OSCE 06/2026

NDH

1. The Elderly Patient

Scenario: A 78-year-old woman is brought to the AED after an unwitnessed fall at home. She lives alone and is usually independent. She denies any dizziness, chest pain, or palpitations before the fall.

PMH: Hypertension on amlodipine and thiazide diuretic

Vitals: BP 138/78 mmHg, HR 82 bpm, RR 16/min, SpO_2 96% on air, Temp 36.8°C, GCS 15/15.

Blood results: Na **123** mmol/L, K 4.1, urea 4.2, Cr 88 µmol/L.

1a. What are the possible causes of hyponatraemia you would consider at this stage?
1b. What is your initial approach and management in the ED?

Answer

1a.

- Hypovolaemic: GI losses, diuretics, poor intake
- Euvolemic: SIADH (CNS, malignancy, drugs, pulmonary), hypothyroidism, glucocorticoid deficiency
- Hypervolaemic: CHF, cirrhosis, nephrotic syndrome
- Others: Pseudohyponatraemia, hyperglycaemia, psychogenic polydipsia

1b.

- ABCDE assessment + full history & examination (including volume status)
- ECG, CXR
- If asymptomatic/mild symptoms → fluid restriction + admit for workup
- If severe symptoms (seizure, coma) → hypertonic (3%) saline
- +(Check serum osmolality, glucose, TSH, cortisol. Paired urine osmolality and urine sodium)

Additional Information:

On further questioning, the patient reports mild fatigue and generalised weakness for 3 weeks. Her daughter reports ~3 kg weight loss over the last 3 months and intermittent post-menopausal vaginal bleeding for the past year. The patient did not seek medical advice as she thought it was normal.

Physical Examination:

- Moist mucous membranes, normal skin turgor, JVP not raised, no peripheral oedema.
- Mildly slow responses but oriented. No focal neurology.
- Speculum examination: small amount of blood-stained vaginal discharge.

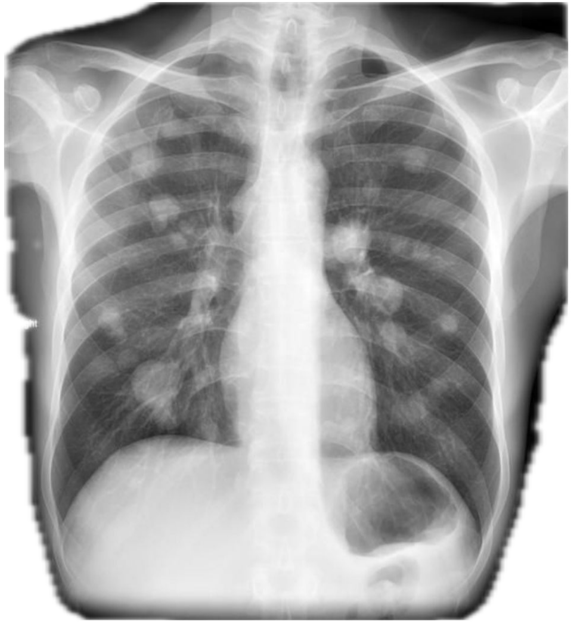
1c. What key investigations would help you classify the hyponatraemia?

Answer

- Serum osmolality
- Urine osmolality
- Urine sodium
- TSH
- Morning (9 am) cortisol
- Glucose
- CXR (looking for malignancy or lung pathology)
- Consider CT head if there is focal neurology

Additional Results:

- Serum osmolality (measured): **258** mOsm/kg
- Calculated osmolality: 2 × Na (123) + urea (4.2) + glucose (6.1) = **256** mOsm/kg
- Urine osmolality: **420** mOsm/kg
- Urine sodium: **68** mmol/L
- TSH and 9 am cortisol: normal



1d. Calculate the osmolar gap and explain its significance in the evaluation of hyponatraemia.

1e. What is the most likely diagnosis now?

Answer

1d.

Osmolar gap = Measured serum osmolality – Calculated serum osmolality = 258 – 256 = **2 mOsm/kg** (normal gap is <10 mOsm/kg).

