Griffith University Medicine Society
Gold Coast, Australia

Year 1 Peer Based Learning 2018
Renal System

Please note – this learning resource has been produced by the GUMS Academic Team. It is possible that there are some minor errors in the questions/answers, and other possible answers that are not included below. Make sure to check with other resources.

Scenario 1:

Stem 1: Ned Ron presents to your clinic. He has had congestive cardiac failure for 10 years. This was caused by long standing cardiac hypertrophy due to aortic stenosis.

Q1: Define ejection fraction

Stroke volume/total ventricular volume - explain that not all of the volume is ejected out of the ventricle during each systole

Q2: Ned Ron had aortic stenosis. Complete the following table by outlining the hypertrophic consequence of this:

	Aortic stenosis	Aortic regurgitation
Type of hypertrophy that will result?	**Concentric**	**Eccentric**
How are the sarcomeres added? **Draw this - see diagram below**	**Parallel**	**Series**
Does this lead to systolic or diastolic dysfunction?	**Diastolic**	**systolic**

Q3: A naive bond student asks you 'what about non-valvular causes of heart failure'? Fill in the following table, looking at other causes of heart failure:

	Concentric cardiac hypertrophy	Previous MI
Type of heart failure (systolic or diastolic) that will result?	**Diastolic**	**systolic**
Comment on the ejection fraction **(and link this to the exampels above)**	**Normal**	**reduced**

Note: use an example here where the total volume in a ventricle is 100mL, and usually 60mL is ejected.

If someone has had an MI, then their heart fill ok, but can't pump as much blood out - so, whilst the total ventricular volume is 100mL, only 40mL is ejected → reduces EF.

If someone has concentric hypertrophy, the problem is with the heart filling. However, as the muscle grows in parallel (he had aortic stenosis), it physically grows into the ventricle and their is less space → less filling. Now, when the stroke volume decreases, so does the total volume - 40/70 (for example) will produce a ***normal*** *ejection fraction, but it is still heart failure because it is still low*

Q4: name a synonym for diastolic and systolic heart failure

Diastolic: HFpEF; HFrEF - heart failure with preserved/reduced ejection fraction

Stem 2: Ned Ron comes back to your clinic later complaining of urinating infrequently. You take some bloods and find the following:

- Elevated Cr
- Elevated urea
- BUN:Cr > 15
- FENa <1%
- Urine Osm > 500

Q5: Given his history of heart failure, what is the most likely diagnosis? Briefly explain

Heart failure → cardiogenic shock → ***pre-renal AKI/azotaemia.***

Stress here that FENa for example isn't that important exam wise, but is good to make note of know as it is used clinically

Q6: Contrast the ratio of BUN to creatinine reabsorption in someone without kidney issues (i.e. what is the normal BUN:Cr ratio)

Creatinine is reabsorbed far less; urea is absorbed far more - usually in a ratio of 15:1

Q7: Outline the pathophysiological mechanism for increased BUN:Cr ratio in patients with pre-renal AKI

Less blood flow to kidney → RAAS activates → more water reabsorption from aldosterone's action - this is coupled to more urea reabsorption, relative to Cr reabsorption (what normally

happens) → elevated BUN:Cr - point of this question = ***RAAS -*** *FEF and urine Osm are explained in a later question*

Q8: briefly explain how the following would also lead to pre-renal AKI:

- Pulmonary embolism - **pulmonary blood vessels obstructed → less blood flow to left side of heart → less blood ejected to systemic circulation AND PE causes right heart failure → further exacerbates lack of blood flow**
- Tension pneumothorax - **kinking of veins returning into RA → same as above**
- Anaphylaxis - **widespread oedema leads to increased volume in interstitium and hence decreased volume in plasma**

Stem 3: Ned Ron soon develops the following findings after his findings:

- BUN:Cr < 15
- FEFNa > 2%
- Urine Osm < 500
- Hyperkalaemia
- Metabolic acidosis
- Casts in urine

Q9: what is the most likely diagnosis? What has caused this?

