Ace Your SCE Exam

European Specialty Examination in Nephrology (ESENeph)Sample MCQs

SCE Recalls Group

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European Specialty Examination in Nephrology (ESENeph) 30 Sample MCQs (2022–2024)

02

Q1

A 56-year-old man is 3 months post–deceased donor kidney transplant for diabetic nephropathy. Induction was basiliximab; maintenance is tacrolimus 3 mg bid (troughs 10–11 ng/mL), mycophenolate mofetil (MMF) 1 g bid, and prednisolone 5 mg/day. He feels well but routine labs show creatinine rising from 1.3 to 2.0 mg/dL over 4 weeks. Urinalysis is bland, with no haematuria or casts. Doppler ultrasound shows normal perfusion and no hydronephrosis. Plasma BK PCR has increased from 3.4×10^3 to 1.2×10^5 copies/mL over 6 weeks. Urine "decoy cells" are present. Allograft biopsy demonstrates tubular epithelial viral cytopathic changes with SV40 (large T-antigen) positivity, interstitial fibrosis is minimal, and there is no tubulitis or intimal arteritis. C4d is negative and there is no microvascular inflammation. Donor-specific antibodies (DSA) are negative. What is the most appropriate immediate management?

Options

A. Start low-dose cidofovir

B. Switch tacrolimus to cyclosporine

C. Reduce immunosuppression (hold/reduce MMF and lower tacrolimus target)

D. Begin leflunomide while maintaining current tacrolimus/MMF

E. Give IVIG 2 g/kg once

Correct Answer

C

Key reasoning

This is classic BK polyomavirus nephropathy (BKPyVAN) with significant viremia and compatible histology in the absence of rejection. The intervention that improves outcomes most consistently is stepwise reduction of immunosuppression—typically reduce/hold the antimetabolite (MMF) and lower the calcineurin inhibitor (CNI) trough to a safer range (e.g., 3–5 ng/mL for tacrolimus), while closely monitoring creatinine and BK PCR. Antivirals and adjuncts have weaker evidence or higher toxicity and are generally reserved for refractory viremia after immunosuppression minimization.

Guideline points

Thresholds that trigger action: persistent plasma BK DNAemia $\geq 10^4$ copies/mL or biopsy-proven BKPyVAN.

First line: decrease total immunosuppressive load—hold/reduce MMF and reduce tacrolimus trough (e.g., to 3–5 ng/mL).

Monitor BK PCR every 2–4 weeks and titrate IS based on virologic response.

If viremia persists: consider leflunomide (levels if available), IVIG as adjunct, or cidofovir in exceptional refractory cases (nephrotoxic; use cautiously).

Be alert for acute rejection after IS reduction—recheck graft function and consider repeat biopsy if creatinine worsens without virologic improvement.

Why distractors are wrong

A/E: Cidofovir and IVIG are not first-line; evidence is limited and cidofovir is nephrotoxic.

B: Switching to cyclosporine does not address total IS burden; reduction is needed.

D: Adding leflunomide without reducing baseline IS misses the primary intervention.

Exam tip

Think “BK = Back off immunosuppression.” Reduce/hold MMF first and bring tacrolimus into a lower target range.

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03

Q2

A 44-year-old woman is post–living donor kidney transplant day 10. She has new tremor and headaches. Blood pressure is 162/96 mmHg (baseline 128/78). Creatinine rose from 1.1 to 2.1 mg/dL over 48 hours. Potassium is 5.4 mmol/L; magnesium is 1.5 mg/dL. Tacrolimus troughs have been 15–16 ng/mL for the past 3 days after starting diltiazem for rate control. Urinalysis is bland (no proteinuria or haematuria). DSA screen is negative.

Allograft biopsy shows isometric fine vacuolization of proximal tubules, arteriolar smooth muscle cell swelling/vasoconstriction changes, no interstitial infiltrate or tubulitis, C4d negative, and no thrombi. What is the best next step?

Options

A. Pulse methylprednisolone 500 mg/day × 3 (treat for T-cell rejection)

B. Plasmapheresis + IVIG (treat for antibody-mediated rejection)

C. Reduce tacrolimus dose to target trough 5–7 ng/mL and optimize BP/volume; review drug interactions

D. Add MMF 1 g bid to intensify immunosuppression

E. Switch tacrolimus immediately to sirolimus

Correct Answer

C

Key reasoning

The clinical picture—high tacrolimus trough, neurotremor, hypertension, hyperkalaemia, hypomagnesaemia—and biopsy with isometric tubular vacuolization supports CNI nephrotoxicity, not rejection. The correct move is to reduce tacrolimus exposure (and correct contributing factors: stop diltiazem interaction, manage blood pressure/volume, replete magnesium). Treating presumed rejection here would add harm without benefit.

Guideline points

CNI toxicity presents with hypertension, tremor, electrolyte disturbances; biopsy: isometric vacuolization ± arteriolar changes, no tubulitis/microvascular inflammation.

First steps: lower CNI dose/target, address DDIs (e.g., diltiazem, azoles, macrolides), treat hypertension (prefer CCBs that don't raise tac levels, or beta-blockers), and optimize intravascular volume.

Avoid early conversion to mTOR inhibitors (E) in the immediate post-op period due to wound-healing and lymphocele risks.

Reassess kidney function and tac trough within 24–48 h after adjustments.

Why distractors are wrong

A/D: No histologic rejection—unnecessary escalation of IS.

B: AMR therapy is inappropriate with negative DSA/C4d and no microvascular inflammation.

E: Immediate mTOR switch on day 10 is risk-laden and unnecessary; dose reduction is adequate.

Exam tip

“Vacuoles + high tac = turn the tac down.” Always check for drug–drug interactions.

04

Q3

A 48-year-old renal transplant recipient with a history of anaphylaxis to sulfonamides needs Pneumocystis jirovecii pneumonia (PJP) prophylaxis for the first 6–12 months post-transplant. Baseline labs: Hb 12.6 g/dL, normal reticulocytes, LDH normal, bilirubin normal. G6PD status is unknown. Liver tests are normal. He cannot tolerate inhaled medications due to severe reactive airway disease. Which regimen is most appropriate?

Options

A. Dapsone 100 mg daily after confirming normal G6PD

B. Atovaquone 1,500 mg daily with food + check G6PD

C. Monthly inhaled pentamidine + daily folic acid

D. Azithromycin 500 mg three times weekly

E. Fluconazole 200 mg daily

Correct Answer

A

Key reasoning

TMP-SMX is first-line for PJP prophylaxis but contraindicated here. Dapsone 100 mg daily is an established alternative; G6PD testing is mandatory before initiation to avoid haemolysis and methemoglobinaemia. Atovaquone is a valid alternative when dapsone is unsuitable, but it does not require G6PD testing—the stem's "+ check G6PD" is a deliberate trap. Inhaled pentamidine can be used when oral options are intolerable but provides less systemic protection (e.g., against toxoplasma/nocardia) and is not ideal in severe airway hyperreactivity.

Guideline points

Alternatives to TMP-SMX:

Dapsone 100 mg daily (only if G6PD normal; monitor for haemolysis/metHb).

Atovaquone 1,500 mg daily with fatty meal (GI tolerance, cost; no G6PD issue).

Inhaled pentamidine 300 mg monthly (less systemic coverage; bronchospasm risk).

Typical duration: 6–12 months, longer if augmented immunosuppression (e.g., after rejection treatment).

Counsel that non–TMP-SMX regimens do not protect well against nocardiosis/toxoplasmosis.

Why distractors are wrong

B: Atovaquone is reasonable, but G6PD testing is not required—option wording is inaccurate.

C: Acceptable but inferior systemic coverage and problematic in severe airway disease.

D/E: Do not prevent PJP.

Exam tip

For sulfa anaphylaxis, Dapsone (if G6PD normal) is a strong first choice; otherwise atovaquone with food.

05

Q4

A 32-year-old man with biopsy-proven primary FSGS (steroid-dependent, calcineurin-sensitive) receives a pre-emptive living-related kidney transplant. He previously had nephrotic-range proteinuria and no diabetes. Donor–recipient HLA mismatch is low; cold ischaemia time 2 hours. Within 48 hours post-transplant, his urine protein–creatinine ratio is >8 g/g and serum albumin has fallen from 38 to 25 g/L. Serum creatinine is stable at 1.1 mg/dL. Graft Doppler is normal. Biopsy shows podocytopathy with diffuse foot process effacement but no rejection or vascular lesions. PLA2R is negative; complements are normal; DSA negative. What is the best next step?

Options

A. Start ACE inhibitor and observe

B. Increase tacrolimus target to 10–12 ng/mL

C. Therapeutic plasma exchange (TPE) promptly

D. Oral cyclophosphamide 2 mg/kg/day

E. High-dose loop diuretics to control oedema

Correct Answer

C

Key reasoning

Abrupt heavy proteinuria within days of transplantation with compatible biopsy in a patient with primary FSGS strongly indicates early recurrence driven by a circulating permeability factor. Therapeutic plasma exchange initiated promptly (often daily or every other day initially) improves remission rates and graft survival. Rituximab is commonly added after initial TPE or for partial responders. RAAS blockade and diuretics are supportive but do not address the underlying pathophysiology.

Guideline points

Risk factors for recurrence: younger age, rapid native disease progression, prior graft loss from FSGS, certain genetic backgrounds (non–monogenic primary FSGS).

First-line: TPE (5–10 sessions initially, tailored to response), then consider rituximab (e.g., 375 mg/m² ×1–2).

Continue baseline IS; avoid delays —earlier TPE = better chance of remission.

RAAS blockade for proteinuria adjunctively; monitor albumin, lipids, thrombosis risk.

Why distractors are wrong

A/E: Symptomatic measures only; do not halt podocyte injury.

B: Higher tacrolimus may reduce proteinuria marginally but won't neutralize circulating factor.

D: Cyclophosphamide is not first-line in early recurrent FSGS post-transplant.

Exam tip

"Nephrotic within 24–72 h post-Tx + prior primary FSGS" → start TPE now; add rituximab if response is incomplete.

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06

Q5

On post-transplant day 5, a 58-year-old man after a deceased donor transplant develops rising creatinine (1.1 → 2.4 mg/dL), oedema, and new difficult-to-control hypertension. He had low-level pre-formed anti-DQ DSA pre-op with a negative flow crossmatch. Current IS: tacrolimus (trough 7–8 ng/mL), MMF 1 g bid, prednisolone 5 mg/day. Urinalysis shows mild proteinuria and microscopic haematuria. Repeat immunology reveals rising class II DSA (DQ) MFI. Allograft biopsy shows glomerulitis (gl–2) and peritubular capillaritis (ptcl–2) with C4d positivity in peritubular capillaries; no significant tubulitis or intimal arteritis. What is the best initial treatment?

Options

A. High-dose methylprednisolone alone

B. Plasmapheresis (PLEX) plus IVIG

C. Rituximab monotherapy

D. Bortezomib monotherapy

E. Convert tacrolimus to belatacept

Correct Answer

B

Key reasoning

Biopsy and serology fulfil AMR criteria: DSA present, microvascular inflammation (g/ptc), and C4d positivity. The best initial therapy is antibody removal with plasmapheresis plus immune modulation with IVIG (often alongside high-dose steroids). Additional agents—rituximab (B-cell depletion), bortezomib (plasma cell targeting), or complement inhibitors—are used for severe or refractory AMR but are not typically first-line alone.

Guideline points

Diagnostic triad for AMR: DSA + microvascular injury (g/ptc) ± C4d with graft dysfunction.

Initial regimen:

PLEX (e.g., every other day for 4–6 sessions),

IVIG (either 100 mg/kg after each PLEX or 2 g/kg total divided),

High-dose steroids (e.g., methylprednisolone 500–1000 mg/day × 3) as part of the bundle.

Consider rituximab (e.g., 375 mg/m²) if high-risk or poor early response; bortezomib or eculizumab in refractory/early hyperacute-like cases.

Maintain adequate baseline IS (avoid under-immunosuppression during treatment) and manage BP/volume.

Monitor DSA titres/MFI and creatinine to gauge response.

Why distractors are wrong

A: Steroids alone are insufficient in AMR.

C/D: Reasonable adjuncts, but not adequate as monotherapy initially.

E: Changing maintenance agent does not address circulating antibodies acutely.

Exam tip

AMR = “remove and neutralize”: PLEX + IVIG up front; escalate with rituximab or other agents if the response is suboptimal.

07

Q6

A 60-year-old woman has recurrent UTIs with Proteus mirabilis and a staghorn calculus occupying the right renal pelvis and calyces. She has intermittent fever and flank pain; creatinine is stable; the left kidney is normal. Urinalysis: alkaline urine with struvite crystals. What is the most appropriate definitive management?

Options

A. Chronic suppressive antibiotics only

B. Urinary acidification and hydration; avoid surgery

C. Complete stone removal with percutaneous nephrolithotomy (possibly staged) plus culture-directed antibiotics; remove all infected stone material

D. ESWL monotherapy

E. Observe unless obstruction occurs

Correct Answer

C

Key reasoning

Struvite (infection) stones form biofilm-laden staghorn calculi that perpetuate infection. Cure requires complete stone clearance—usually PCNL, sometimes staged—with culture-guided antibiotics. ESWL alone is ineffective for large staghorns; suppressive antibiotics without clearance fail.

Why distractors are wrong

A/B/E: Won't eradicate nidus → ongoing infection/renal damage.

D: Poor efficacy for staghorns.

Exam tip

Staghorn struvite = PCNL + antibiotics, aim complete clearance.

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08

Q7

A 47-year-old woman has resistant hypertension (on ACE inhibitor, amlodipine, thiazide) and spontaneous hypokalaemia (K^+ 3.0 mmol/L). BP averages 158/94. Plasma aldosterone 24 ng/dL, renin suppressed (ARR elevated). CT adrenal shows a 1.8-cm right adrenal nodule. She is 47, with no clear unilateral features on labs. What is the most appropriate next step to guide definitive therapy?

Options

A. Start spironolactone and stop evaluation

B. Adrenal venous sampling (AVS) to lateralize aldosterone secretion before surgery

C. Proceed directly to right adrenalectomy based on CT

D. PET scan to confirm functionality

E. Genetic testing for Liddle syndrome

Correct Answer

B

Key reasoning

In primary aldosteronism, imaging does not reliably distinguish unilateral adenoma from bilateral hyperplasia, especially in patients >35–40 years. AVS is the gold standard to determine lateralization and select candidates for adrenalectomy vs medical therapy (MRA) for bilateral disease. Start MRA for BP/K^+ control during workup, but AVSremains the key decision test.

Why distractors are wrong

A: Treats empirically but misses potential cure.

C: CT alone may mislead; many nonfunctioning incidentalomas exist.

D: PET not standard.

E: Liddle has low renin and low aldosterone.

Exam tip

PA with adrenal nodule in adults → do AVS before surgery (unless young with clear unilateral disease).

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09

Q8

A 63-year-old man on haemodialysis via a 5-year-old left brachiocephalic AV fistula has progressively enlarging aneurysmal segments with shiny, thinned skin and a recent episode of spontaneous oozing after scratching ("sentinel bleed"). Exam: large saccules with areas difficult to cannulate; distal pulses intact. What is the most appropriate management?

Options

A. Continue cannulation away from the thin area and observe

B. Urgent vascular surgery evaluation for access revision (aneurysmorrhaphy with patch/graft interposition) or ligation with new access creation; avoid cannulation over thin skin

C. Percutaneous thrombin injection into the aneurysm sac

D. Tight tourniquet banding by dialysis staff to reduce size

E. Immediate ligation without planning alternate access

Correct Answer

B

Key reasoning

Aneurysmal AVF with skin thinning or sentinel bleeding risks rupture. Requires prompt surgical intervention: revision (aneurysmorrhaphy/graft interposition) or ligation with planned new access. Meanwhile, do not cannulate over compromised skin. Thrombin injection is for pseudoaneurysms of grafts/catheters, not degenerative AVF aneurysms.

Why distractors are wrong

A/D: Unsafe; risk catastrophic bleed.

C: Not appropriate for true AVF aneurysm.

E: Don't sacrifice access without a plan for alternative access.

Exam tip

AVF thin shiny skin / sentinel bleed → urgent surgical revision.

10

Q9

A 55-year-old man on CAPD for 8 years presents with recurrent ultrafiltration failure, weight loss, early satiety, and intermittent small-bowel obstruction symptoms. CT shows peritoneal thickening, calcification, and loculated fluid; PET reveals adhesions. He had multiple prior bacterial peritonitis episodes. What is the most appropriate management?

Options

A. Increase glucose concentration of PD fluid and continue PD

B. Stop PD and transition to haemodialysis; provide nutritional support; initiate medical therapy (e.g., corticosteroids ± tamoxifen); refer to an experienced centre for consideration of surgical peritonectomy/adhesiolysis if refractory

C. IP fibrinolytics and continue PD indefinitely

D. Start long-term antibiotics

E. Observe; this is self-limited

Correct Answer

B

Key reasoning

This is encapsulating peritoneal sclerosis (EPS), a late PD complication. Management is cessation of PD, nutritional support, and medical therapy (steroids ± tamoxifen). In specialized centres, surgical enterolysis/peritonectomy may improve obstruction in refractory cases. Continuing high-glucose PD worsens injury.

Why distractors are wrong

A/C/D/E: Do not address the fibrotic encapsulation; risk progression and malnutrition.

Exam tip

EPS → stop PD, support nutrition, steroids±tamoxifen; consider surgery at expert centres.

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II

Q10

An 82-year-old nursing-home resident with dementia presents with hypernatraemia Na^+ 163 mmol/L after several days of poor intake during a heatwave. He is mildly confused but arousable; vitals stable; exam suggests hypovolaemia (dry mucosa, orthostasis). Glucose normal, BUN elevated. What is the most appropriate initial and ongoing correction strategy?

Options

A. Restore intravascular volume first with isotonic crystalloid, then correct free-water deficit with enteral water or IV D5W to lower Na^+ by no more than ~10 mmol/L per 24 h; monitor Na^+ q4–6 h and adjust

B. Rapidly correct Na^+ by 20 mmol/L in first 6 hours with D5W

C. Use 0.9% saline alone until Na^+ normalizes

D. Start desmopressin to increase renal water retention

E. Restrict fluids to avoid cerebral oedema

Correct Answer

A

Key reasoning

In chronic hypernatraemia from water deficit, first resuscitate with isotonic fluid to restore perfusion, then administer free water (enteral preferred; otherwise IV D5W) to correct slowly (generally ≤10–12 mmol/L/day). Frequent monitoring prevents overcorrection or undercorrection. DDAVP is not indicated unless diabetes insipidus.

Why distractors are wrong

B: Overly rapid shift risks cerebral oedema.

C: Normal saline maintains or worsens hypernatraemia after initial resuscitation.

D: No evidence of DI.

E: Opposite of needed therapy.

Exam tip

Chronic hyperNa$^+$ → volume first, then free water, correct slowly with close monitoring.

12

Q11

A 58-year-old man with decompensated alcoholic cirrhosis (ascites, varices) is admitted with AKI (creatinine 2.6 mg/dLfrom 1.1 a month ago). He is jaundiced but afebrile, with tense ascites, MAP 68–72 mmHg, no overt bleeding, and no nephrotoxin exposure. Urinalysis is bland; urine Na^+ <10 mmol/L. After albumin 1 g/kg over 48 h, diagnostic paracentesis excludes SBP, and creatinine rises to 3.0 mg/dL. He remains hypotensive despite careful fluid optimization. He has known ischemic heart disease with recent NSTEMI and peripheral vascular disease, so the team is concerned about splanchnic vasoconstrictors causing ischemia. ICU can admit him today. What is the most appropriate initial vasoconstrictor strategy?

Options:

A. Midodrine + octreotide + albumin on the ward

B. Norepinephrine infusion in ICU with continued albumin and diuretic cessation

C. High-dose terlipressin boluses on the ward

D. Immediate TIPS

E. Start ACE inhibitor to improve renal perfusion

Answer: B

Key reasoning:He meets HRS-AKI criteria (cirrhosis with ascites, AKI not responsive to albumin, no shock/sepsis/nephrotoxins, bland urine, low urine Na^+). When terlipressin is contraindicated or risky (recent ischemia, peripheral vascular disease) or ICU is available, norepinephrine is an accepted first-line alternative to restore effective arterial volume. It's titratable, monitored in ICU, and paired with albumin and diuretic withdrawal. Midodrine—octreotide is less effective and slower; TIPS is not a first step in an unstable patient; ACE inhibitors worsen renal perfusion in HRS.

Why distractors are wrong:A: Inferior efficacy to norepinephrine/terlipressin in HRS-AKI.C: Terlipressin poses ischemia risk here.D: Consider only after stabilization and careful selection.E: RAAS blockade decreases GFR in this setting.

Exam tip:HRS-AKI with vascular disease → ICU norepinephrine + albumin is a strong alternative to terlipressin.

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Q12

A 45-year-old kidney transplant recipient (8 months post–deceased donor) has creatinine creeping from 1.3 → 1.8 mg/dLover 6 weeks. He is asymptomatic. Immunosuppression: tacrolimus (trough 6–7 ng/mL), mycophenolate mofetil (MMF) 1 g bid, prednisone 5 mg. Urinalysis: bland; protein/creatinine 0.3 g/g. Ultrasound: normal perfusion, no hydronephrosis. BK viral load in plasma is 2.8×10^4 copies/mL on two tests 2 weeks apart; urine load is very high.

Donor-specific antibodies negative. What is the most appropriate management now?

Options:

A. Start leflunomide and continue current immunosuppression

B. Reduce immunosuppression in a stepwise fashion (hold/reduce MMF first; modestly lower CNI target), monitor BK PCR q2–4 weeks; biopsy if creatinine continues to rise or if high-level viremia persists

C. Switch tacrolimus to sirolimus and increase dose to target 10–12 ng/mL

D. Begin IV cidofovir

E. Treat as acute rejection with pulse steroids

Answer: B

Key reasoning:This is BK viremia with graft dysfunction. First-line therapy is immunosuppression reduction, typically holding/reducing the antimetabolite (MMF) and lowering CNI trough modestly, with close viral-load monitoring. A biopsy is warranted for persistent/worsening dysfunction to assess BK nephropathy vs rejection. Routine leflunomide/cidofovir lacks robust benefit and has toxicity. Treating as rejection without evidence will worsen BK replication.

Why distractors are wrong:A/D: Second/third-line and toxic; not initial.C: mTOR alone doesn't solve viremia; higher levels may harm.E: Steroids fuel viral replication.

Exam tip:Rising BK PCR → back off IS first, follow PCR, biopsy if not improving.

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Q13

A 63-year-old man presents with subacute fatigue, 7-kg weight loss, mild renal insufficiency (creatinine 1.9 mg/dL, baseline 1.1), and sterile pyuria with low-grade proteinuria (UPCR 0.7 g/g). He has painless salivary gland enlargement, submandibular swelling, and pruritic plaques; CT shows diffuse pancreatic enlargement ("sausage-shaped" pancreas). Serum IgG4 is elevated. Kidney biopsy (done for diagnostic clarity) shows dense IgG4-positive plasma cell–rich tubulointerstitial nephritis with storiform fibrosis and obliterative phlebitis; glomeruli largely spared. What is the most appropriate initial treatment?

Options:

A. Observation; this often remits

B. Glucocorticoids (e.g., prednisone ~0.6 mg/kg/day) with a slow taper

C. High-dose cyclophosphamide

D. Plasmapheresis

E. Antibiotics for occult infection

Answer: B

Key reasoning:This is IgG4-related kidney disease (IgG4-RKD), typically tubulointerstitial nephritis within systemic IgG4-related disease. First-line therapy is glucocorticoids, which usually normalize function and shrink lesions. Many require slow taper and relapse prevention (rituximab or AZA/MMF). Cytotoxic agents and PLEX are not indicated; antibiotics are irrelevant.

Why distractors are wrong:A: Risk of irreversible fibrosis without therapy.C/D: Not standard or necessary.E: There's no infection.

Exam tip:IgG4-RKD → steroids first, plan for relapse prevention if needed.

15

Q14

A 38-year-old woman with systemic lupus erythematosus has recurrent arterial and venous thromboses and livedo reticularis. She presents with AKI (creatinine 2.2 mg/dL), malignant-range hypertension, and proteinuria 2.5 g/day. Biopsy shows thrombotic microangiopathy with fibrin thrombi in arterioles and glomeruli; immunofluorescence lacks immune complexes. She is triple positive for antiphospholipid antibodies (lupus anticoagulant, high-titre aCL IgG, anti–β2GPI IgG). What is the most appropriate long-term antithrombotic strategy?

Options:

A. Aspirin alone

B. Warfarin with target INR 2–3 and add low-dose aspirin if arterial events; avoid DOACs in triple-positive APS

C. Apixaban 5 mg bid

D. Clopidogrel monotherapy

E. No anticoagulation—risk of bleeding outweighs benefit

Answer: B

Key reasoning:This is antiphospholipid syndrome nephropathy. Triple-positive APS has high thrombotic risk; warfarin remains standard, with higher-intensity INR considered for arterial recurrence. DOACs are not recommended in triple-positiveAPS due to higher recurrence in trials. Low-dose aspirin is reasonable adjunct for arterial disease. Immunosuppression targets SLE activity but doesn't replace anticoagulation.

Why distractors are wrong:A/D/E: Insufficient protection given risk.C: DOACs underperform in triple-positive APS.

Exam tip:APS (triple positive) → warfarin (± aspirin for arterial); avoid DOACs.

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Q15

A 72-year-old woman with CKD G4 (eGFR 24), frailty, vertebral compression fracture, and DXA T-score -3.1 asks about osteoporosis treatment. Labs: 25(OH)D 22 ng/mL, PTH 110 pg/mL (mildly elevated for CKD 4), Ca^{2+} 2.28 mmol/L, PO_4 1.4 mmol/L, ALP normal. She has no evidence of high-turnover bone disease (PTH not markedly high, ALP normal) and no history of hypocalcaemia. What is the most appropriate antiresorptive approach?

Options:

A. Oral alendronate for 5 years; CKD4 is a contraindication

B. Denosumab with careful Ca^{2+}/PO_4/Mg monitoring and vitamin D repletion

C. Teriparatide

D. No therapy; fracture risk tools are invalid in CKD

E. Zoledronic acid IV annually

Answer: B

Key reasoning:In CKD 4–5, bisphosphonates are often avoided or used cautiously due to accumulation and adynamic bone concerns. Denosumab is not renally cleared and reduces fractures in CKD, but it can cause hypocalcaemia, so ensure vitamin D repletion, adequate calcium intake, and lab monitoring after each dose. Teriparatide is contraindicated with unexplained high ALP or bone metastasis, and data in CKD 4–5 require caution; here the phenotype is not clearly high-turnover but antiresorptive is reasonable. Zoledronic acid is nephrotoxic and avoided in CKD 4.

Why distractors are wrong:A/E: Bisphosphonates (esp. IV) risky in CKD 4.C: Anabolic therapy requires careful selection; not first-line here.D: High fracture risk warrants treatment.

Exam tip:CKD 4 + fragility fracture → denosumab is often preferred; monitor Ca^{2+}/PTH closely.

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Q16

A 51-year-old man has weight loss, painful mononeuritis multiplex, livedo, abdominal pain after meals, and malignant hypertension. Serum creatinine 1.6 mg/dL. HBsAg is negative. ANCA negative; complements normal. CT angiography reveals multiple small renal and mesenteric artery microaneurysms and irregular stenoses; urinalysis shows mild proteinuria without RBC casts. What is the most appropriate induction therapy?

Options:

A. High-dose steroids only

B. High-dose glucocorticoids plus cyclophosphamide for systemic polyarteritis nodosa; add antiviral/PLEX only if Hep B–associated

C. Rituximab monotherapy first-line

D. Plasma exchange alone

E. Mycophenolate monotherapy

Answer: B

Key reasoning:Medium-vessel polyarteritis nodosa causes ischemic renal disease (microaneurysms/stenoses) rather than GN. With HBV-negative disease, induction is steroids + cyclophosphamide. If HBV-positive, antivirals + short steroid course ± PLEX is preferred.

Why distractors are wrong:A/E: Insufficient for extensive systemic PAN.C: RTX not standard first-line in PAN.D: PLEX alone is not disease-modifying.

Exam tip:Microaneurysms + neuropathy + HTN, ANCA-/HBV- → PAN → steroids + CYC.

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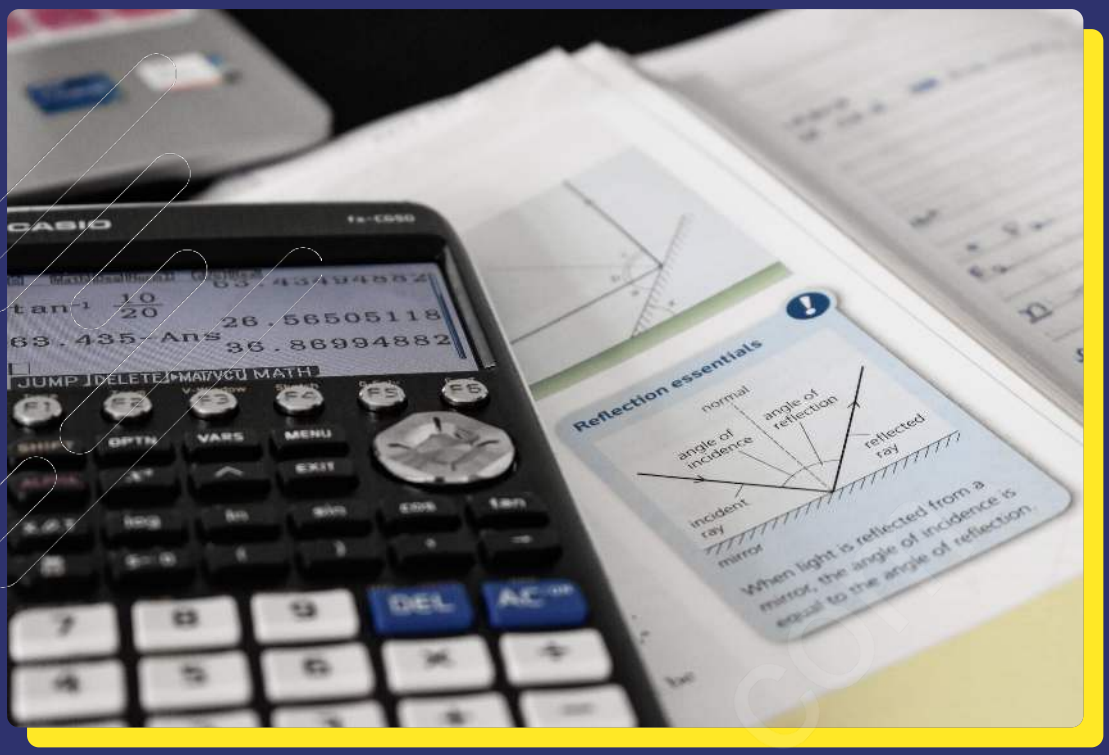
Q17



A 62-year-old kidney transplant recipient on tacrolimus/MMF presents with tremor, headache, hypertension, and rising creatinine 1.3 → 2.1 mg/dL over 10 days. Two weeks ago he started voriconazole for aspergillosis. Tacrolimus trough today is 23 ng/mL. Potassium is 5.7 mmol/L. What is the most appropriate immediate management?

Options:

A. Continue tacrolimus; voriconazole lowers levels

B. Hold or drastically reduce tacrolimus with close trough monitoring.

C. Stop all antifungals to normalize tacrolimus

D. Give kayexalate only and recheck in a week

E. Switch tacrolimus to sirolimus at high dose today

Answer: B

Key reasoning:Azoles (voriconazole/posaconazole) are strong CYP3A4 inhibitors → tacrolimus toxicity (neuro, nephro, hyperK, HTN). Immediate action is to hold/markedly reduce tacrolimus and monitor troughs frequently. If antifungal alternatives exist (echinocandin), switch; otherwise co-administer with large tacro dose reductions.

Why distractors are wrong:A: Directionally wrong.C: Stopping antifungal in invasive disease is unsafe.D: Treats K^+ but ignores root cause.E: mTOR inhibitors have their own risks; not an urgent swap without plan.

Exam tip:New azole + rising tacro level/Cr → CYP3A4 interaction → slash tacro dose/hold + monitor.

19

Q18

A 74-year-old man develops AKI and livedo reticularis one week after coronary angiography. Exam shows blue toeswith intact pulses. Labs: creatinine 2.6 mg/dL (from 1.1), eosinophilia, low complement C3, and urinalysis bland. What is the most appropriate management plan?

Options:A. Start high-intensity anticoagulation indefinitely

B. Supportive care for cholesterol atheroembolic renal disease

C. Immediate plasmapheresis for 7 sessions

D. High-dose cyclophosphamide

E. Thrombolysis of distal arteries

Answer: B

Key reasoning:Post-procedural cholesterol crystal embolization causes AKI, livedo/blue toes, eosinophilia, and low C3 with bland UA. Treatment is supportive: avoid repeat vascular trauma and unnecessary anticoagulation, start statin, and manage BP/complications. Immunosuppression/PLEX lack consistent benefit.

Why distractors are wrong:A/E: Worsen embolization risk or are futile.C/D: Not evidence-based first-line.

Exam tip:After cath: AKI + eosinophilia + livedo → atheroemboli → supportive + statin; avoid further triggers.

20

Q19

A 58-year-old man with gout has recurrent radiolucent stones. 24-hour urine shows low urine volume (1.2 L/day), uric acid 1,100 mg/day, and urine pH 5.2. eGFR 56. What is the most effective preventive strategy?

Options:A. Thiazide diuretic and low-calcium diet

B. Increase fluid intake to target urine ≥2.5 L/day, institute urine alkalinization with potassium citrate to pH 6.5–7.0.

C. Urinary acidification and cranberry extract

D. Calcium supplementation with meals

E. Stop RAAS inhibitor to raise urine pH

Answer: B

Key reasoning:Uric acid stones form in acidic urine and with hyperuricosuria. Prevention is high fluid, alkalinize urine to ~6.5–7.0(potassium citrate), dietary purine/fructose reduction, weight loss, and allopurinol for persistent hyperuricosuria. Thiazides target calcium stones, not uric acid.

Why distractors are wrong:A/C/D/E: Misaligned with uric acid pathophysiology.

Exam tip:Radiolucent stone + pH ~5 → alkalinize (K-citrate) + fluids ± allopurinol.

21

Q20

A 64-year-old man starts hemodialysis with a new dialyzer. Minutes into the first session, he develops generalized pruritus, wheeze, hypotension, and flushing. There is no fever. Staff stop the treatment. He is on an ACE inhibitor for heart failure. What is the most appropriate acute and preventive management?

Options:A. Continue dialysis at lower blood flow; give diphenhydramine only

B. Treat as a Type A dialyzer reaction.

C. Give vancomycin and resume dialysis

D. Switch to peritoneal dialysis permanently

E. No precautions needed at next session; this was anxiety

Answer: B

Key reasoning:Immediate anaphylactoid reactions occur with ETO-sterilized dialyzers or AN69 + ACEi (bradykinin-mediated). Acute care is anaphylaxis protocol and stop the exposure. Prevention: change membrane/sterilization method and review ACEi use with AN69. Type B reactions are complement-mediated and milder later in treatment.

Why distractors are wrong:A/E: Unsafe minimization.C: Not infectious.D: Unnecessary if preventive steps work.

Exam tip:Early wheeze/hypotension on first use → Type A reaction → epi + new dialyzer type (and reconsider ACEi).

22

Q21

A 29-year-old woman had an uncomplicated vaginal delivery 8 days ago. She returns with headache, visual blurring, and oliguria. BP 156/98. Exam: mild pedal edema, no RUQ tenderness, no neuro focality. Labs: platelets 78×10^9/L, Hb 8.9 g/dL with schistocytes, LDH high, bilirubin mildly elevated, creatinine 3.1 mg/dL (baseline 0.7), AST/ALT near normal, haptoglobin low. UA: RBCs 2+, protein 1+. Coombs negative. ADAMTS13 is pending. Obstetric team suspects a pregnancy-related thrombotic microangiopathy. What is the most appropriate initial disease-directed therapy?