Significance:

- A **normal osmolar gap** (as in this case) confirms that there are no significant unmeasured osmoles contributing to the picture.
- A **high osmolar gap** (>10) in a patient with hyponatraemia suggests:
  - Toxic alcohols (ethanol, methanol, ethylene glycol, isopropyl alcohol)
  - Mannitol or glycine (e.g., post-TURP)
  - Severe hyperlipidaemia or hyperproteinaemia (pseudohyponatraemia – iso-osmolar)
- In this patient, the normal gap helps **rule out** pseudohyponatraemia and toxic ingestions, confirming true **hypo-osmolar (hypotonic) hyponatraemia**.

Answer

1e.

- Low measured serum osmolality (258 mOsm/kg) + normal osmolar gap → **True hypotonic hyponatraemia**.
- Inappropriately elevated urine osmolality (420 mOsm/kg > 100) despite low serum osmolality → impaired free water excretion (ADH effect).
- High urine sodium (68 mmol/L) in a euvolemic patient → consistent with SIADH.

Overall, this is a classic laboratory profile of **SIADH**.

Therefore, the patient is likely to have **paraneoplastic SIADH secondary to metastatic endometrial carcinoma** (cannonball lung metastases + post-menopausal bleeding + prior missed endometrial thickening).

1f. List the common causes of SIADH using the MAD CHOP mnemonic

Answer

1f.

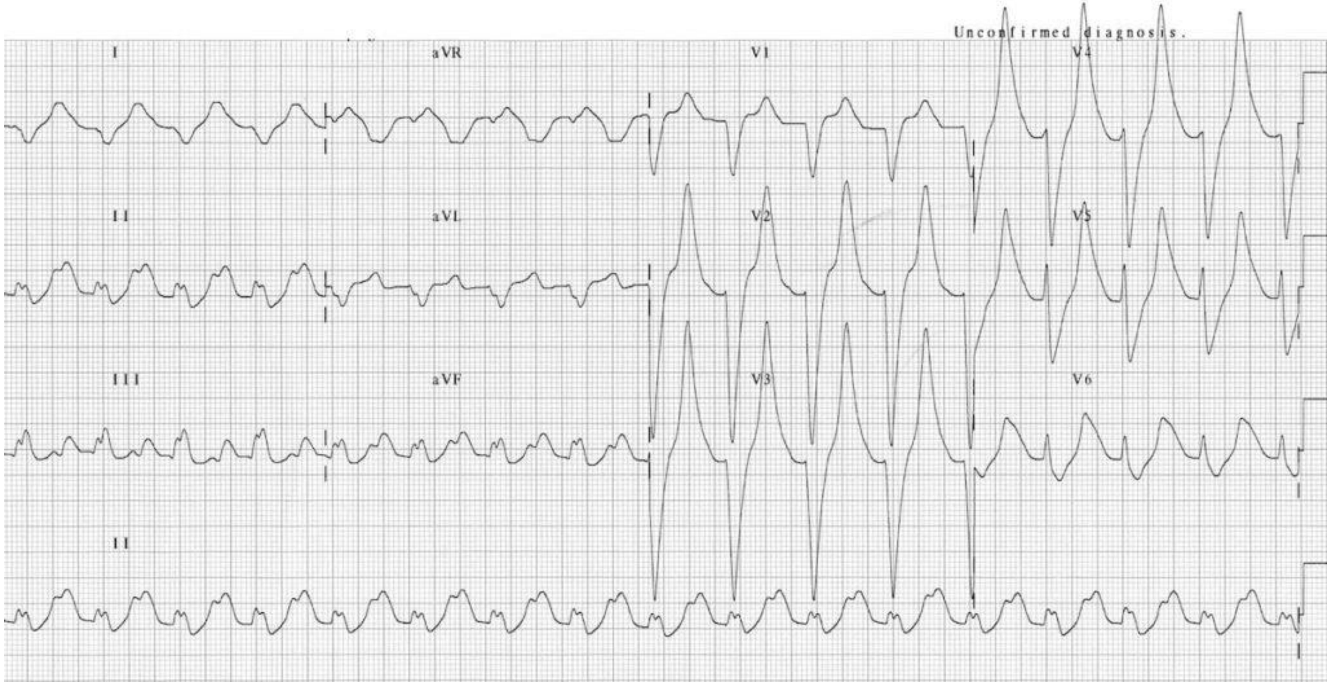
Letter	Category	Examples
M	Malignancy	Small cell lung cancer, endometrial cancer, lymphoma, pancreatic cancer
A	Autoimmune / CNS	Meningitis, encephalitis, stroke, Guillain-Barré
D	Drugs	SSRIs, carbamazepine, vincristine, cyclophosphamide, antipsychotics
C	CNS disorders	Head injury, brain tumour, subarachnoid haemorrhage
H	Hormonal	Hypothyroidism, adrenal insufficiency
O	Others	Pain, nausea, major surgery, pneumonia
P	Pulmonary	Pneumonia, TB, lung abscess, **lung metastases**

