Revise Nephrology

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2nd set of Pre-MCQs Answers 2023

Answers

16. Answer D

CKD with enlarged kidney size is seen in diabetic nephropathy, HIV nephropathy, polycystic kidneys, hydronephrosis (except in retroperitoneal fibrosis, where the hydronephrosis is encased within a thick fibrous envelop), amyloidosis, acromegaly and compensatory hypertrophy in a single kidney.

17. Answer C.

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.

18. Answer C.

The rapidity of onset, blood and protein in the urine and new HTN makes the diagnosis very likely to be rapidly progressive glomerulonephritis (RPGN). It is important to clinically suspect RPGN due to the poor prognosis if not adequately treated. Diagnosis is confirmed by renal biopsy but when the clinical suspicion is high, treatment is initiated even while awaiting final report of renal biopsy.
RPGN is characterised histologically by glomerular crescents which are defined as two or more layers of proliferating parietal epithelial cells in Bowman's space (Bowman's space normally has a single layer of parietal and visceral epithelial cells each)
RPGN may be due to:

- Pauciimmune RPGN: Negative staining on IF; causing more than 50% of all RPGN, this group is due to ANCA-associated vasculitis
- Anti-GBM antibody associated RPGN: Positive 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 presence of coarse immune deposits in the glomeruli; about 25% of all RPGN and may be due to lupus (commonest) IgA nephropathy, infection related GN, or MCGN.

Initial therapy consists of pulse methylprednisolone for three days followed by daily oral prednisone and oral or intravenous cyclophosphamide or rituximab

Plasmapheresis is offered if patient has haemoptysis which may be seen in RPGN associated with anti-GBM disease or ANCA vasculitis.

19. Answer E

- Loop diuretics must enter the tubular fluid in order to exert their diuretic effect. Loop diuretics are highly (≥95 percent) protein bound. As a result, they primarily enter the tubular lumen by secretion by the proximal tubule, not by glomerular filtration.
- Because loop diuretics are highly protein bound, severe hypoalbuminemia (<2 g/dL) associated with the nephrotic syndrome may reduce the delivery of diuretic to the renal tubule and therefore, decrease tubular secretion. In addition, filtered albumin in nephrotic patients may bind loop diuretics in the tubular lumen, thereby interfering with their function.
- Some patients have partial or relatively complete resistance to a loop diuretic despite adequate secretion of the diuretic into the tubular fluid. This is due to the compensatory increase in sodium reabsorption in other parts of the tubules when sodium reabsorption is blocked in the TAL by loop diuretics.
- Intravascular depletion caused by loop diuretics leads to activation of the RAS system leading to increased sodium reabsorption in the collecting duct.

20. Answer E

Tubular disorders are more often responsible for renal failure than glomerular pathologies in patients with multiple myeloma. The various renal pathologies seen in this condition are:

Tubular

Light chain cast nephropathy (myeloma kidney): 30-50%

Interstitial nephritis/fibrosis: 20-30%

Acute Tubular Necrosis: 10%

Glomerular

Amyloidosis: 10%

Monoclonal immunoglobulin deposition disease (mostly light chain deposition disease): 5%

Rare: Cryoglobulinemia, MPGN, DDD, Fibrillary GN and immunotactoid glomerulopathy

Others

Hypercalcaemia, Urate nephropathy, Fanconi Syndrome

21. Answer A.

Two third cases have no known association- termed primary MN. Antibody to phospholipase A2 receptor(anti-PLA2R) seen in 70% of primary MN but not in secondary cases.

1/3rd cases are associated with following conditions (commoner conditions in bold):

- Connective tissue disease: **SLE** rheumatoid arthritis
- Infections: **Hepatitis B,** Hepatitis C, schistosomiasis, malaria, filariasis
- Drugs**: Penicillamine**, **NSAIDs, anti-TNF(Infliximab),** gold, captopril
- Tumours (specially in >65 years): **Mostly solid organs malignancies like prostate, lung, or gastrointestinal track**, less often CLL.

22. Answer C

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 for diagnosing obstruction and identify renal calculi, it is less reliable in detecting ureteric stones.

23. Answer C

A tendency to phosphate retention, beginning early in CKD due to a decrease in the filtered phosphate load, is thought to play a central role in the development of secondary hyperparathyroidism and the associated CKD-MBD. Reduction of GFR to less than 70 ml/min leads to a decrease in renal phosphate clearance. The resultant phosphate retention is postulated to be the trigger to the development of CKD-MBD.

Phosphate retention leads to hypocalcaemia and the resultant increase in parathyroid (PTH) secretion by different mechanisms-

- Hypocalcaemia: Whenever the Calcium x Phosphate product is excessively high, both calcium and phosphate began to get deposited at various sites all over the body, leading to lowering of serum calcium levels.

- Decreased formation of calcitriol (1, 25-dihydroxyvitamin D, the active form of vitamin D): Decreased activity of 1-alpha hydroxylase in the deceased kidney leads to reduced formation of calcitriol, and hence reduced intestinal absorption of calcium.

