REVISE NEPHROLOGY

Week 2

MCQs and Answers

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Week 2

1. A 52-year-old woman presents with rising blood urea and creatinine over a 4-week period. Her urine has dysmorphic RBCs and the serum complement levels are low. Which one of the following causes is unlikely to be the cause of this presentation?

a. Infective endocarditis
b. Cryoglobulinemia
c. MPGN
d. Systemic lupus erythematosus
e. Cholesterol atheroembolic disease

2. A 34-year-old male has been diagnosed minimum change disease (MCD) on renal biopsy when he presented with nephrotic syndrome. After six months his urine is still heavily proteinuric and he has generalised oedema despite being compliant with 25 mg prednisolone and 10 mg perindopril. What is the next most appropriate step?

a. Increase the dose of prednisolone to 60 mg
b. Start rituximab
c. Repeat renal biopsy
d. Extend the treatment to 1 year
e. Replace prednisolone with cyclosporine

3. A 35-year-old male presents with acute onset of right loin pain radiating to groin with vomiting. He is afebrile and hemodynamically stable. Urine dipstick shows blood and leukocytes. Which of the following imaging test is most reliable in confirming or excluding ureteric colic due to suspected calculus obstruction?

a. Plain X-ray abdomen AP & Right lateral views
b. Ultrasound KUB
c. Non-contrast CT scan KUB
d. Gadolinium enhanced MRI scan of KUB

4. Solid organ malignancy is most commonly associated with -

a. Membranous nephropathy
b. FSGS
c. MCD
d. Mesangiocapillary GN
e. IgA nephropathy

5. A 54-year-old woman with diabetic nephropathy has undergone renal transplantation. On her fifth postoperative day the urine output is still 20 ml/hour. Which one of the following is not a potential cause of this prolonged post-transplant oliguria?

a. Acute tubular necrosis
b. Arterial anastomotic stenosis
c. Hyperacute rejection
d. Volume depletion
e. Ureteral lymphocele

6**. A 54-year-old man presents with constipation and polyuria. His serum calcium is elevated with suppressed serum PTH. All of the following might be responsible for these findings, except**:

a. Metastatic breast cancer
b. Familial hypocalciuric hypercalcaemia (FHH)
c. Thiazide abuse
d. Milk-alkali syndrome
e. Addison's disease

7. A 26-year-old young woman who had had thyroid surgery in the past presents with intermittent hypertension, headache, sweating and palpitations. Further work-up discloses the presence of bilateral adrenal pheochromocytomas. Her older brother has asymptomatic hypercalcaemia. Which one of the following should be considered as the likely diagnosis?

a. Multiple endocrine neoplasia type I (MEN 1)
b. Cushing syndrome
c. Multiple endocrine neoplasia type 2A (MEN2A)
d. MEN 2B
e. Neurofibromatosis type I

8. Dialysis disequilibrium syndrome refers to neurological symptoms and signs during or shortly after dialysis. The pathogenesis of dialysis disequilibrium is most commonly due to:

a. Relative hypotension
b. Cerebral oedema
c. Uremic platelet dysfunction
d. Aberrant calcium and phosphate metabolism
e. Aluminium toxicity

9. Which of the following conditions is least likely to lead to rapidly progressive glomerulonephritis (RPGN)?

a. Lupus nephritis
b. IgA nephropathy
c. Membranous nephropathy
d. Microscopic polyangiitis
e. Anti-GBM antibody disease

10. A 70-year-old man with a history hypertension has a CT abdomen for investigation of abdominal pain. There is an incidental finding of 80% right renal artery stenosis. His BP is 154/98 mm Hg and his current medications are perindopril 10mg daily and amlodipine 10mg daily. What is the best course of management for this gentleman?

a. Continue current therapy
b. Add hydrochlorothiazide
c. Change amlodipine to metoprolol
d. Renal artery angioplasty
e. Renal sympathetic denervation