He now has ***intra-renal AKI****, in particular,* ***acute tubular necrosis (most common cause of intra-renal AKI).*** *explain that intra-renal AKI is commonly caused by two things - nephrotoxins (e.g. some medications) and ischaemia - key concept here =* ***intra-renal AKI is commonly preceded by pre-renal AKI***

Q10: explain his FEFNa and urine Osm, as well as the reason their are casts in the urine

In pre-renal AKI, there is ***no*** *damage to the renal tubules. However, in acute tubular necrosis, there are now less tubular epithelial cells which can do their function. Therefore, less sodium is reabsorbed, which leads to more sodium in the urine (high FEFNa) as well as less ability to concentrate urine because their is less water reabsorption (low urine Osm). Casts are from dead tubular cells sloughing into tubular lumen*

Stem 4: Ned Ron later presents to clinic with a fractured neck of femur. His kidney function has continued to deteriorate. He is found to have chronic kidney disease

Q11: list the major findings in CKD:

Encourage them all to use this mnemonic - if all of them use it and discuss it then they will remember the content better

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Consequences (**MAD HUNGER**):

- **M**etabolic **A**cidosis
- **D**yslipidemia (especially ↑ triglycerides)
- **H**yperkalemia
- **U**remia—clinical syndrome marked by:
  - Nausea and anorexia
  - Pericarditis
  - Asterixis
  - Encephalopathy
  - Platelet dysfunction
- **N**a^+/H_2O retention (HF, pulmonary edema, hypertension)
- **G**rowth retardation and developmental delay
- **E**rythropoietin failure (anemia)
- **R**enal osteodystrophy

Q12: explain the mechanisms for the following:

- Hyperkalaemia - **decreased K+ secretion in tubules**
- Hyperparathyroidism - **less hydroxylation of vit D → less intestinal Ca2+ absorption → hyperparathyroidism**

Q13: explain how his CKD can make his heart failure worse

Na+/H2O retention → more blood volume → more work on heart → creates a viscious cycle

W1: the patient had BPH? List the main complications

Post-renal AKI, hydronephrosis, pyelonpehritis

W2: is a renal colic in a ureter or chronic BPH more likely to cause elevated Cr? Why?

Chronic BPH - if the blockage is at the urethra, both kidneys will be affected, whereas a colic in one ureter will only affect one kidney. To elevate Cr, this will more likely be from bilateral kidney disease

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Scenario 2:

Four patients: Kate, Duncan, Victor and Patricia all come into your clinic. Duncan has end-stage hyperaldosteronism and is about to die. The others all don't cope well. Kate has an opioid overdose, Victor has a panic attack and Patricia starts drinking a lot of coke, aggravating his diabetes which gives him DKA.

Q1. Outline the pathophysiology of these four conditions, in terms of the acid base disorder it will cause:

1. Hyperaldosteronism → Na+ and H2O reabsorbed in exchange for K+ and H+ → excess H+ secretion leaves more HCO3- in comparison → leads to metabolic alkalosis
2. Panic attack → hyperventilation → excessive loss of CO2 → respiratory alkalosis
3. Opioid overdose → hypoventilation → CO2 retention → respiratory acidosis
4. DKA – ketones are acidic themselves → metabolic acidosis

Q2. Outline the following findings in each of the four acid-base disorders: pH, pCO2, HCO3- and compensation

	pH	P_{CO_2}	$[HCO_3^-]$	COMPENSATORY RESPONSE
Metabolic acidosis	↓	↓	↓	Hyperventilation (immediate)
Metabolic alkalosis	↑	↑	↑	Hypoventilation (immediate)
Respiratory acidosis	↓	↑	↑	↑ renal $[HCO_3^-]$ reabsorption (delayed)
Respiratory alkalosis	↑	↓	↓	↓ renal $[HCO_3^-]$ reabsorption (delayed)

Key: ↑ ↓ = 1° disturbance; ↓ ↑ = compensatory response.

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Renal Scenario

You are on the renal ward at GCUH. You have a few patients who have various kidney diseases each presenting with symptoms of the nephrotic syndrome.

What are the components of the filtration barrier in the kidney?

- *Capillary endothelium*
- *Basement membrane*
- *Foot processes of podocytes*

Explain the mechanism leading to each of the following symptoms of the nephrotic syndrome. *(Hint: remember that the nephrotic syndrome involves derangement of the kidney filtration barrier)*

Proteinuria	Derangement of the kidney filtration barrier leading to increased permeability. This can be due to immune mechanisms or non immune mechanisms. This leads to loss of protein and lipids into filtrate
Peripheral oedema	Loss of albumin/protein from blood = ↓ plasma oncotic pressure = fluid movement to interstitial fluid
Lipiduria	In response to low protein, the liver upregulates all protein and lipid production leading to hyperlipidaemia. Lipiduria is loss of these lipids via urine, due to damage to the filtration barrier.
Decreased prothrombin time	Loss of anticoagulant proteins (such as anti-thrombin III) in the urine – main mechanism. Additionally, in response to low protein, the liver upregulates all protein production including clotting factors.
Increased risk of infection	Hypo-gammaglobulinaemia results – again, these proteins are lost in the urine

Summarise it to them as: **hypoalbuminaemia, hyperlipidaemia, hypercoaguable state, hypogammaglobulinaemia**

A Bond student also on placement with you suggests to the consultant using IV saline and albumin treat some of these patients. The consultant asks if you agree? Explain your answer.

- You definitely disagree, as you will just piss this out!

Some of these patients go on to develop various other complications. Explain the mechanism of the following signs and symptoms for THESE patients.

Unilateral calf pain and swelling at rest	Patient has a DVT Increased clotting factor production and loss of antithrombin III =

	hypercoagulable state = predisposed to DVT. Patient is also in hospital (likely on bed rest) so there is likely a stasis of blood. Increased risk of endothelial damage due to hyperlipidaemia as a result of nephrotic syndrome.
Dyspnoea, stony dull percussion note and decreased breath sounds at the base of the lungs bilaterally	Patient has pleural effusion, due to loss of protein from blood (same mechanism as peripheral oedema above). **Here, prompt discussion of exudate vs transudative pleural effusions – use this table as a guide:** **Exudate**: Cellular (cloudy); ↑ protein (> 2.9 g/dL); Due to: Lymphatic obstruction (chylous), Inflammation/infection, Malignancy **Transudate**: Hypocellular (clear); ↓ protein (< 2.5 g/dL); Due to: ↑ hydrostatic pressure (eg, HF, Na^+ retention), ↓ oncotic pressure (eg, cirrhosis, nephrotic syndrome)
Sudden onset flank pain with an acute decline in GFR	Renal vein thrombosis, caused by hypercoagulable state (as per DVT answer above)

Recall, main features of nephrotic syndrome are:

- *Proteinuria*
- *Hypoalbuminaemia & hypo-gammaglobulinaemia*
- *Peripheral oedema*
- *Hyperlipidaemia*
- *Lipiduria*

What if another patient (not necessarily with nephrotic syndrome) presented with the following findings on urine dipstick analysis? Indicate whether they are normal or not, and indicate a possible cause (other than filtration barrier problems or medications)?

	Is this normally found in urine?	**If not, describe a possible cause of the pathology (other than a filtration barrier problem)**
Bilirubin	No	Post-hepatic causes of jaundice (eg, bile duct obstruction)
Urobilinogen	Yes	Some urobilinogen is normally excreted in urine
Glucose	No	Diabetes mellitus
Haemoglobin	No	Haemolysis, cancer, pyelonephritis

Another patient presents with suprapubic pain, dysuria, urinary urgency and frequency. Their urine dipstick is positive for nitrites and leukocytes. Explain why.

- Cystitis (UTI involving bladder and urethra)
- Likely caused by *E. coli*, because *E. coli* has an enzyme that converts nitrates to nitrites
    - If no nitrites present, UTI likely due to non-E coli bacteria.
- Leukocytes present due to infection