- Increased PTH gene expression: Increased serum phosphate has a direct effect in increasing PTH secretion.

- FGF-23: Under normal circumstances, FGF-23 suppresses PTH secretion by the parathyroid gland. However, among CKD patients, markedly decreased expression of fibroblast growth factor-receptor 1 and klotho protein in the hyperplastic parathyroid gland causes the parathyroid gland to become resistant to the elevated FGF-23 levels. Thus, an elevated level of FGF-23 is not able to suppress PTH production in the CKD patient as it would do in a non-CKD patient.

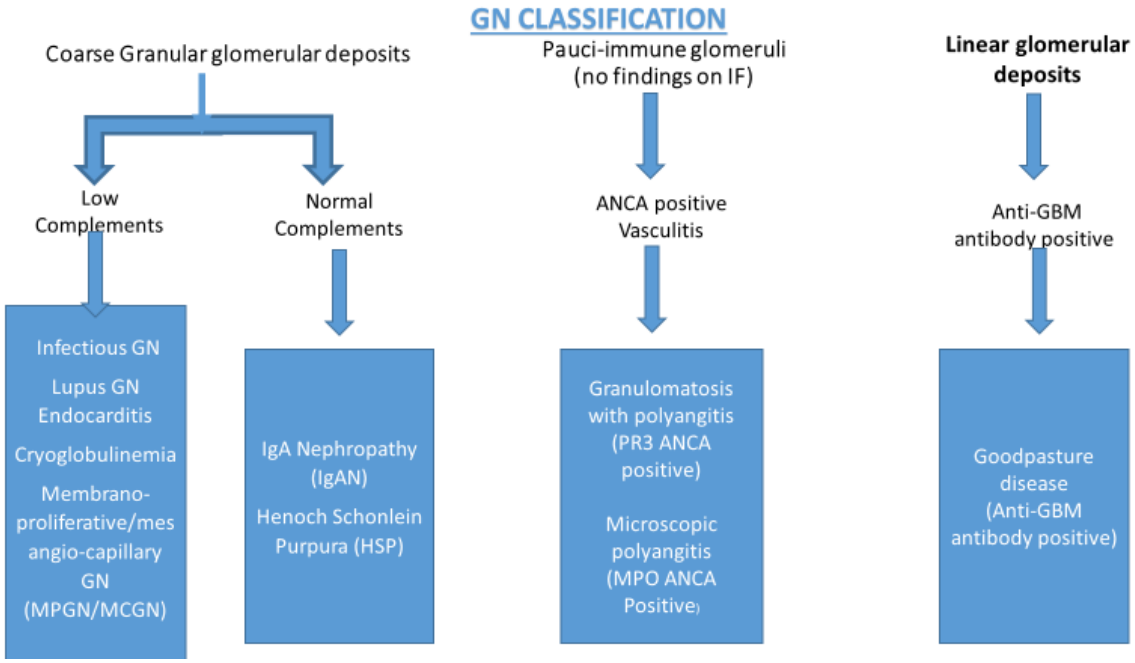
24 Answer E

The answer is '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.**

Differential of GN (This is based on IF findings and you can see the low complement causes include all the other choices in this Q)



25. Answer C.

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 the biopsy.

26. Answer B

- In a DM with microvascular disease like retinopathy and proteinuric renal impairment associated with benign urine sediment, a presumptive diagnosis of diabetic nephropathy can be made with reasonable certainty
- Choice ‘a’ has a high possibility of being rapidly progressive GN and needs renal biopsy diagnosis
- Biopsy evaluation needed in systemic disease like SLE with renal involvement (choice c) where treatment decision will depend on severity of histological abnormalities
- Establishing a diagnosis of myeloma cast nephropathy will modify therapeutic options in a patient with myeloma and AKI and biopsy is essential to guide therapy (choice d)

27. Answer E.

Patients effected with DRA most commonly present with shoulder pain. The amyloid protein in DRA is composed primarily of beta2-microglobulin and the condition is almost exclusively seen in ESRD patients on dialysis. The incidence of DRA is now much lower than had been reported previously due to the increased use of high-flux biocompatible dialyzers with enhanced clearance of beta2-microglobulin. Heart failure (restrictive) and peripheral neuropathy are commonly seen in AL and AA amyloidosis but not in DRA.

28 Answer C.

Primary FSGS typically recurs in a florid fashion, often very early with pronounced proteinuria and is treated with plasmapheresis. MCGN and IGA nephropathy tend to recur in a more progressive fashion, with deterioration in graft function over a period of years. Goodpasture's syndrome typically does not recur post transplantation.

29. Answer C

In most cases, CT can accurately diagnose RCC. It also provides information on function and morphology of the kidneys, tumour extension, venous involvement, lymph node involvement and surrounding structures. A change of more than 15 Hounsfield Units with contrast is significant and suggests a malignant process.

30. Answer D.

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.