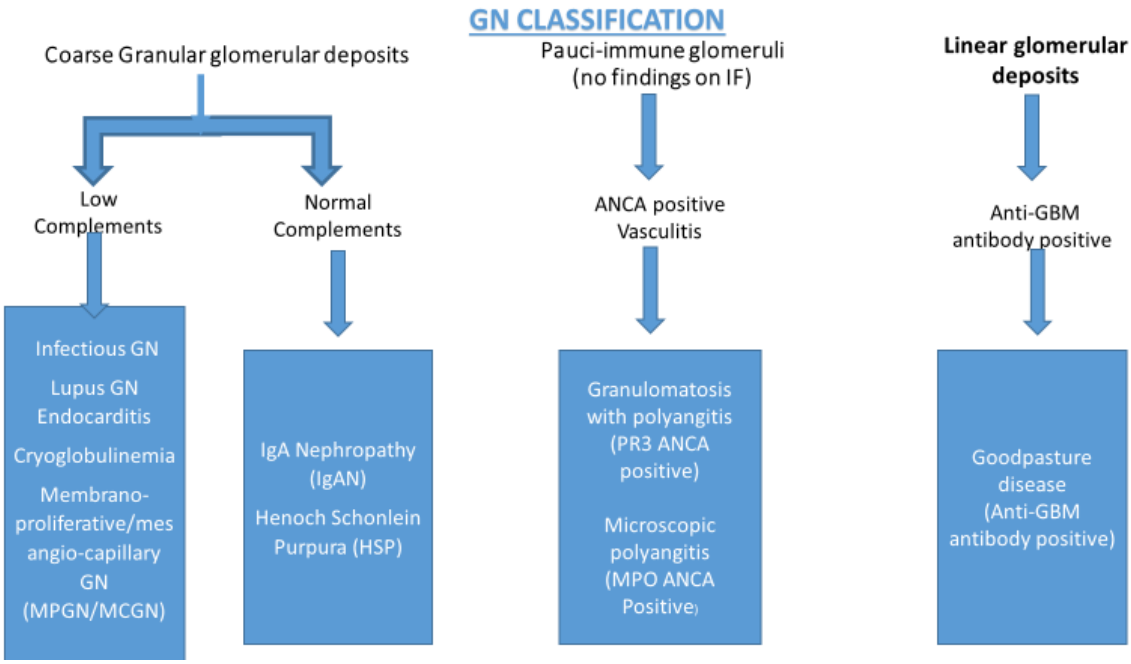
Answers

1. E. Cholesterol atheroembolic disease

Embolism of small usually distal arteries by cholesterol crystals/small pieces of atheromatous material originating from an atherosclerotic plaque causes Cholesterol atheroembolic disease. It is not associated with GN and urine may show white cells or white cell casts (but no dysmorphic RBC or RBC casts). Other features include:

- Usually follow angiography/angioplasty (commonest cause) or vascular surgery
- Effects skin (livedo reticularis, blue toes), GI (abdominal pain, diarrhea, bleed), CNS (stroke, TIA, confusion), eyes (Hollenhorst plaques) and kidney (**subacute AKI with bland urine**)
- May have transient hypocomplementemia and eosinophilia (eosinophiluria not very specific or sensitive)
- **Unlike contrast nephropathy (which presents in a few days) presents few weeks to months after the inciting event and associated with a poor prognosis.**

Following is a differential of GN based on IF findings and you can see the low complement causes include all the other choices in this Q.



2. C. Repeat renal biopsy

- Both MCD and FSGS are associated with diffuse foot process effacement and hence biopsy needed to confirm diagnosis is MCD and not FSGS in this case
- Due to the focal nature of FSGS it may be mistaken for MCD if not enough glomeruli are visualised in biopsy
- If the repeat biopsy confirms MCD then to consider cyclophosphamide, cyclosporine or rituximab

3. C. Non-contrast CT scan KUB

Non-contrast CT scan is highly sensitive to detect stones including uric acid stones which are radiolucent in plain X-ray. CT scan also has the advantage of being able to accurately locate the level of obstruction. While ultrasound is excellent to diagnose obstruction and identify renal calculi, it is less reliable in detecting ureteric stones.

4. A. Membranous nephropathy

Up to two thirds of cases of membranous nephropathy (MN) are primary or idiopathic and the majority of these are associated with anti PLA2 receptor antibody. The following conditions and agents are associated with secondary MN:

- **Autoimmune disorders**: SLE (common), rheumatoid arthritis, Sjogren's syndrome, Grave's disease, Dermatomyositis, Mixed connective tissue disorder, Systemic sclerosis

- **Infections:** Hepatitis B (common), Hepatitis C, Schistosomiasis, Malaria, Leprosy, Filariasis

- **Drugs:** Gold, Penicillamine, NSAIDs, Captopril

- **Malignancy:** Solid organ tumours e.g. prostate, lung or colon, less commonly haematological malignancies like CLL

5. C. Hyperacute rejection

Acute, but not hyperacute, rejection is the cause here. Hyperacute rejection happens in the first 24 hours. The causes mentioned in the question should always be kept in mind in this clinical setting. The common causes of rejection < 7 days post-transplant include acute rejection and acute tubular necrosis (due to prolonged ischaemic injury during the operation). Volume depletion can also lead to this scenario. Ureteric obstruction by a haematoma or lymphocele is rare and can be ruled out by ultrasound examination.

6. B. FHH

Causes of hypercalcaemia with normal (which is inappropriate for the high serum calcium) or elevated serum PTH include-
- primary hyperparathyroidism
- Tertiary hyperparathyroidism (there will be history of secondary hyperparathyroidism which would be in the context of CKD or rickets/osteomalacia)
- Lithium-induced hyperparathyroidism
- Familial hypocalciuric hypercalcaemia (FHH).