Options:

A. Magnesium sulfate and expedited delivery

B. High-dose steroids and observe for 48 h

C. Start complement inhibition with eculizumab

D. Daily therapeutic plasma exchange as routine for all pregnancy TMAs

E. Begin rituximab

Answer: C

Key reasoning:On postpartum day 8 with AKI + thrombocytopenia + MAHA and normal LFTs, pre-eclampsia/HELLP is less likely (usually antepartum/peripartum with transaminitis). TTP remains a differential, but postpartum complement-mediated aHUS is classic; early eculizumab prevents irreversible kidney injury. If ADAMTS13 returns severely deficient, pivot to TTP management (PLEX + steroids ± caplacizumab). Delivery is already done.

Why distractors are wrong:A: Delivery is not the issue postpartum.B: Steroids alone won't halt aHUS.D: PLEX isn't standard for aHUS and delays targeted therapy.E: Not first-line for pregnancy TMAs.

Exam tip:Postpartum TMA with AKI and normal LFTs → think aHUS → eculizumab early.

23

Q22

A 67-year-old woman on thrice-weekly haemodialysis has bone pain and a new low-trauma wrist fracture. Labs (averaged over 3 months): PTH 65 pg/mL (low–normal for ESKD), Ca^{2+} 2.60 mmol/L, PO_4 1.2 mmol/L, alkaline phosphatase low–normal. She takes calcium acetate, calcitriol, and dialyzes on 1.5 mmol/L (3.0 mEq/L) calcium bath. Coronary CT shows vascular calcification. What is the most appropriate bone–mineral management change?

Options:

A. Increase calcitriol to raise bone turnover

B. Suspected adynamic bone disease: stop calcium-based binders, switch to sevelamer/lanthanum

C. Start cinacalcet to suppress PTH further

D. Add IV bisphosphonate monthly

E. Keep regimen; fracture risk is unrelated to dialysis calcium

Answer: B

Key reasoning:Low PTH in ESKD with high calcium, low ALP, fracture, and calcifications suggests adynamic bone disease(oversuppressed turnover). Reduce calcium load (binders and dialysate), hold/limit active vitamin D, and permit PTH to rise to restore turnover. Cinacalcet worsens suppression; bisphosphonates don't fix ABD physiology and carry risk in ESKD.

Why distractors are wrong:A/C: Further suppress turnover.D: Not first-line and potentially harmful.E: Dialysate Ca is central to calcium balance.

Exam tip:ESKD + low PTH + high Ca + fracture → adynamic bone → lower calcium & vit D, raise PTH target.

24

Q23

A 58-year-old man on haemodialysis via a tunnelled right IJ catheter presents with fever and rigors during dialysis. Blood cultures from the dialysis unit and periphery grow methicillin-sensitive Staphylococcus aureus in both sets. Echo is pending. He feels better after fluids and antipyretic. What is the most appropriate access/infection management?

Options:

A. Attempt catheter salvage with antibiotic lock and keep using it

B. Remove the catheter promptly, begin IV anti-staphylococcal therapy (e.g., cefazolin for MSSA) for at least 4 weeks

C. Exchange over guidewire and shorten antibiotics to 7–10 days

D. Treat with oral dicloxacillin for 2 weeks and continue catheter

E. Wait for echo; if no vegetations, no antibiotics required

Answer: B

Key reasoning:S. aureus catheter-related bloodstream infection mandates catheter removal, parenteral antibiotics, and evaluation for metastatic infection. Salvage is unsafe with S. aureus. Duration is ≥4 weeks (longer if complications). Reinsert access after culture clearance.

Why distractors are wrong:A/C/D/E: Under-treat a high-risk bacteremia and risk relapse/endocarditis.

Exam tip:HD catheter + S. aureus bacteremia → pull the line + IV antibiotics ≥4 wks + echo.

25

Q24

A 49-year-old man with advanced CKD presents with pleuritic chest pain and pericardial friction rub. He is volume overloaded, has severe uremic symptoms, and no signs of tamponade. ECG: diffuse ST–T changes. Echo: moderate pericardial effusion without hemodynamic compromise. What is the most appropriate primary therapy?

Options:

A. High-dose NSAIDs and colchicine only

B. Initiate urgent dialysis (or intensify regimen) as first-line therapy for uremic pericarditis

C. Start full-dose anticoagulation due to effusion

D. Routine pericardiocentesis now

E. Low-dose steroids as monotherapy

Answer: B

Key reasoning:Uremic pericarditis responds to dialysis initiation/intensification; effusions often regress. Anticoagulation increases hemopericardium risk. Drain only for tamponade or non-response.

Why distractors are wrong:A/E: Adjuncts at best; not definitive in uremia.C: Hazardous.D: Invasive without indication.

Exam tip:Uremic pericarditis/effusion → dialyze, avoid anticoagulation; tap only for tamponade.

26

Q25

A 46-year-old man is found confused after working in a garage. He complains of "snowfield" vision. Vitals: RR 30, BP 138/84. Labs: pH 7.21, HCO_3^- 10 mmol/L, anion gap 28, serum osmolality 340 mOsm/kg, ethanol 0, osmolar gap elevated, creatinine 1.3. He admits to drinking from an unlabeled solvent bottle. What is the most appropriate immediate management?

Options:

A. Large-volume normal saline and observe

B. Start fomepizole, administer bicarbonate, and initiate hemodialysis

C. N-acetylcysteine infusion

D. High-dose thiamine only

E. Charcoal and whole-bowel irrigation

Answer: B

Key reasoning:Methanol → formic acid toxicity: visual symptoms, AG acidosis, osmolar gap. Treat with alcohol dehydrogenase blockade (fomepizole), bicarbonate, and hemodialysis to remove methanol/formate and correct acidosis; add folinic acid to enhance formate metabolism.

Why distractors are wrong:A: Delays life-saving therapy.C/D/E: Wrong toxidrome/ineffective.

Exam tip:AG acidosis + visual complaints + osm gap → methanol → fomepizole + dialysis + bicarbonate.

27

Q26

A 69-year-old man with CKD G4 (eGFR 27) and resistant hypertension (home BP 154/88 on lisinopril 40 mg, amlodipine 10 mg, and furosemide 80 mg/day) asks about next steps. K^+ 4.3, Na^+ 139, HCO_3^- 22. He denies orthostatic symptoms. What is the most appropriate antihypertensive addition?

Options:

A. Spironolactone 50 mg/day immediately

B. Add chlorthalidone (e.g., 12.5 mg daily) with careful monitoring (Na^+, K^+, Cr, BP)

C. Switch furosemide to hydrochlorothiazide 12.5 mg

D. Double amlodipine to 20 mg/day

E. Alpha-blocker only at bedtime

Answer: B

Key reasoning:Evidence supports chlorthalidone efficacy in CKD stage 4 for BP control and albuminuria reduction (often synergisticwith a loop diuretic). Spironolactone carries hyperkalaemia risk in CKD G4; if used, it requires concurrent K^+ management. HCTZ is weak in low eGFR; maxing amlodipine offers limited incremental benefit.

Why distractors are wrong:A: Hyperkalaemia risk without a plan to mitigate.C: HCTZ underperforms in CKD G4.D/E: May help modestly but less effective than targeted diuretic strategy.

Exam tip:Resistant HTN in CKD G4 → consider chlorthalidone (with labs/volume monitoring).

28

Q27

A 21-year-old man develops cola-colored urine, edema, and hypertension 2 weeks after a streptococcal pharyngitis. Labs: Cr 1.6 mg/dL (baseline 0.8), C3 low, C4 normal, ASO titer elevated. UA: dysmorphic RBCs, RBC casts, mild proteinuria. He is afebrile and appears well. What is the most appropriate management?

Options:

A. High-dose steroids for 3 months

B. Supportive therapy: salt restriction, loop diuretic for edema/BP control, RAAS blockade if proteinuria persists.

C. Immediate plasmapheresis

D. Cyclophosphamide induction

E. Kidney biopsy now in all cases

Answer: B

Key reasoning:This is classic post-streptococcal GN (latent period, low C3 with normal C4, elevated ASO). It is typically self-limited, treated with supportive care; steroids/cytotoxics are not indicated unless atypical (rapidly progressive, severe crescents). C3 returns to normal within weeks —persistent hypocomplementemia warrants further evaluation.

Why distractors are wrong:A/C/D: Unnecessary/harmful.E: Biopsy reserved for atypical or severe presentations.

Exam tip:Post-infectious GN → supportive; no routine immunosuppression; C3 should recover.

29

Q28

A 74-year-old man with metastatic solid tumor and CKD G3b (eGFR 35) presents with severe hypercalcaemia (Ca^{2+} 3.35 mmol/L), confusion, dehydration, and AKI (Cr 2.1). He received IV fluids and calcitonin in the ED. The oncology team asks which antiresorptive to use acutely given his kidney function. What is the best choice?

Options:

A. Zoledronic acid standard dose

B. Use denosumab (RANKL inhibitor)

C. Pamidronate at high dose

D. Alendronate orally

E. No antiresorptive—fluids only

Answer: B

Key reasoning:In hypercalcaemia of malignancy with CKD, bisphosphonates (zoledronate/pamidronate) risk worsening renal function and require dose adjustments. Denosumab is safe in CKD (though monitor for hypocalcaemia, especially with low vitamin D). Calcitonin works quickly but tachyphylaxis occurs.

Why distractors are wrong:A/C: Nephrotoxic potential and dosing limits in CKD.D: Slow onset, inadequate acutely.E: Fluids alone are often insufficient.

Exam tip:Severe hyperCa + CKD → denosumab after fluids + calcitonin; watch for hypocalcaemia.

30

Q29

A 65-year-old woman with osteoarthritis and diabetes develops acute colicky flank pain, gross hematuria, and passage of tissue-like fragments in urine. She has taken NSAIDs daily for years. CT shows sloughed papilla obstructing the distal ureter with mild hydronephrosis; no radiopaque stones. Creatinine 1.4 (baseline 1.0). What is the most appropriate management?

Options:

A. High-dose NSAIDs for analgesia and observation

B. Urologic relief of obstruction (e.g., ureteroscopic extraction or stent) with hydration and opioid/acetaminophen-analgesia

C. Alkalinize urine and wait for dissolution

D. Start tamsulosin and watchful waiting for 4 weeks

E. Immediate nephrectomy

Answer: B

Key reasoning:Renal papillary necrosis (diabetes/NSAIDs) can produce sloughed papillae causing ureteral obstruction and hematuria. Management is urologic decompression/removal plus analgesia (avoid further NSAIDs) and supportive care. Dissolution/α-blockade won't resolve obstructing tissue reliably.

Why distractors are wrong:A: NSAIDs contributed to pathology and harm kidneys.C/D: Ineffective for necrotic papilla.E: Reserved for nonviable kidneys or uncontrolled sepsis/bleeding.

Exam tip:Sloughed papilla obstruction → decompress/remove, stop NSAIDs.

31

Q30

A 58-year-old woman with severe hypertriglyceridaemia (TG 2,400 mg/dL) is found to have serum sodium 122 mmol/L on a chemistry panel. She is asymptomatic and euvolemic; measured serum osmolality is 289 mOsm/kg. Urine osmolality 300 mOsm/kg. Repeat sodium by direct ion-selective electrode (blood gas analyzer) is 137 mmol/L. What is the correct interpretation and management?

Options:

1. True hyponatraemia; give hypertonic saline
2. This is pseudohyponatraemia
3. SIADH; start fluid restriction
4. Hyperglycaemia-related dilution; give insulin
5. Lab error; ignore and discharge

Answer: B

Key reasoning:Normal measured osmolality with low Na^+ by indirect ISE in severe hypertriglyceridaemia indicates pseudohyponatraemia (spuriously low Na^+ from displaced plasma water). Direct ISE confirms normal true sodium. Manage the lipids; do not give hypertonic fluids.

Why distractors are wrong:A/C/D: Misdiagnoses leading to harmful therapy.E: Not an error—methodology issue.

Exam tip:Low Na^+ + normal osmolality + high TG → pseudohyponatraemia → direct ISE; treat lipids, not Na^+.

European...

This essential guide prepares nephrology candidates for the European Specialty Examination with a comprehensive collection of sample multiple-choice questions. Each question is meticulously crafted to reflect real-world clinical scenarios, emphasizing critical interventions such as immunosuppression management and addressing common post-transplant complications. Enhance your knowledge and confidence with expert insights and exam strategies tailored for nephrology practitioners.