ACE Your Acute Medicine SCE Exam

Acute Medicine SCE Sample Questions 20Q

SCE Recall Group

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Acute Medicine SCE Sample Questions

02

Q1

Stem:

A 32-year-old woman presents to ED with sudden pleuritic chest pain, collapse, and profound breathlessness on day 5 after an emergency Caesarean section for obstructed labour. She has right calf pain and swelling since yesterday. On arrival she is obtunded but arousable. Observations: T 37.1 °C, HR 132 bpm, BP 78/48 mmHg, RR 32, SpO_2 86% on 15 L non-rebreather. JVP is elevated; chest is clear; heart sounds normal; no wheeze. There is a tender, swollen right calf with pitting oedema. ECG shows sinus tachycardia with new incomplete RBBB and SIQ3T3 pattern. Bedside echo (focused) reveals a dilated RV with septal flattening (D-shaped LV) and reduced TAPSE. Arterial lactate is 5.2 mmol/L. The CT scanner is occupied with a major trauma for the next 45–60 minutes. What is the single best next step?

Options:

A. Arrange immediate CTPA to confirm pulmonary embolism before initiating treatment

B. Start therapeutic low-molecular-weight heparin and await urgent CTPA when available

C. Administer systemic thrombolysis now for presumed high-risk PE and arrange ICU transfer; defer imaging until stabilized

D. Give 2–3 litres of rapid IV crystalloid to improve RV preload before other therapy

E. Insert an urgent IVC filter due to recent Caesarean section and bleeding risk

Answer:

C

Explanation:

• Key reasoning (diagnosis/decision pathway; priorities; contraindications; timing): She has high-risk (massive) PE: obstructive shock (SBP <90 mmHg), hypoxaemia, raised lactate, and echo evidence of RV failure, with a compelling postpartum VTE history. In unstable PE, the priority is immediate reperfusion—systemic thrombolysis if not absolutely contraindicated—because delay markedly increases mortality. Post-partum/C-section is a relative contraindication; in life-threatening PE, thrombolysis is indicated. Do not delay for CTPA. Use cautious small fluid boluses only (250 mL) as large volumes worsen RV strain; start norepinephrine if needed. Begin UFH/LMWH after lysis once bleeding risk is assessed; consider catheter-directed therapy or surgical embolectomy if thrombolysis fails or is truly contraindicated.

• Guideline points (UK 2024–25): ESC PE guideline: For high-risk PE with shock, give immediate systemic thrombolysis (e.g., alteplase 100 mg over 2 h) without waiting for imaging if unstable and high clinical probability; consider surgical/catheter options if lysis contraindicated or ineffective. NICE NG158 (VTE): In suspected PE with haemodynamic instability, do not delay treatment for imaging; manage as per high-risk PE pathways.

RCUK ALS / obstetric VTE practice: In peri-arrest/unstable patients with suspected PE—even postpartum—thrombolysis should be given when benefits outweigh bleeding risk; postpartum status is not an absolutecontraindication in shock. Fluid/vasopressor strategy: Avoid large fluid boluses; use norepinephrine to support RV perfusion; oxygenate and arrange ICU.

• Why distractors are wrong:

A. Waiting for CTPA delays life-saving reperfusion in unstable PE; mortality rises with delay. B. Anticoagulation alone is insufficient in obstructive shock; definitive reperfusion is required now. D. Large-volume fluids can worsen RV dilation and septal shift, reducing LV output; give only small cautious boluses and start vasopressors. E. IVC filters do not treat the current embolus and are not first-line in shock; bleeding risk does not preclude thrombolysis when PE is life-threatening.

• Exam tip/pitfall: Post-partum ≠ absolute "no lysis." In obstructive shock from presumed PE, thrombolyse first, image later—then reassess bleeding risk and institute anticoagulation/definitive planning.

03

Q2

Stem:

A 62-year-old woman presents with recurrent presyncope and a brief collapse at home. She started erythromycin 2 days ago for pneumonia and takes citalopram 40 mg daily. In ED she is anxious and pale. Observations between episodes: HR 54 bpm, BP 92/56 mmHg, RR 22, SpO_2 95% on air. Telemetry shows recurrent 10–15 s runs of polymorphic VTseparated by sinus bradycardia. 12-lead ECG between runs: QTc 520 ms with notched T waves. Labs: K^+ 3.0 mmol/L, Mg^{2+} 0.5 mmol/L (low), Ca^{2+} 2.26 mmol/L, troponin mildly elevated in the context of pneumonia. She has a palpable pulse and briefly rouses after each episode but remains hypotensive. Pads are on; a defib is at the bedside. What is the single best next step now?

Options:

A. Deliver immediate synchronised DC cardioversion for each episode

B. Give IV amiodarone bolus followed by infusion

C. Give IV magnesium sulfate 2 g (8 mmol) over 10–15 minutes, urgently replete potassium to high-normal, and stop QT-prolonging drugs

D. Give IV lidocaine 1 mg/kg bolus

E. Start an isoprenaline (isoproterenol) infusion to increase heart rate to >90 bpm

Answer:

C

Explanation:

• Key reasoning (diagnosis/decision pathway; priorities; contraindications; timing): This is torsades de pointes due to acquired long-QT (bradycardia + QT-prolonging drugs + hypokalaemia + hypomagnesaemia). She has a pulse and self-terminating runs with peri-syncope and hypotension. The priority is to stabilise the repolarisation abnormality: immediate IV magnesium sulfate 2 g even if serum Mg is normal, aggressive K^+ repletion to 4.5–5.0 mmol/L, stop QT-prolonging agents (erythromycin, high-dose citalopram), treat bradycardia trigger (temporary overdrive pacing or isoprenaline may be considered if refractory/bradycardia-triggered). DC shock may be needed if a run becomes sustained with loss of output, but first-line for ongoing torsades with a pulse is magnesium + electrolyte correction.

• Guideline points (UK 2024–25):

RCUK Adult Advanced Life Support / Tachycardia algorithms: For polymorphic VT (torsades) with prolonged QT, give magnesium sulfate 2 g IV over 10–15 min; correct K^+ to high-normal; discontinue QT-prolonging drugs. Consider overdrive pacing or isoprenaline if episodes are pause-dependent/bradycardia-mediated. Avoid amiodarone as it can prolong QT. Perform unsynchronised shocks only if the patient becomes pulseless; if sustained and unstable with a pulse, cardioversion may be required while giving magnesium. NICE/BNF safety: Macrolides and citalopram prolong QT; high-dose citalopram (40 mg) increases risk —stop/adjust.

• Why distractors are wrong:

A. Cardioversion may terminate a sustained episode but does not prevent recurrence in pause-dependent torsades and risks delay to definitive repolarisation therapy (Mg/K^+). B. Amiodarone prolongs QT and can worsen torsades—contraindicated here. D. Lidocaine may help in ischaemic VT but has limited efficacy in long-QT torsades vs magnesium/electrolyte correction. E. Isoprenaline can be considered after magnesium and K^+ if episodes are bradycardia-triggered or while arranging pacing; it is not first-line and may increase myocardial oxygen demand.

• Exam tip/pitfall: Torsades = Mg first, no amiodarone. Replete K^+ to 4.5–5.0, stop QT-prolongers, and consider overdrive pacing/isoprenaline only if refractory and pause-dependent.

04

Q3

Stem:

A 45-year-old woman presents with a sudden, worst-ever occipital headache that began 10 hours ago while lifting a suitcase. She vomited twice. No focal deficits; neck stiffness is present. Observations: BP 168/92, HR 84, RR 18, SpO_2 99% RA. Non-contrast CT head performed now is reported normal. You remain concerned about aneurysmal subarachnoid haemorrhage (SAH). What is the single best next step?

Options:

A. Plan lumbar puncture with spectrophotometry for xanthochromia at ≥12 hours from headache onset

B. Reassure and discharge; normal CT rules out SAH at any time point

C. Start therapeutic anticoagulation for presumed CVST

D. Arrange MRI brain with contrast today instead of LP

E. Start nimodipine and observe on the ward without further testing

Answer:

A

Explanation:

• Key reasoning (diagnosis/decision pathway; priorities; contraindications; timing): CT sensitivity for SAH falls as time elapses; at ~10 hours, a normal CT does not exclude SAH. The next step is LP for xanthochromia using spectrophotometry, performed ≥12 hours after onset to allow bilirubin formation. If positive, proceed to vascular imaging/neurosurgical pathway.

• Guideline points (UK 2024–25): UK stroke/RCEM pathways: If CT is negative and suspicion of SAH persists >6 hours from onset, perform LP at ≥12 h to detect xanthochromia; if positive, arrange CTA/neuroradiology. (Some centres may use CT/CTA algorithms; LP remains a standard pathway for exclusion when CT is negative beyond the early window.)

• Why distractors are wrong:

B. Normal CT beyond early window cannot safely exclude SAH. C. No evidence of CVST; anticoagulation could be harmful. D. MRI is not the first-line rule-out; LP with spectrophotometry is the validated test in this context. E. Nimodipine is for confirmed aneurysmal SAH; giving it without diagnosis is inappropriate.

• Exam tip/pitfall: Time matters: CT-negative after >6 h → LP ≥12 h for xanthochromia.

05

Q4

Stem:

A 64-year-old man on ramipril presents with rapidly progressive tongue and floor-of-mouth swelling, dysarthria, drooling, and muffled voice that began 45 minutes ago. He is anxious and sitting forward. Observations: T 36.8 °C, HR 104, BP 132/78, RR 28, SpO_2 93% on air. There is no urticaria or wheeze. He received IM adrenaline 0.5 mg from ambulance crew with no improvement. On exam the tongue is markedly enlarged with submandibular firmness; laryngeal stridor is audible. Two attempts at oral airway examination provoke distress. What is the single best next step?

Options:

A. Nebulised adrenaline and IV chlorphenamine; repeat IM adrenaline and observe

B. Urgent senior airway management (awake fibreoptic/intubation in theatre with ENT standby ± front-of-neck access) while stopping the ACE inhibitor; give bradykinin-pathway therapy (e.g., icatibant or C1-esterase inhibitor) as adjunct

C. High-dose IV hydrocortisone and chlorphenamine alone, then reassess in 2 hours

D. Start IV tranexamic acid and discharge if swelling stabilises

E. Arrange CT neck with contrast to assess swelling before intervention

Answer:

B

Explanation:

• Key reasoning (diagnosis/decision pathway; priorities; contraindications; timing): This is bradykinin-mediated angioedema from an ACE inhibitor (no urticaria, no response to adrenaline). Priority is a controlled, senior-led airway—ideally awake fibreoptic with ENT backup and readiness for front-of-neck access. Pharmacologic adjuncts that may help include icatibant (bradykinin B2 antagonist) or C1-esterase inhibitor; stop the ACE inhibitor permanently. Antihistamines/steroids have limited immediate effect in bradykinin angioedema. Do not delay airway control for imaging.

• Guideline points (UK 2024–25): RCEM/UK allergy pathways: Early airway control is paramount in suspected bradykinin angioedema; adrenaline/antihistamines/steroids are often ineffective. Consider icatibant or C1-INH for ACEi-induced angioedema; avoid delays for imaging. Post-event, ACEi is contraindicated permanently.

• Why distractors are wrong:

A. These target histamine-mediated anaphylaxis; ineffective here and risk delay. C. Steroids/antihistamines alone act too slowly and don't address airway threat. D. TXA evidence is limited and should not replace airway control/specific therapy. E. CT delays airway control and may be unsafe (lying flat).

• Exam tip/pitfall: ACEi angioedema ≠ anaphylaxis—secure the airway first, then icatibant/C1-INH; antihistamines/steroids won't save the day.

06

Q5

Stem:

A 66-year-old woman with heart failure on sotalol and citalopram presents after a syncopal episode. Telemetry in ED captures runs of polymorphic VT with a twisting axis that self-terminate; between runs she is awake with borderline perfusion (BP 92/58). ECG between episodes: sinus bradycardia 54 bpm, QTc 520 ms. Labs: K^+ 3.4 mmol/L, Mg^{2+} 0.62 mmol/L, Ca^{2+} normal. What is the single best immediate treatment?

Options:

A. IV amiodarone bolus then infusion

B. IV magnesium sulfate 2 g over 10–15 minutes (repeat if needed), with rapid correction of K^+ to high-normal; stop QT-prolonging drugs; consider overdrive pacing/isoproterenol if recurrent and bradycardia-dependent; DC shock if haemodynamically unstable

C. Lidocaine bolus and infusion as first-line

D. Urgent thrombolysis for suspected myocardial ischaemia

E. Carotid sinus massage to terminate tachycardia

Answer:

B

Explanation:

• Key reasoning: Torsades de pointes due to prolonged QT (sotalol + citalopram, mild hypokalaemia/hypomagnesaemia). First-line is IV magnesium, K^+ repletion to 4.5–5.0, remove offenders, treat bradycardia (overdrive pacing/isoproterenol) if pause-dependent. Synchronised shock if unstable sustained TdP. Avoid class III antiarrhythmics.

• Guideline points (UK 2024–25): Resus Council UK arrhythmia algorithms: For TdP → $MgSO_4$ 2 g IV, correct electrolytes, consider overdrive pacing/isoproterenol if recurrent with bradycardia; avoid amiodarone. Shock if unstable.

• Why distractors are wrong:

A. Amiodarone prolongs QT and can worsen TdP. C. Lidocaine has limited benefit in TdP from QT prolongation. D. No evidence of STEMI; thrombolysis is inappropriate. E. Carotid massage is for AVNRT, not polymorphic VT.

• Exam tip/pitfall: TdP = magnesium + K^+, stop QT offenders; don't give amiodarone.

07

Q6

Stem:

A 69-year-old man with known lung adenocarcinoma presents with progressive breathlessness and right-sided chest discomfort. He is sitting forward, tachypnoeic and hypotensive. Observations: HR 118, BP 86/52, RR 30, SpO_2 90% on air. Trachea deviates to the left; percussion is stony dull and breath sounds are absent over the right hemithorax. Urgent bedside ultrasound confirms a very large right pleural effusion with mediastinal shift; no lung sliding to suggest pneumothorax. What is the single best immediate management?

Options:

A. Insert an ultrasound-guided pleural drain/large-bore catheter urgently to decompress the tension hydrothorax, with controlled drainage (initially ≤1–1.5 L, stop if chest tightness/cough), and send pleural fluid for analysis

B. Give high-dose IV furosemide and observe overnight

C. Arrange CT pulmonary angiography to exclude PE first

D. Perform complete drainage of the effusion (>3 L) as fast as possible to normalise BP

E. Start systemic anticoagulation for presumed malignant PE

Answer:

A

Explanation:

• Key reasoning: Tension hydrothorax causing obstructive shock (mediastinal shift, hypotension) from massive malignant effusion. Priority is urgent decompression with a pleural drain under ultrasound guidance. To reduce risk of re-expansion pulmonary oedema, use controlled initial drainage (≈1–1.5 L) and stop if symptoms arise; further staged drainage/IPC can follow once stabilised.

• Guideline points (UK 2024–25): BTS Pleural Disease: For large pleural effusions causing respiratory/hemodynamic compromise, perform urgent therapeutic drainage under ultrasound; initial removal ≤1–1.5 L with caution for re-expansion oedema; consider indwelling pleural catheter for malignant effusions.

• Why distractors are wrong:

B. Diuretics won't treat malignant effusion and delay decompression. C. Imaging for PE delays source control; signs point to obstructive physiology. D. Rapid complete drainage increases re-expansion oedema risk. E. Anticoagulation is inappropriate and unsafe without PE evidence.

• Exam tip/pitfall: Shift + stony dull hemithorax = tension hydrothorax—drain now, but limit first litre to avoid re-expansion oedema.

08

Q7

Stem:

A 58-year-old man with no known pituitary disease develops a sudden "blow to the head" bifrontal headache while lifting boxes at work. He vomits and notices rapidly worsening blurred vision. On arrival he is photophobic and distressed. Observations: T 37.6 °C, HR 104, BP 102/64 mmHg, RR 20, SpO_2 98% RA. Neurology: bitemporal hemianopia, partial right third-nerve palsy (ptosis, "down and out," anisocoria). He is pale, sweaty, and says he "nearly fainted" in the ambulance. Point-of-care glucose 3.2 mmol/L. Labs: Na^+ 129 mmol/L, K^+ 4.7 mmol/L, urea 10.6 mmol/L, creatinine 104 µmol/L. You suspect pituitary apoplexy. MRI is available within 90 minutes; neurosurgery can be contacted immediately.

Options:

A. Give IV mannitol and arrange MRI; no steroids until imaging confirms diagnosis

B. Start high-dose IV hydrocortisone immediately, correct hypoglycaemia, give fluids, arrange urgent MRI and neurosurgical review; protect airway as needed

C. Give desmopressin immediately for suspected DI, then image

D. Start IV broad-spectrum antibiotics for meningitis and perform LP now

E. Observe with analgesia and outpatient MRI within 48–72 hours

Answer:

B

Explanation:

• Key reasoning: Pituitary apoplexy is an endocrine-neurosurgical emergency with acute headache, ophthalmoplegia/visual loss, and haemodynamic/biochemical evidence of acute secondary adrenal insufficiency. Immediate stress-dose steroids (e.g., hydrocortisone 100 mg IV bolus then 50–100 mg q6h or infusion) should be given before imaging to avert cardiovascular collapse and reduce oedema; correct hypoglycaemia and fluid deficits. Urgent MRI and neurosurgical/ophthalmology input determine need/timing of decompression.

• Guideline points (UK 2024–25): Society for Endocrinology/UK: Give IV hydrocortisone urgently when apoplexy suspected; do not delay for imaging. Arrange urgent MRI and neurosurgical review; formal visual assessment; fluid/electrolyte management.

• Why distractors are wrong:

A. Delays life-saving glucocorticoids. C. DI is uncommon at presentation and desmopressin can worsen hyponatraemia; assess osmolality/urine output first. D. LP risks herniation and is not indicated with classic apoplexy signs. E. Outpatient imaging is unsafe; vision/endocrine function at risk.

• Exam tip/pitfall: In thunderclap headache with ophthalmoplegia and hyponatraemia—give hydrocortisone first, scan second.

09

Q8

Stem:

A 67-year-old man is day 12 of severe acute pancreatitis (gallstone aetiology). Over the last 24 hours he has spiking fevers, increasing abdominal pain, rising inflammatory markers (CRP 320 mg/L), and new septic physiology despite broad-spectrum antibiotics. CT pancreas (contrast-enhanced) shows a large walled-off necrotic collection with gas bubbles. He is haemodynamically stable on low-dose vasopressors.

Options:

A. Continue antibiotics alone and plan elective necrosectomy at 6–8 weeks

B. Immediate open necrosectomy

C. Start targeted broad-spectrum antibiotics and arrange urgent minimally invasive source control (e.g., step-up percutaneous/endoscopic drainage)

D. Stop antibiotics and repeat CT in 72 hours

E. Give octreotide infusion to reduce pancreatic secretions as definitive therapy

Answer:

C

Explanation:

• Key reasoning: Infected pancreatic necrosis (gas in collection + sepsis) requires antibiotics plus early source control. “Step-up” minimally invasive approaches (percutaneous or endoscopic drainage, with delayed necrosectomy if needed) reduce morbidity versus early open surgery.

• Guideline points (UK 2024–25): BSG/UK IAP–APA: In suspected/confirmed infected necrosis, give broad-spectrum antibiotics with good pancreatic penetration and perform minimally invasive drainage (step-up) once walled-off; reserve open necrosectomy for failure/complications.

• Why distractors are wrong:

A. Antibiotics alone are inadequate with gas and sepsis. B. Early open surgery increases harm; step-up first. D. Withholding therapy risks deterioration. E. Octreotide is not definitive.

• Exam tip/pitfall: “Gas in necrosis” = infection → antibiotics plus drain now; cut later if needed.

10

Q9

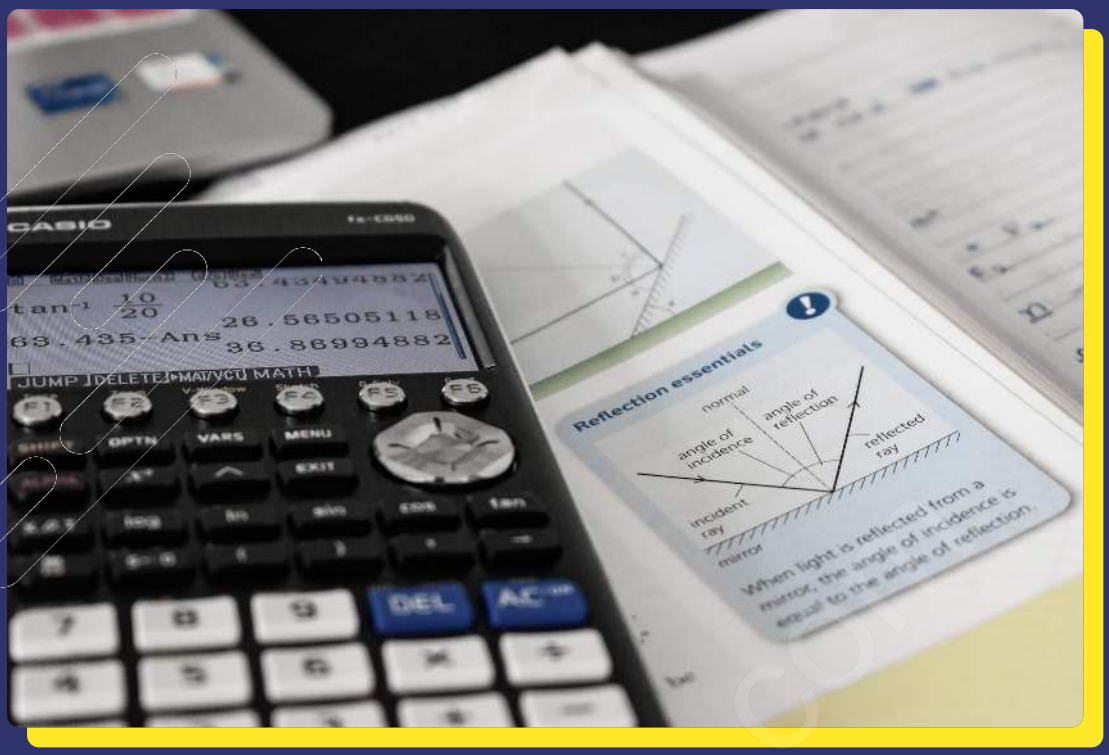
Stem:

A 45-year-old woman with idiopathic pulmonary arterial hypertension (PAH) on continuous IV epoprostenol via pump and sildenafil presents with escalating dyspnoea, syncope, and abdominal distension. Observations: HR 118, BP 84/52, RR 28, SpO_2 93% on 4 L O_2. JVP to angle of jaw, loud P2, cool peripheries, clear lungs; tender hepatomegaly and oedema. POCUS: severely dilated RV with poor TAPSE, small LV, plethoric IVC; no pneumothorax or effusion. The epoprostenol line is patent and running. What is the single best immediate haemodynamic strategy?

Options:

A. Large fluid boluses to raise preload

B. Intubate and ventilate early to reduce work of breathing



C. Start norepinephrine to support systemic pressure and RV perfusion, continue epoprostenol, avoid large fluids, consider inhaled nitric oxide/epoprostenol and cautious IV diuresis; ICU care

D. Start IV diltiazem for rate control

E. Stop the prostacyclin to avoid systemic vasodilation

Answer:

C

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Decompensated PAH/right-ventricular failure with cardiogenic shock. Priorities: maintain coronary perfusion with norepinephrine, never stop prostacyclin, avoid intubation if possible (positive pressure may precipitate collapse), add inhaled pulmonary vasodilator (iNO/nebulised prostacyclin) and cautious diuresis to decongest RV; early ICU.

• Guideline points (UK 2024–25): BTS/ESC PAH crisis: maintain RV perfusion with norepinephrine ± vasopressin; continue/optimize prostacyclin; add inhaled vasodilators; avoid large fluids; intubate only as last resort with expert team; diurese judiciously.

• Why distractors are wrong:

A. Large fluids worsen RV dilation/septal shift. B. Intubation can be catastrophic; avoid if possible. D. Calcium-channel blockers are for rare vasoreactive PAH and contraindicated in decompensation. E. Abrupt cessation of epoprostenol can be fatal.

• Exam tip/pitfall: PAH crash = pressors + inhaled vasodilator + keep prostacyclin on; don't flood or intubate early.

II

Q10

Stem:

A 38-year-old man with obesity and poorly controlled diabetes presents with severe epigastric pain and vomiting. Lipase is 2,400 U/L; triglycerides 54 mmol/L; glucose 16 mmol/L; pH 7.31; Ca^{2+} 2.08 mmol/L. CT (done for severe pain) confirms acute pancreatitis without necrosis. He is tachycardic but normotensive. What is the single best disease-modifying therapy to acutely lower triglycerides?

Options:

A. Start therapeutic plasmapheresis immediately as first-line

B. Start IV insulin infusion with dextrose (e.g., 0.1 U/kg/h) to drive lipoprotein lipase activity, plus fluids and electrolyte monitoring; consider plasmapheresis only if refractory or organ failure

C. Begin high-dose statin

D. Start heparin infusion to release lipoprotein lipase

E. Nil by mouth and observe—TG will fall spontaneously

Answer:

B

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Hypertriglyceridaemia-induced pancreatitis: insulin infusion lowers TGs rapidly by activating LPL; give with dextrose and tight glucose/potassium monitoring and standard pancreatitis care. Apheresis reserved for refractory cases or severe organ failure. Heparin depletes LPL and is not recommended.

• Guideline points (UK 2024–25): BSG/critical-care practice: insulin infusion is first-line to reduce TG; plasmapheresis considered in fulminant disease or failure of medical therapy; avoid routine heparin.

• Why distractors are wrong:

A. Not universally first-line; invasive and resource-intense. C. Statins act too slowly. D. Heparin transiently raises then depletes LPL—can worsen. E. Observation risks ongoing pancreatic injury.

• Exam tip/pitfall: Think insulin, not heparin, for TG pancreatitis; apheresis is a rescue.

12

Q11

Stem:

A 26-year-old man returns from Nigeria 5 days ago with fever, confusion, jaundice, and vomiting. Observations: T 39.6 °C, HR 128, BP 96/58, RR 24, SpO_2 95% RA. Labs: Hb 86 g/L, platelets 42 $\times 10^9$/L, bilirubin 96 µmol/L, creatinine 212 µmol/L, lactate 4.8 mmol/L, glucose 2.6 mmol/L. Rapid test positive for P. falciparum; blood film shows parasitaemia 8%. What is the single best immediate antimalarial therapy?

Options:

A. Oral artemether–lumefantrine

B. IV artesunate with ICU admission, alongside hypoglycaemia management and supportive care

C. IV quinine plus doxycycline only if artesunate unavailable

D. Await parasite speciation and sensitivity panel

E. Oral chloroquine

Answer:

B

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Severe falciparum malaria (altered mental status, high parasitaemia, AKI, hypoglycaemia). First-line is IV artesunate, which reduces mortality versus quinine; treat hypoglycaemia aggressively and provide ICU-level supportive care.

• Guideline points (UK 2024–25): UKHSA/BSAC malaria: severe P. falciparum → IV artesunate; if unavailable, IV quinine + doxycycline/clindamycin; monitor for delayed haemolysis after artesunate.

• Why distractors are wrong:

A/E. Oral therapy inadequate and chloroquine-resistant. C. Acceptable only if artesunate unavailable. D. Do not delay treatment for speciation/sensitivities.

• Exam tip/pitfall: Any severe feature or parasitaemia ≥2%—give IV artesunate now, then worry about nuances.

13

Q12

Stem:

A 74-year-old man develops fever, rigors, chest/back pain, and dark urine 20 minutes into a packed red cell transfusion. He becomes hypotensive. Observations: T 39.6 °C, HR 128, BP 78/46. Urine in catheter bag is red–brown. Labs later: falling Hb, rising bilirubin, LDH, K^+ 5.8 mmol/L, aPTT prolonged. What is the single best immediate action?

Options:

A. Slow the transfusion and give IV chlorphenamine

B. Stop the transfusion immediately, maintain IV access with saline, send the blood and samples to the transfusion lab, commence aggressive IV fluids ± diuresis to protect kidneys, and manage DIC

C. Continue transfusion and give hydrocortisone

D. Switch to group O-negative blood and continue

E. Start broad-spectrum antibiotics for presumed sepsis and ignore transfusion pathway

Answer:

B

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Acute haemolytic transfusion reaction (ABO incompatibility until proven otherwise): fever, pain, hypotension, haemoglobinuria. Stop transfusion immediately, inform lab, return unit, draw reaction samples, give aggressive IV fluids (aim urine output >l mL/kg/h), treat hyperkalaemia/DIC as needed.

• Guideline points (UK 2024–25): SHOT/BCSH—serious transfusion reaction: stop transfusion, keep IV saline, notify lab, full reaction workup; manage shock/AKI; do not restart; future transfusion only after investigation.

• Why distractors are wrong:

A. Do not “slow” a suspected haemolytic reaction. C/D. Continuing transfusion can be fatal. E. Sepsis can mimic but transfusion protocol is mandatory; antibiotics may be secondary.

• Exam tip/pitfall: Pain + fever + haemoglobinuria during transfusion = stop and call lab—don’t restart.

14

Q13

Stem:

A 78-year-old woman is brought in during winter with progressive lethargy, confusion, and cold intolerance. She lives alone and has hypothyroidism but stopped levothyroxine “months ago.” On arrival she is obtunded and hypothermic (core 32.4 °C). Observations: HR 48, BP 92/54 mmHg, RR 10, SpO_2 92% on air. She has puffy facies, dry skin, delayed reflex relaxation, and periorbital oedema. ABG on 28% Venturi O_2: pH 7.29, $PaCO_2$ 7.2 kPa, PaO_2 8.9 kPa. Labs: Na^+ 121 mmol/L, glucose 3.4 mmol/L, TSH 48 mU/L, free T4 undetectable, cortisol pending. CXR: cardiomegaly, pleural effusions. What is the single best immediate treatment strategy?

Options:

A. Active external rewarming with hot-air blankets to 39 °C and oral levothyroxine when awake

B. IV liothyronine (T3) alone in high dose to rapidly reverse coma

C. IV hydrocortisone immediately, cautious passive rewarming, controlled ventilation for hypoventilation, IV levothyroxine loading dose then daily, correct glucose/Na^+, ICU admission

D. Start oral levothyroxine 25 µg/day and observe on ward

E. Give tolvaptan for hyponatraemia first, then consider thyroid hormones later

Answer:

C

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Myxoedema coma: hypothermia, bradycardia, hypoventilation with hypercapnia, hyponatraemia, and severe hypothyroidism. Priorities are airway/ventilation, stress-dose hydrocortisone before/with thyroid hormone (occult adrenal insufficiency risk), IV levothyroxine loading (e.g., 200–300 µg) then 50–100 µg/day, passive warming (avoid rapid vasodilation), correct hypoglycaemia/hyponatraemia carefully, treat precipitant; ICU level care.

• Guideline points (UK 2024–25): Society for Endocrinology emergency guidance and RCEM: hydrocortisone 100 mg IV stat then 50 mg q6h; levothyroxine IV preferred; liothyronine only as adjunct in specialist care; avoid aggressive active rewarming; manage sodium slowly.

• Why distractors are wrong:

A. Aggressive active rewarming risks vasodilation and collapse; oral T4 unreliable. B. T3 alone can precipitate arrhythmias/ischaemia; give stress steroids and T4 regimen first. D. Oral low-dose T4 is inadequate and too slow. E. Vaptans are inappropriate; fix the endocrine crisis first with cautious fluid/Na^+ strategy.

• Exam tip/pitfall: Treat airway + steroids → IV T4, not T3 monotherapy; rewarm passively.

15

Q14

Stem:

A 66-year-old man with Waldenström macroglobulinaemia presents with headaches, blurred vision, epistaxis, and lethargy. He has retinal vein engorgement and “sausaging” on fundoscopy. Labs: Hb 98 g/L, platelets 160×10⁹/L, WBC 7×10⁹/L, total protein 96 g/L, serum viscosity raised. What is the single best immediate intervention?

Options:

A. High-dose dexamethasone

B. Urgent plasmapheresis to relieve hyperviscosity, then disease-directed therapy

C. Packed red cell transfusion

D. Start R-CHOP chemotherapy immediately

E. Desmopressin for bleeding

Answer:

B

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Symptomatic hyperviscosity from IgM paraprotein requires urgent plasma exchange to remove IgM and relieve ocular/neurological/bleeding symptoms; definitive treatment (BTK inhibitor/rituximab-based) follows. Avoid immediate RBC transfusion (↑ viscosity).

• Guideline points (UK 2024–25): BSH Waldenström: plasmapheresis for symptomatic hyperviscosity before rituximab (to avoid IgM flare); then systemic therapy.

• Why distractors are wrong:

A/D. Disease therapy needed but not before viscosity is corrected. C. Increases viscosity and worsens symptoms. E. DDAVP won't fix the rheology problem.

• Exam tip/pitfall: See "sausage" veins + neuro/bleeding → plasma exchange now, chemo later.

16

Q15

Stem:

A 30-year-old man on valproate for generalised epilepsy presents with subacute confusion, vomiting, and new asterixis. He is afebrile with normal LFTs and bilirubin. Ammonia is 132 µmol/L (high), valproate level therapeutic. CT head is normal. What is the single best treatment?

Options:

A. Lactulose and rifaximin only

B. Increase valproate dose to suppress seizures

C. Stop valproate and give IV L-carnitine, supportive care, and neurology review for antiseizure switch

D. Start mannitol for raised ICP

E. Start steroids for autoimmune encephalitis

Answer:

C

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Valproate-induced hyperammonaemic encephalopathy can occur with normal LFTs; treat by withdrawing valproate and giving L-carnitine to enhance mitochondrial metabolism; supportive care and alternative ASM (e.g., levetiracetam).

• Guideline points (UK 2024–25): BNFC/BNF/TOXBASE: consider L-carnitine IV for symptomatic hyperammonaemia from valproate; check ammonia in altered mental status on VPA; switch antiepileptic.

• Why distractors are wrong:

A. Targets portal-systemic encephalopathy, not drug-induced mitochondrial dysfunction. B. More VPA worsens the problem. D/E. Off-target and delay definitive therapy.

• Exam tip/pitfall: Confusion on valproate with normal LFTs? Check ammonia—stop VPA, give L-carnitine.

17

Q16

Stem:

A 72-year-old man presents 6 hours after sudden left hand clumsiness and dysarthria that largely improved but left subtle weakness (NIHSS 2). CT head shows no haemorrhage; CTA shows no LVO. ECG sinus rhythm. He has hypertension and hyperlipidaemia; not taking antiplatelets. What is the single best antithrombotic strategy now?

Options:

A. Aspirin alone

B. Dual antiplatelet therapy with aspirin plus clopidogrel for 21 days, then single antiplatelet

C. Full-dose LMWH

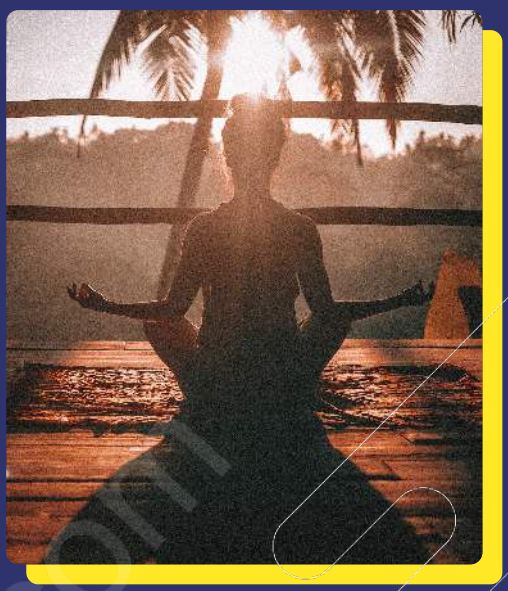
D. No antithrombotic therapy until MRI

E. Start ticagrelor monotherapy

Answer:

B

Explanation:



• Key reasoning: Minor non-cardioembolic ischaemic stroke within 24 h, no haemorrhage/LVO. Short-course DAPT (21 days) reduces early recurrence compared to aspirin alone, then switch to monotherapy.

• Guideline points (UK 2024–25): NICE NG128/RCP Stroke—offer 21 days DAPT (aspirin + clopidogrel) for minor stroke (NIHSS ≤3) or high-risk TIA if within 24 h and haemorrhage excluded; then single agent.

• Why distractors are wrong:

A. Inferior to short-course DAPT for early prevention. C. Anticoagulation not indicated without cardioembolism. D. Delay loses benefit. E. Ticagrelor monotherapy isn't standard first-line in this setting.

• Exam tip/pitfall: NIHSS ≤3 + early presentation → 21-day DAPT.

18

Q17

Stem:

A 69-year-old woman with type 2 diabetes on gliclazide is found confused and sweaty at home. Capillary glucose 1.9 mmol/L; she improves after IV dextrose but has recurrent hypoglycaemia over the next 4 hours despite continuous 10% dextrose infusion. Creatinine is mildly elevated (AKI). She denies insulin access. What is the single best next therapy?

Options:

A. Glucagon IM

B. Stop dextrose and give a large carbohydrate meal

C. Octreotide (e.g., 50–100 µg SC/IV q6–8 h or infusion) to suppress sulfonylurea-induced insulin release, plus dextrose as needed and observation

D. High-dose steroids

E. Start insulin–glucose infusion

Answer:

C

Explanation:

• Key reasoning: Sulfonylurea-induced hypoglycaemia causes rebound lows with dextrose alone. Octreotide suppresses pancreatic insulin release, preventing recurrence; continue glucose support and monitor.

• Guideline points (UK 2024–25): TOXBASE/RCEM—use octreotide for sulfonylurea toxicity with recurrent hypoglycaemia; admit for prolonged observation, especially with renal impairment.

• Why distractors are wrong:

A. Glucagon gives transient rise and may worsen rebound insulin release. B. Oral intake alone is unreliable. D. Steroids are not indicated. E. Insulin would worsen hypoglycaemia.

• Exam tip/pitfall: Recurrent hypos + sulfonylurea → think octreotide.

19

Q18

Stem:

A 82-year-old man with known severe calcific aortic stenosis (AVA 0.6 cm^2) develops acute pulmonary oedema and hypotension after sepsis treatment. Observations: HR 90, BP 78/46, RR 30, SpO_2 88% on CPAP; lungs wet; systolic ejection murmur; no dynamic ECG changes. He received IV nitrates pre-hospital. What is the single best haemodynamic strategy now while arranging definitive valve intervention?

Options:

A. High-dose nitrates and diuretics to offload the LV

B. Start a vasopressor (e.g., norepinephrine) to maintain coronary perfusion pressure, stop vasodilators, give small cautious fluid bolus if preload depleted, and urgently involve cardiology for rescue balloon valvuloplasty/TAVI

C. Aggressive afterload reduction with nitroprusside

D. Large fluid boluses (2–3 L) to increase preload

E. Immediate thrombolysis for presumed MI

Answer:

B

Explanation:

• Key reasoning: In cardiogenic shock from critical AS, coronary perfusion depends on aortic pressure; vasodilators/nitrates can precipitate collapse. Use norepinephrine (or phenylephrine) to support MAP, cautiously optimise preload, avoid afterload reducers, and expedite urgent valve intervention (balloon valvuloplasty as a bridge/TAVI).

• Guideline points (UK 2024–25): ESC valvular disease/RCEM—shock in severe AS: vasopressor support to preserve perfusion; avoid vasodilators; consider urgent BAV/TAVI; diuretics only if clearly volume overloaded and BP tolerates.

• Why distractors are wrong:

A/C. Vasodilators worsen hypotension in fixed outflow obstruction. D. Large boluses risk pulmonary oedema. E. No evidence of MI; thrombolysis is hazardous.

• Exam tip/pitfall: Fixed obstruction ≠ afterload reduction—support MAP and call the valve team.

20

Q19

Stem:

A 33-year-old man from East Asia presents with sudden flaccid weakness of all limbs on waking. He had a week of palpitations and weight loss. Observations: HR 128 (irregularly irregular), BP 132/76, RR 18, SpO_2 98%. Neurology: symmetric proximal > distal weakness (2/5), areflexia; sensation intact. Labs: K^+ 2.1 mmol/L, Mg^{2+} 0.6 mmol/L, phosphate 0.6 mmol/L, TSH <0.01, free T4 43 pmol/L. What is the single best immediate management?

Options:

A. Insulin–dextrose infusion to shift potassium

B. Careful IV potassium chloride replacement with cardiac monitoring plus a non-selective β-blocker (e.g., propranolol), avoid dextrose-containing fluids; treat thyrotoxicosis after stabilisation

C. IV calcium gluconate

D. Start carbimazole and delay potassium until thyroid hormones fall

E. Gabapentin for presumed neuropathy

Answer:

B

Explanation:

• Key reasoning: Thyrotoxic periodic paralysis: profound hypokalaemia from intracellular shift (not depletion). Treat with controlled IV KCl (watch for rebound), correct Mg^{2+}/phosphate, give non-selective β-blocker to stop shift; avoid dextrose (worsens intracellular K^+ uptake). Manage hyperthyroidism once stable.

• Guideline points (UK 2024–25): BTA/RCEM—replace K^+ cautiously (often 10–40 mmol IV with ECG monitoring), give propranolol; initiate antithyroid therapy after acute episode control.

• Why distractors are wrong:

A. Insulin worsens hypokalaemia. C. Calcium treats hyperkalaemia conduction issues. D. Delaying potassium risks arrhythmia/respiratory failure. E. Not neuropathy.

• Exam tip/pitfall: Low K^+ + hyperthyroid + paresis → replace K^+ + propranolol; no dextrose.

21

Q20

Stem:

A 55-year-old man is found drowsy at home with vomiting and clammy skin 4 hours after an intentional overdose of his partner's medications. Empty blister packs of amlodipine 10 mg × 20 and propranolol 40 mg × 10 are at the scene. On arrival: GCS 12, cold peripheries, capillary refill 5 s. Observations: HR 46 (junctional), BP 72/38 mmHg, RR 20, SpO_2 96% RA, T 35.8 °C. Glucose 3.8 mmol/L. ECG: junctional bradycardia, QRS 100 ms, QTc 470 ms. Labs: K^+ 4.8 mmol/L, Ca^{2+} 2.12 mmol/L, lactate 6.2 mmol/L, creatinine 126 µmol/L. He has received two 500-mL boluses of warmed 0.9% saline with minimal change in BP. Bedside echo: small LV cavity with severely reduced contractility; no pericardial effusion. What is the single best next therapy?

Options:

A. Give glucagon 10 mg IV bolus followed by infusion and reassess over 2–3 hours

B. Start high-dose insulin euglycaemia therapy (e.g., 1 U/kg IV bolus then 1–10 U/kg/h with dextrose to maintain euglycaemia) plus calcium, vasopressors, and close electrolyte monitoring

C. Start intravenous lipid emulsion as first-line therapy

D. Give IV calcium chloride only and observe for response

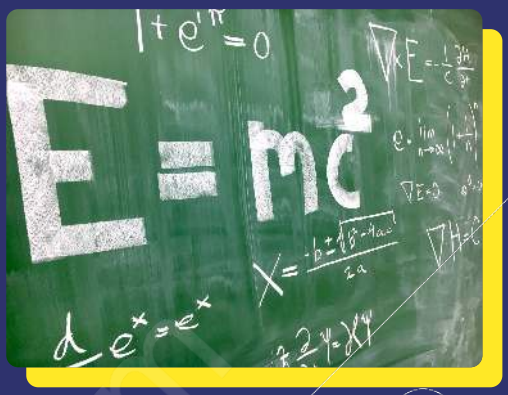
E. Immediate transvenous pacing before any pharmacological therapy

Answer:

B

Explanation:

• Key reasoning: (diagnosis/decision pathway; priorities; contraindications; timing) Mixed calcium-channel blocker (dihydropyridine) and β-blocker overdose with bradycardic shock and myocardial depression. After airway/oxygen, fluids, and atropine fail, the therapy with strongest haemodynamic evidence is high-dose insulin euglycaemia therapy (HIET) to improve inotropy and cellular metabolism, with concurrent dextrose, potassium monitoring, IV calcium, and vasopressors as needed. Glucagon may transiently help β-blocker toxicity but is unreliable in CCB-predominant shock. Early HIET improves outcomes; start in ED with ICU involvement.



• Guideline points (UK 2024–25): TOXBASE/RCEM toxicology: for CCB/β-blocker cardiogenic shock, initiate HIET early with dextrose and aggressive electrolyte monitoring; give IV calcium (for CCB), vasopressors (often noradrenaline ± adrenaline), consider lipid emulsion as rescue in lipophilic overdoses; pacing rarely effective when myocardial depression dominates.

• Why distractors are wrong:

A. Glucagon alone seldom reverses CCB-driven pump failure and is short-lived with nausea/vomiting. C. Lipid is rescue, not first-line for amlodipine/propranolol mix; start HIET first. D. Calcium is necessary adjunct for CCB but insufficient as sole therapy in shock. E. Pacing does not correct profound CCB-induced negative inotropy; unstable for transport without metabolic therapy.

• Exam tip/pitfall: Don't "wait and see" after calcium/glucagon—start HIET early and titrate with hourly glucose/K^+ checks.

Acute Medicine...

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