FHH can be diagnosed by inappropriately suppressed urinary 24 hours Ca or urinary Ca/creatinine ratio < 0.01. Hypercalcemia due to all other causes will lead to the kidneys trying to excrete Ca.
Both lithium and FHH lead to abnormality in the Ca sensing receptors in the kidneys (allowing uncontrolled renal Ca reabsorption leading to hypocalciuria and hypercalcemia) as well as Ca sensing receptors in the parathyroid gland (allowing ongoing PTH secretion despite hypercalcemia).
All the other choices here will lead to hypercalcemia with suppressed PTH levels.

7. C. MEN2A

MEN 2A consists of medullary thyroid carcinoma often presenting early in life, pheochromocytoma in upto 50% and multiglandular hyperparathyroidism in upto 25%. MEN 2b has medullary thyroid carcinoma and pheochromocytoma but not hyperparathyroidism. MEN2B also includes mucosal neuromas, typically involving the lips and tongue, intestinal ganglioneuromas and often a marfanoid habitus. The pheochromocytomas are often bilateral and multiple in the MEN 2 syndromes.

MEN1 consists of multiple parathyroid tumours causing hyperparathyroidism (100% penetrance by 40 to 50 years age), pituitary adenomas in up to 60% (gastrinoma, insulinoma, non-functioning tumours) and pancreatic islet cell or gastrointestinal endocrine cell tumors in up to one third.

8. B. Cerebral oedema

The dialysis disequilibrium syndrome (DDS) is characterized by a range of neurologic symptoms that affect patients on haemodialysis, particularly when they are first started on dialysis or have missed consecutive dialysis.
Urea is generally considered an "ineffective" osmole because of its ability to permeate cell membranes. However, equilibration of urea across cell membranes may take several hours to reach completion. Haemodialysis rapidly removes small solutes such as urea. The rapid decline in the blood urea content in patients significantly lowers plasma osmolality, while the neurons with a higher urea concentration have a high intracellular osmolality.
This gradient leads to water shift into neurons that produces cerebral oedema.
To prevent the development of DDS, haemodialysis is initiated in shorter sessions using low blood flow initially. While patients well established on haemodialysis usually have three sessions of 4 to 5 hours each week with blood flows of up to 300 mls/minute, those being initiated to haemodialysis usually start with two-hour session increasing by half hours with blood flows of about 200 mls/minute.

9. C. membranous nephropathy

Membranous nephropathy is associated with nephrotic syndrome and NOT glomerulonephritis and hence does not lead to RPGN.
RPGN refers to the clinical syndrome of nephritic syndrome along with rapid deterioration in kidney function over days to weeks. Histologically RPGN is characterised by the finding of crescent in the glomeruli (crescentic GN).
Normally Bowman's space has a single layer of parietal and visceral epithelial cells each**.** Crescents ar**e** defined by the presence of **t**wo or more layers of proliferating parietal epithelial cells in Bowman's space; they are essentially a marker of severe glomerular inflammation.
Rents in the glomerular capillary wall due to glomerular inflammation leads to leakage of plasma products including fibrinogen, into bowman's space followed by influx of macrophages and T cells and the release of proinflammatory cytokines.
Crescentic GN leading to RPGN is classified into three types:

- Pauciimmune RPGN: Causing more than 50% of all RPGN, this group is due to ANCA-associated vasculitis such as granulomatosis with polyangiitis, microscopic polyangitis or rarely eosinophilic granulomatosis with polyangitis (Churg-Strauss).

- Anti-GBM antibody disease associated RPGN: Characterised by the presence of anti-GBM antibodies and linear staining of the GBM on IF, this leads to 20% of all RPGN.
- Immune complex mediated RPGN: IF characteristically shows the presence of coarse immune deposits in the glomeruli; about 25% of all RPGN is immune complex mediated and may be due to lupus nephritis (commonest in this group), IgA nephropathy, infectious GN, or mesangiocapillary glomerulonephritis (MCGN).

10. B. Add hydrochlorothiazide

The first line management of renal artery stenosis (RAS) is most often medical, while fibromuscular dysplasia (FMD) patients (who are usually younger) are often treated with revascularization procedures. In RAS, revascularization is only offered (in conjunction with medical therapy) in the following group of patients:
a) Short duration of blood pressure elevation prior to the diagnosis of renovascular disease
b) Failure of optimal medical therapy
c) Intolerance to optimal medical therapy
d) Recurrent flash pulmonary oedema
e) Unexplained progressive renal insufficiency

Renovascular hypertension is one of the most common causes of secondary HTN. There are two common clinical variants of renovascular HTN: atherosclerotic RAS and FMD. About 80% of renovascular HTN cases are due to atherosclerotic disease and 20% are related to FMD. HTN appearing in younger individuals (i.e. children or young adults) is suggestive of FMD, while atherosclerotic renal artery stenosis should be suspected in recent onset of HTN in previously normotensive individuals above the age of 55 years. Other situations where renovascular HTN is suspected are resistant or malignant HTN, more than 1.5 cm discrepancy in the kidney sizes, episodes of unexplained flash pulmonary oedema and a rise in serum creatinine of more than 30% after initiating an angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB).