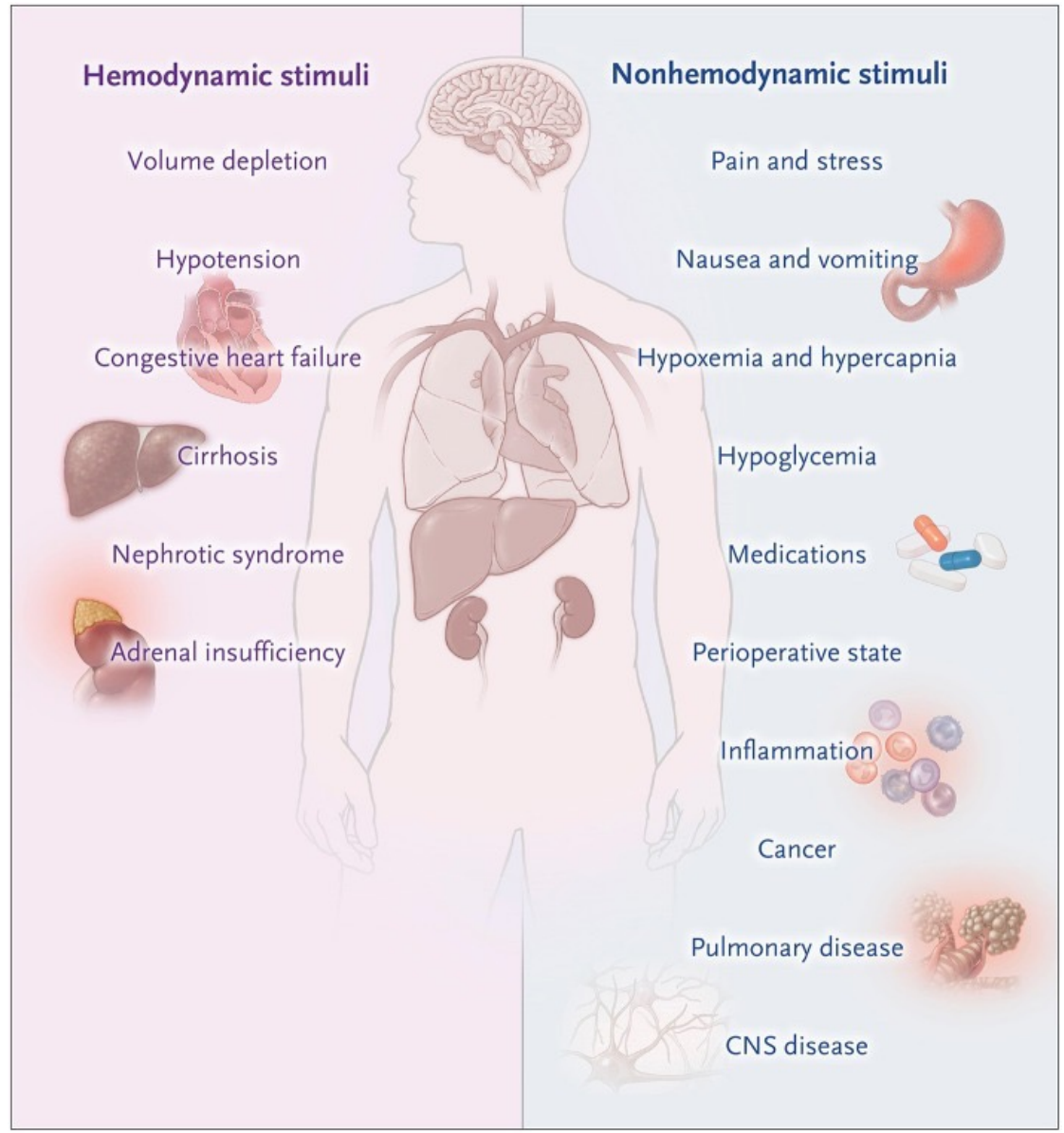
Hyponatraemia

- Patients with hyponatraemia have excess water to sodium
  - Almost all patients with hyponatraemia have high ADH
  - So in all these cases the urine osmolality is higher than the plasma osmolality – you should be getting rid of the excess water when you're hyponatremic, but for some reason you can't
  - Diagnosis of SIADH secretion dependent on you excluding all the *appropriate* causes of ADH secretion
- When is ADH appropriately present ie: haemodynamic stimuli?
  - Decreased effective circulating volume – Cardiac failure, liver failure, nephrotic syndrome
  - Actual reduced circulating volume – Hypovolemia
- When does ADH increase for non-physiological/non-haemodynamic reasons?
  - 'Stress' – Fear, pain, major trauma, surgery


Hemodynamic stimuli
Volume depletion
Hypotension
Congestive heart failure
Cirrhosis
Nephrotic syndrome
Adrenal insufficiency
Nonhemodynamic stimuli
Pain and stress
Nausea and vomiting
Hypoxemia and hypercapnia
Hypoglycemia
Medications
Perioperative state
Inflammation
Cancer
Pulmonary disease
CNS disease

How does one cause water intoxication?

- One cannot excrete free water, minimum urine osmolality is ~ 50 mmol/L
- To excrete water, need osmoles to follow it
- To overwhelm one's ability to excrete free water you need to drink ~12 to 18 litres of water per day (average dietary intake is 600 - 900 mosmoles)
- Patients with hyponatremia due to water intoxication and primary polydipsia usually have concurrent SIADH
- The 'tea and toast' patient or 'beer drinker's potomania' = low osmoles + lots of free water

SIADH

