REVISE
NEPHROLOGY

Week 1
MCQs and Answers

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1. Which of the following is true about the diagnosis of Hepatorenal syndrome (HRS)-

a. It can be definitively diagnosed only with renal biopsy
b. Urinary eosinophils with worsening renal function are virtually diagnostic
c. A liver biopsy can be helpful in establishing the diagnosis
d. White cell casts in urine are diagnostic
e. It is a diagnosis of exclusion

2. The following electrolyte profile may be associated with both thiazide and loop diuretics-

a. Hyperkalaemia with metabolic alkalosis
b. Hypokalaemia with metabolic alkalosis
c. Hyperkalaemia with metabolic acidosis
d. Hypokalaemia with metabolic acidosis
e. Hyperkalaemia

3. Fanconi syndrome should be suspected in-

a. Hyperuricemia
b. Hyperphosphatemia
c. Hypophosphatemia
d. Hyperkalaemia
e. Alkalosis

4.Which of the following is true about membranous nephropathy (MN)?

a. It is a common cause of nephrotic syndrome and ESRF in children
b. It is caused by auto-antibodies to C3 convertase called C3 nephritic factor
c. The characteristic biopsy finding is presence of "spikes" in the GBM on silver methenamine stain.
d. Immunosuppressive treatment is recommended in those with persistent sub-nephrotic range proteinuria

5. The best marker for susceptibility to thromboembolism in a patient with active nephrotic syndrome is:

a. Serum creatinine
b. Degree of hypertension
c. Degree of proteinuria
d. Serum albumin
e. Leg swelling

6. The most important risk factor for the development of post-transplant lymphoproliferative disorder (PTLD) in a solid organ transplant patient is-

a. Epstein-Barr virus mismatch between host and recipient
b. Kidney transplant
c. Previous infection with cytomegalovirus (CMV)
d. High serum creatinine
e. Use of alemtuzumab (an anti-CD52 antibody)

7. Which of the following is not a risk factor for developing contrast induced AKI?

a. CCF
b. Multiple myeloma
c. Sepsis
d. Use of perindopril
e. Use of metformin

8. The commonest diagnosis seen on renal biopsy of patients with asymptomatic haematuria is-

a. Thin basement membrane nephropathy
b. Alport's syndrome
c. Post infectious glomerulonephritis
d. IgA nephropathy
e. Lupus nephritis

9. Antibiotic of choice while pending culture reports in a suspected case of pyelonephritis in patient with ADPKD is -

a. Intravenous ampicillin
b. Ceftriaxone
c. High dose penicillin
d. Ciprofloxacin
e. Aminoglycoside

10 The commonest renal disease associated with myeloma is:

a. ATN
b. AKI secondary to Hypercalcemia
c. Fanconi syndrome
d. Myeloma cast nephropathy
e. Nephrotic syndrome

Answers:

1. **E. It is a diagnosis of exclusion**

There is no histological abnormality in the HRS kidney and the disease is due to unopposed renal vasoconstriction. Any significant findings on renal biopsy or urine examination should make one look for a cause other than HRS causing the renal failure.

Criterion for the diagnosis of HRS:

- Not pre or post renal failure/ recent nephrotoxic drugs/infections
- Serum creatinine > 133 umol/l
- No significant haematuria (<50 RBC/HPF) and none or minimal proteinuria (< 500 mg/day)
- No improvement in renal function (to < 133umol/L) after volume expansion with intravenous albumin (1 g/kg of body weight per day up to 100 g/day) for at least two days and withdrawal of diuretics

2. **B. Hypokalaemia with metabolic alkalosis**

All diuretics act by inhibiting the tubular reabsorption of Na^+. With more Na^+ flowing to the distal nephron, the tubular cells try to reclaim the excess Na^+, leading to decrease in positive charges within the lumen. The tubular epithelial cells try to compensate for this by secretion of the positively charged K^+ and H^+ into the lumen. Also, intravascular volume contraction because of the diuretics causes activation of the renin-angiotensin-aldosterone (RAS) which in turn leads to the urinary K^+ and H^+ losses under the influence of aldosterone.

3. **C. Hypophosphatemia**

Type 2 RTA is due to a proximal tubular defect in the reabsorption of $HCO3^-$ leading to urinary loss of $HCO3^-$ and metabolic acidosis with hypokalaemia. The absorption of HCO3- in this segment is linked to the coupled exchange of H^+ and Na^+. As this reabsorption of Na^+ is linked to the reabsorption of glucose, amino acids, phosphate and uric acid, the patient may present with glycosuria, hypophosphatemia and hypouricemia (due to urinary losses of phosphate and uric acid respectively) and this condition is termed as Fanconi syndrome.

4. **C. The characteristic biopsy finding is presence of "spikes" in the GBM on silver methenamine stain.**

Idiopathic MN is common in Caucasian males and quite uncommon in children. C3 nephritic factor is involved in the pathogenesis of mesangiocapillary GN (MCGN). More than 70% of idiopathic MN have circulating auto-antibodies against phospholipase A2 receptor 1(PLA2R1) located on the surface of podocytes. The characteristic abnormality on light microscopy is diffuse global capillary wall thickening due to subepithelial immune-complex deposition which appears as "spikes" in silver methenamine stain. Patients with sub-nephrotic range proteinuria are treated with ACEI or ARB alone and immunosuppressive therapy is only indicated in those with persistent high-grade proteinuria > 4 g/daily despite the use of ACEI or ARB.

5. **D. Serum albumin**

A low serum albumin concentration at the time of diagnosis, but not the degree of proteinuria, independently predicts venous thromboembolic event in patients with nephrotic syndrome. The risk of thrombosis varies among the causes of nephrotic syndrome and appears to be highest in patients with membranous nephropathy.

6. A. Epstein-Barr virus mismatch between host and recipient

The pathogenesis of PTLD in post-transplant patients is mostly related to B cell proliferation induced by infection with Epstein-Barr virus (EBV) in the setting of immunosuppression. In most parts of the world, 90 to 95 percent of adults show serologic evidence of EBV infection. Acute EBV infection leads to a polyclonal expansion of B cells harbouring the virus. In the immunocompetent, these virally infected B cells elicit a T cell response that eliminates the vast majority of the infected B cells. However, a small population of the virally infected B cells downregulate viral antigen expression and thus escape immune surveillance.
The incidence of PTLD is highest in the first year after transplantation, probably due to the more intense immunosuppression in this period (which suppresses the T cell response). The incidence of PTLD is much greater in heart transplant recipients probably due to the more intense immunosuppression in this group of patients. While there is some evidence linking the use of tacrolimus to PTLD, mycophenolate and alemtuzumab use do not seem to be associated with higher incidence of PTLD.

7. E. Use of metformin

Contrast nephropathy is a generally reversible form of AKI due to ATN that occurs soon after the administration of radio contrast media. The increase in creatinine is generally observed within 24 to 48 hours and usually starts to decline within three to seven days (unlike other causes of ATN where recovery usually takes longer).
The risk factors for developing contrast induced AKI are:-

- CCF (causing reduced renal perfusion)
- CKD (particularly from diabetic nephropathy)
- Type of radiologic procedure: Highest risk is associated with interventional (rather than diagnostic coronary angiography (particularly in the setting of AMI)
- Myeloma
- Use of ACEI or ARB, NSAIDs
- Type and dose of contrast

Metformin is not a nephrotoxic medication and thereby not a risk factor for developing contrast induced AKI.

8. A. Thin basement membrane nephropathy

It is estimated that 20 to 25% of patients who have renal biopsy for asymptomatic haematuria have thin basement membrane nephropathy. IgA nephropathy is the commonest cause of glomerulonephritis world-wide, but the question was for patients with asymptomatic haematuria and not glomerulonephritis. Patients with post infectious glomerulonephritis and lupus nephritis are unlikely to present with just asymptomatic haematuria. Though Alport's syndrome is the commonest cause of hereditary nephritis and can initially present with asymptomatic haematuria, it is much less common than thin basement membrane nephropathy.

9. D. Ciprofloxacin

Trimethoprim-sulfamethoxazole and fluoroquinolones by virtue of their lipophilic nature are able to cross the lipid rich cyst wall and achieve good intracystic concentration. Aminoglycosides and the penicillins frequently do not penetrate cysts and are not predictably effective treatment for infected cysts in patients with ADPKD.

10. D. Myeloma cast nephropathy

Free light chains, which are always produced in excess in patents with myeloma, are extensively filtered across the glomeruli, unlike the non-filterable bigger immunoglobulin molecules. The filtered light chains are reabsorbed by the proximal convoluted tubular (PTC) cells. Once endocytosed, the free light chains are toxic to the PCT cells, and this can lead to the picture of proximal renal tubular acidosis (type 2 RTA) and Fanconi syndrome as well as ATN. Gross injury to the PTC cells allows overflow of free light chains to the ascending limb of loop of Henle where they bind to the Tamm-Horsfall protein, leading to the formation of tubular casts. These tubular casts occlude the tubular lumen leading to cast nephropathy (myeloma kidney) and the resultant renal failure. The greater the urinary excretion of free light chains, the greater is the risk for renal failure. While hypercalcemia can contribute to the severity of AKI in patients with myeloma, it is only uncommonly the solitary reason for ongoing renal failure in these patients. Myeloma can lead to amyloidosis which may manifest as nephrotic syndrome.
Following is the list of histological lesions present with distinct clinical entities in monoclonal plasma cell disorders (RPGN- rapidly progressive glomerulonephritis)

Histology	**Renal function**	**Clinical presentation and urinalysis**
Cast nephropathy (30-50 %)	Reduced GFR	AKI Tubular proteinuria (generally <1-2g/day), light chains in urine, no haematuria
ATN (10%)	Reduced GFR	AKI Normal urinalysis
Monoclonal Immunoglobulin deposition disease (5%)	Normal or reduced GFR.	Nephrotic syndrome; AKI Nephrotic range proteinuria (>3 g/day), light chains in the urine, no haematuria
AL Amyloidosis (10%)	Normal or reduced GFR	Nephrotic syndrome Nephrotic range proteinuria (>3 g/day), light chains in the urine, no haematuria. Hypotension is commonly seen secondary to autonomic nervous system and/or cardiac involvement
Cryoglobulinaemic glomerulonephritis (<1%)	Reduced GFR	AKI, RPGN Haematuria, proteinuria, casts.