- Diagnosis of SIADH
  - Decreased plasma osmolality
  - Inappropriately concentrated urine
  - Euvolemic (patient starts off with excess water in SIADH but then through natriuresis lose sodium to become euvolemic), this is very difficult to judge when patient is slightly over or under
  - High urine sodium
  - TFT, cortisol normal
- Common causes of SIADH
  - CNS (CT brain)
  - Lung (CXR)
  - Malignancy
  - Drugs

Treatment

- Give hypertonic saline if patient has severe neurological manifestations – seizure, rapidly decreasing sodium
  - Bolus of 100 – 150 ml of 3% saline
- However a lot of fear (me included) of overshooting because of the concern of osmotic demyelination syndrome/central pontine myelinolysis

- Correcting the reason why the ADH is high in the first place
- Fluid restriction
- Giving osmoles (high protein diet, urea, salt tablets)

Thiazide induced hyponatremia

- Multifactorial
  - Increased water intake
  - Impaired water excretion
    - Urine cannot be maximally dilute because the thiazide causes salt to be lost in the urine
    - Thiazides can promote the effect of antidiuretic hormone as seen in diabetes insipidus patients
    - This is a problem if there is increased water intake *and probably not that many osmoles in the diet (elderly patients more susceptible)*

Renal physiology (1)

What is not a key component of the juxtaglomerular apparatus?

- A. Distal tubule cells
- B. Afferent arteriole
- C. Efferent arteriole
- D. Extraglomerular mesangial cells

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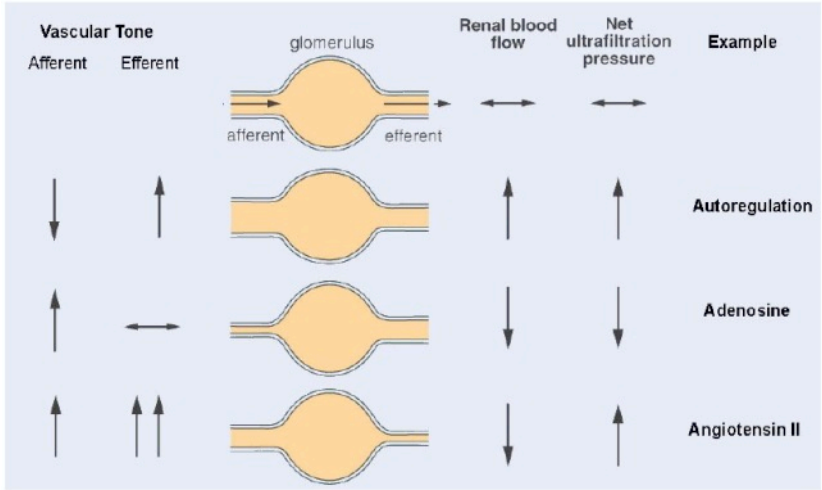
Juxtaglomerular apparatus

- Macula densa cells at the distal tubule detect sodium in the filtrate
  - Reduced salt delivery (proximal tubules and loop of Henle has retained a lot of salt)
    - MD cells produce PGE2, JG cells produce renin → angiotensin 2 → constriction of efferent arteriole >> afferent arteriole
  - Increased salt delivery (pressure natriuresis + proximal tubules are damaged)
    - Macula densa cells produce adenosine which acts on the afferent arteriole to vasoconstrict
- Extraglomerular mesangial cells not the same as juxtaglomerular cells but sandwiched between the afferent and macula densa



Renal physiology (2)

- What is the site of action of thiazide diuretics?
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- B. Distal tubule
- C. Cortical collecting duct
- D. Proximal tubule

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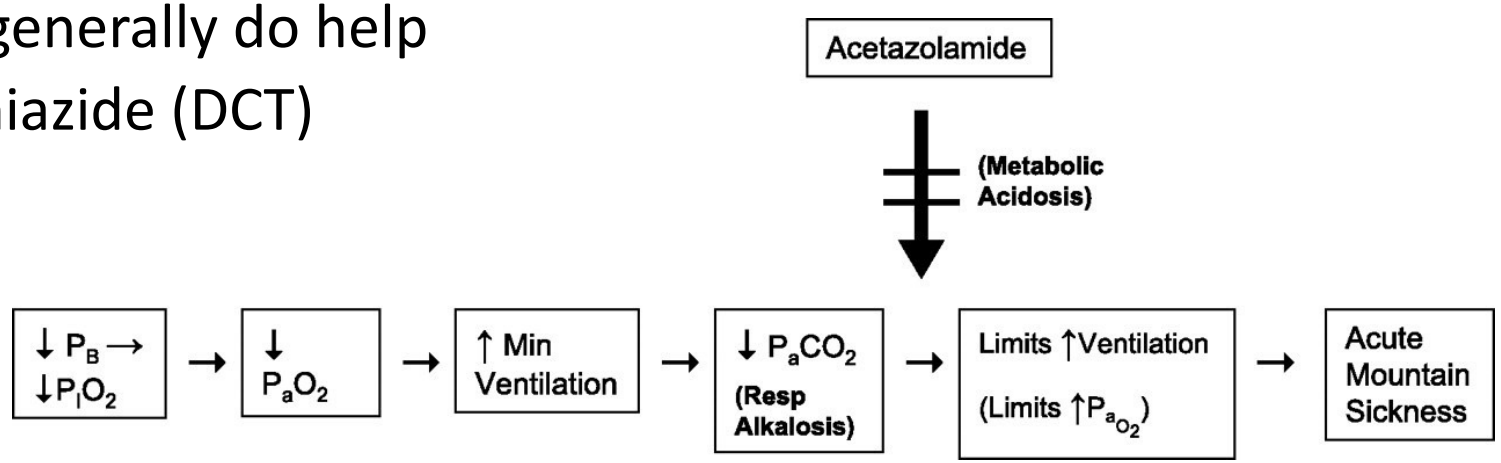
Acetazolamide

- Carbonic anhydrase inhibitor → bicarbonate diuresis
- Treatment and prophylaxis of altitude sickness
- Respiratory alkalosis due to hyperventilation, acetazolamide helps with compensatory metabolic acidosis
- Why does it help in heart failure?
  - Combination diuretics generally do help
  - Loop diuretic (LoH) + thiazide (DCT)



Dialysis (1)

- What is the most likely cause of intradialytic hypotension?

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B – Excessive ultrafiltration rate compared to vascular refilling

C – Cardiac dysfunction

D – Anaphylactoid reaction to dialyser circuits

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Intradialytic hypotension

- Antihypertensive medications may be dialysed off during dialysis
- Cardiac dysfunction not uncommon
- Anaphylactoid reactions to dialyser circuits generally uncommon
- Can cause:
  - Myocardial stunning
  - Cognitive impairment
  - Loss of residual renal function
- Ways to combat intradialytic hypotension:
  - Reduce intradialytic weight gain, use high dose diuretics
  - Extension of dialysis hours to reduce per hour rate of fluid removal (<10 ml/kg/hour) or increase number of sessions (home haemodialysis patients can have dialysis every 2nd day for example to avoid the Monday/Tuesday problem)
  - Ultrafiltration profiling
  - Cooling of dialysate to 36^{o}C
  - Haemodiafiltration
  - Avoid eating during dialysis

Dialysis (2)

- A haemodialysis patient is noted to have Hb=90 g/L, ferritin = 53 and transferrin saturation = 12% on routine blood testing. What is the most appropriate treatment for his anaemia?

A. Blood transfusion

B. Increase in erythropoietin dosage by 25%

C. Vadadustat

D. Iron infusion

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Anaemia in CKD

- Answer – Iron infusion
  - All haemodialysis patients regularly lose iron through the dialysis circuit, each dialysis ~ 20 ml of blood is lost even after flushing/returning blood to the patient
  - CKD patients also have functional iron deficiency, hepcidin is increased inhibiting iron absorption and sequestering iron in macrophages
    - Hepcidin normally
  - Target ferritin > 250 – 300, Sat > 25 – 30% and Hb = 95 – 115
  - Proactive > reactive regimens
- Vadadustat is a HIF-inhibitor which stimulates erythropoietin and red-cell production
  - Not available in Australia
  - Used in dialysis patients for treatment of anaemia
  - In pre-dialysis CKD possible increased risk of cardiac events

Dialysis (3)

- What causes dialysis related amyloidosis?

A. Beta 2 microglobulin

B. AL amyloid

C. AA amyloid

D. Transthyretin amyloid

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Middle molecules in dialysis patients

- Not well removed by dialysis and probably responsible for many problems including:
  - Pigmentation
  - Uraemic neuropathy
  - Amyloidosis – carpal tunnel syndrome

- However haemodiafiltration which removes middle molecules more effectively has ?effect on mortality

Dialysis (4)

- **Which of the following is the most likely cause of inadequate drainage in peritoneal dialysis?**

A. Constipation

B. Fibrin clots

C. Peritonitis

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Drainage/Ultrafiltration problems in peritoneal dialysis

- Drainage problems:
  - Constipation is the most common cause
  - Can also be caused by intraluminal obstruction (fibrin clots) or tube kinking or malpositioning or extraluminal problems such as omental wrapping

- Ultrafiltration problems:
  - Peritonitis can cause increased vascularity of the peritoneal membrane and change membrane characteristics affecting the amount ultrafiltered
  - Poor membrane function more often in patients who have been on peritoneal dialysis for some time due to long term exposure to glucose concentrations

Dialysis (5)

- **Which is the most common cause of hypertension in haemodialysis patients?**

A. Erythropoietin administration

B. Intravascular volume overload

C. Secondary hyperparathyroidism

D. Sympathetic nervous system activation

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Hypertension in dialysis patients

- Most commonly due to fluid overload
- General advice passed from one nephrologist down to the next -Once fluid overload is clinically evident the patient already has 3 – 5 kg of fluid extra
- Often able to reduce antihypertensives if ideal body weight accurately assessed

- Rapid rise in Hb/high dose of erythropoietin can cause hypertension
- Primary hyperparathyroidism may be a secondary cause of hypertension
- SNS activation purported to account for surge in BP at the end of dialysis in response to volume depletion

Polycystic kidney disease

- **A 37 year old man who has a family history of autosomal dominant polycystic kidney disease wishes to donate a kidney to his sister, who has end stage renal failure.**
- **Genetic testing for autosomal polycystic kidney disease is expensive and time-consuming. In order to preserve resources and for the best planning for the future of the organ donation for this family, who should be tested for polycystic kidney disease?**

A. The man

B. His sister

C. His mother

D. His father

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Cystic kidney disease testing now on the Medicare Benefits Schedule

- If the sister has ESKD secondary to polycystic kidney disease this should already be evident
- If either the mother or father have polycystic kidney disease which has not yet manifested as ESKD the man has a 50% chance of developing
- Could also follow the Pei-Ravine ultrasound criteria for the exclusion of ADPKD which is dependent on PKD1 or PKD2 mutation (identified in the sister) followed by ultrasound of the brother

Table 3. Pei-Ravine ultrasound criteria for making the diagnosis of ADPKD Adapted from Pei et al. 2011 [22] (Reprinted with permission).

	ADPKD Mutation Type					
	PKD1		*PKD2*		*Not known*	
Age of first-degree relative at-risk of ADPKD	PPV	Sensitivity	PPV	Sensitivity	PPV	Sensitivity
15-30 years with ≥ 3 renal cysts (unilateral or bilateral)	100%	94.3%	100%	69.5%	100%	81.7%
30-39 years with ≥ 3 renal cysts (unilateral or bilateral)	100%	96.6%	100%	94.9%	100%	95.5%
40-59 years with ≥ 2 renal cysts in each kidney	100%	92.6%	100%	88.8%	100%	90%
≥ 60 years with >4 renal cysts in each kidney	-	100%	-	100%	-	100%

Glomerulonephritis

- **A man presents with nephritic syndrome and bilateral shin petechial rash. Renal biopsy shows a pauci-immune glomerulonephritis. What is the most likely diagnosis?**

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C. ANCA vasculitis

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Glomerulonephritis

- Anti-GBM disease: Linear IgG staining
- Cryoglobulins: Antibody/antigen complexes so can IgG, IgM, C3 or C1q
- Infection related glomerulonephritis: Also antibody/antigen complexes so C3 (even a subset of infection related GN called ‘C3GN’) and IgG
- To make life difficult…
  - Even though ANCA vasculitis has an antibody involved in the pathogenesis, and rituximab is used for treatment, and plasmapheresis is still given in some cases…
  - Tissue damage in ANCA vasculitis is not due to antibody/antigen complexes activating the classical complement pathway but *ANCA-activated neutrophils and activation of the alternate complement pathway*
  - Hence efficacy of avacopan (C5a receptor inhibitor)

Renal artery stenosis

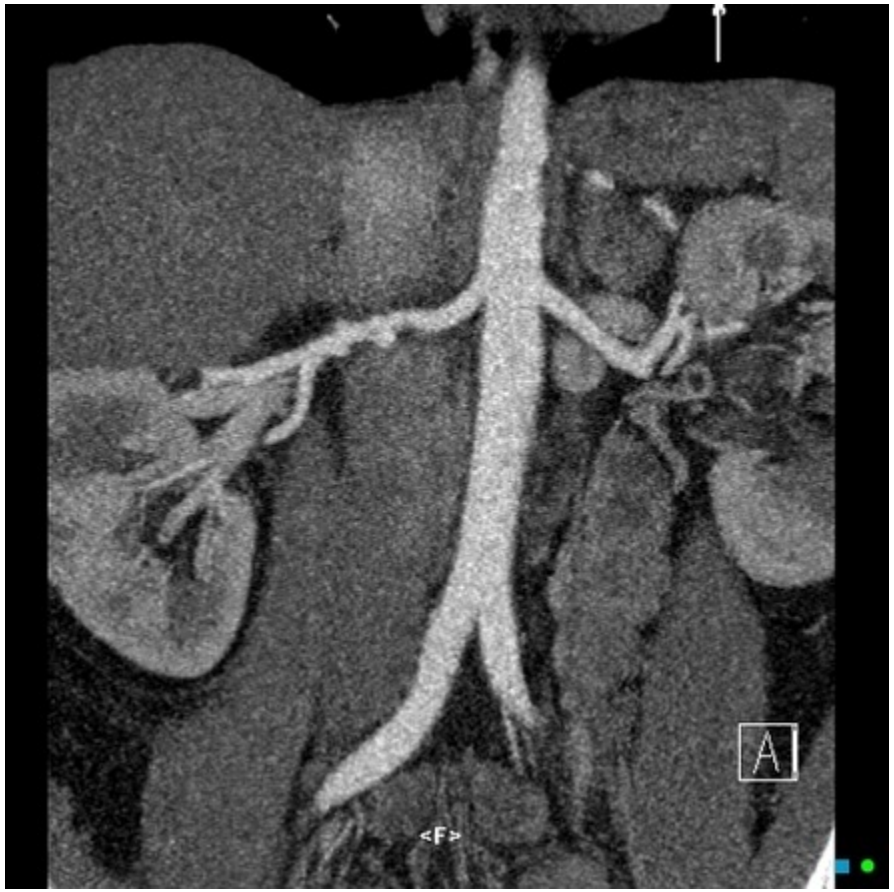
- **A 37 year old woman presents with hypertension. Her GP organises screening for secondary causes of hypertension. Her renal CT with contrast is shown below.**
- **What is the most likely cause of her secondary hypertension?**

A. Atherosclerotic renal artery stenosis

B. Fibromuscular dysplasia

C. Polyarteritis nodosa

D. Renal artery dissection



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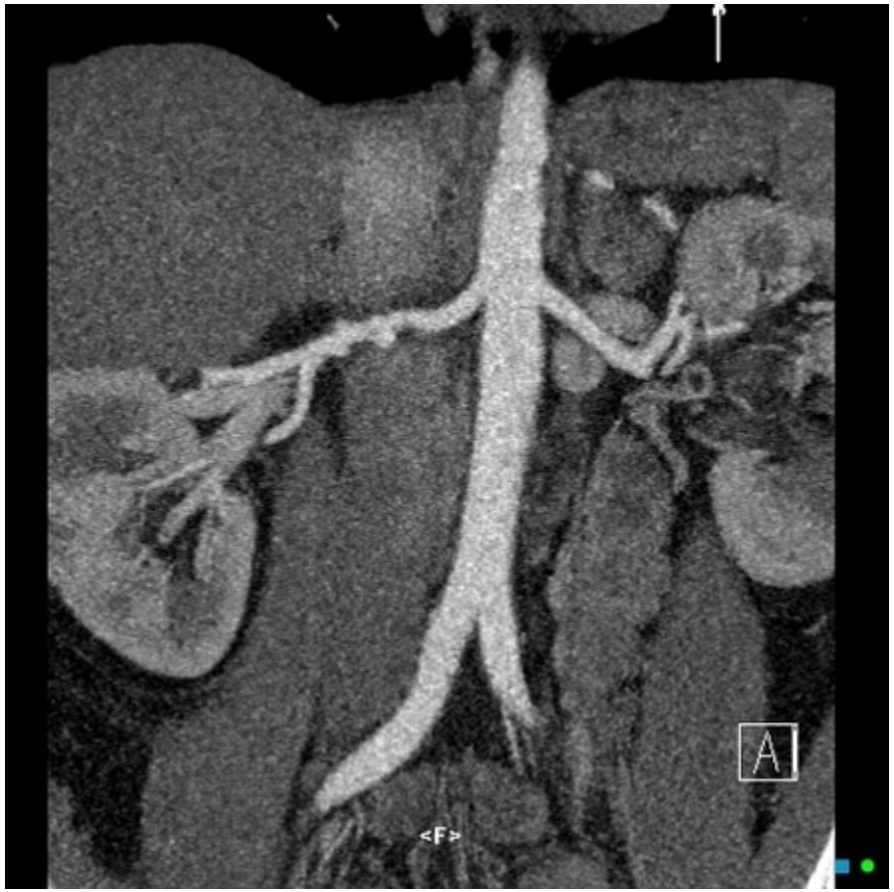

- A. Atherosclerosis causes ostial narrowing
- B. String of pearls appearance – treatment is angioplasty not stenting
- C. Medium vessel vasculitis which should cause aneurysms
- D. Renal artery dissection not seen in this picture

Urinary tract infection

- **An 84 year old female presented to the medical ward for management of congestive cardiac failure. A routine urine MCS was done on admission. Patient has no urinary symptoms.**

Urine culture: E.coli

WCC 10-100

RBC <10

Squamous epithelial cells <10

Sensitivities:

Amoxicillin sensitive

Cefuroxime parenteral sensitive

Cefuroxime oral intermediate

Trimethoprim resistant

What's the next best step?

A. No additional intervention/management

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Asymptomatic bacteruria

- Do not treat unless
  - Pregnant
  - Complex renal anatomy/renal calculi
  - Instrumentation of the urinary tract planned

Diabetes insipidus

- **In a patient with nephrogenic diabetes insipidus due to lithium, adjunctive treatment with amiloride will help by blocking transport of which ion?**

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B. Chloride

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D. Sodium

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Diabetes insipidus

- Central – Treated with intra-nasal ADH
- Nephrogenic – Partial response to ADH in kidney only resulting in water excretion ++
  - Low salt diet
  - Prostaglandin inhibition with NSAIDs
  - Amiloride can block the entry of lithium via ENaC and may prevent lithium induced nephrotoxicity
  - Diuretics (thiazides, amiloride, even acetazolamide) are given with the aim of activating the RAAS to reclaim more sodium and water in the proximal tubule and loop of Henle

Scleroderma renal crisis

- **What drug is most associated with scleroderma renal crisis?**

A. Corticosteroids

B. Cyclophosphamide

C. NSAIDS

D. Penicillamine

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Scleroderma renal crisis

- Precipitated by steroids > 15 mg/day, possible other risk factors but rare condition
- Severe hypertension, renal impairment, microangiopathic haemolytic anaemia
- Differential diagnosis:
  - Thrombotic microangiopathy for any reason
    - HUS, TTP
    - HELLP
    - Malignant hypertension for other reasons such as glomerulonephritis
    - Drug induced (tacrolimus)
- Treatment: ACEI

Thombotic microangiopathy

- Patient presents with altered consciousness and an acute kidney injury. Blood film shows features suggestive of microscopic angiopathic haemolytic anaemia. Which investigation would help decide the course of definitive treatment?

a. ADAMTS13 level

b. fibrinogen

c. d dimer

d. complement C3/C4

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Thrombotic microangiopathy

- If TTP patient needs plasmapheresis and replacement with FFP to top up ADAMTS13 (which may be low in TTP due to antibody against ADAMTS13 or actual deficiency). If level > 10% then…
- If HUS needs consideration of whether due to STEC or other infection
- If not due to STEC and possible atypical HUS then:
  - Consideration of eculizumab
  - Meningococcal vaccination
  - Amoxicillin prophylaxis

Acute kidney injury

- What is the other factor in the KDIGO diagnosis of AKI (apart from urine output)?

A) eGFR

B) FeNa

C) Serum Cr

D) Cr to urea ratio

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Measurement of renal function

- No nephrologist cares what the eGFR is in an acute kidney injury as eGFR is an estimation equation derived from stable outpatients with chronic kidney disease and does not reflect what is happening acutely
  - It's like telling me the renal function is reduced because you calculated the creatinine clearance using the Cockcroft and Gault equation
- FeNa – useful for working out if patient has ATN or pre-renal AKI
- Urea to creatinine ratio – The higher the urea is the more likely it's pre-renal
- Most accurate measurement of renal function in chronic setting is nuclear GFR which is what is used prior to kidney donation
  - 24 hour urine creatinine clearance – limited by the secretion of creatinine at the tubular level

One liners…

- What is the mechanism by which trimethoprim increases creatinine?

Reduces secretion of creatinine by the tubule, creatinine is largely freely filtered which is why it is used as a marked of GFR but is also secreted by the tubule

- What enema should you avoid in patients with chronic kidney disease?

Sodium phosphate – causes acute phosphate nephropathy