2. Iatrogenic Crisis in an Elderly Patient with Multiple Comorbidities

Scenario: A 76-year-old woman with a background chronic kidney disease stage 4 (baseline Cr 180 µmol/L), hypertension, and heart failure with reduced ejection fraction (EF 35%) is brought to the ED. She has been taking ramipril 10mg daily, spironolactone 25mg daily, and recently completed a course of Septrin for a UTI. She presents with generalised weakness, nausea, and palpitations for 2 days.

Vital signs: BP 95/58 mmHg, HR 48 bpm, RR 18, SpO_2 94% on room air, GCS 14/15

2a. Interpret the ECG



Answer

- Prolonged PR interval.
- Broad, bizarre QRS complexes — these merge with both the preceding P wave and subsequent T wave.
- Peaked T waves.

Additional Information:

Labs: K^+ 7.8 mmol/L, Na^+ 130 mmol/L, Cr 320 µmol/L (↑ from baseline), urea 28 mmol/L, pH 7.28, HCO_3^- 16 mmol/L, Hb 9.8 g/dL, glucose 8.2 mmol/L. Troponin is mildly elevated at 45 ng/L

2b. Categorise the likely contributing causes of her hyperkalaemia.

Answer

2b.

- **Decreased excretion** (dominant): CKD (reduced GFR) + ramipril (↓ angiotensin II → ↓ aldosterone) + spironolactone (aldosterone antagonist) + Septrin (ENaC blockade in collecting duct)
- **Transcellular shift**: Metabolic acidosis causes H^+/K^+ exchanger activation.
- **Increased intake**: Less likely but dietary potassium or salt substitutes should be excluded. The combination of baseline impaired excretion + acute insult (UTI/AKI) + multiple RAAS/ENaC inhibitors explains the severe presentation.

Further information:

You administer calcium gluconate, DI drip, Ventolin puffs, and sodium bicarbonate. After 90 minutes, repeat K^+ is 5.1 mmol/L and ECG improves. However, 5 hours later she develops muscle cramps and frequent ventricular ectopics. Repeat K^+ is now 2.9 mmol/L. She remains in heart failure with bilateral crepitations.

2c. Explain the mechanisms responsible for this rapid drop into hypokalemia. What ECG changes would you expect to see now?

Answer

2c.

Mechanisms of rebound hypokalemia:

- Correction of acidosis reverses extracellular K^+ shift.
- Insulin and beta-2 agonists (salbutamol) drive massive intracellular K^+ uptake.
- Improved renal perfusion increases urinary K^+ loss.
- Underlying total body K^+ depletion unmasked.

Expected ECG changes in hypokalaemia: Flattened/inverted T waves, prominent U waves, ST depression, prolonged QT interval, ventricular ectopics or VT (as seen clinically).

2d. Outline your safe potassium repletion strategy in this patient with CKD and heart failure, including dose, route, and monitoring.

Answer

2d.

Repletion strategy:

- Correct hypomagnesaemia first ($MgSO_4$ 2–4 g IV).
- **IV KCl** 20–40 mmol over 1 hour via **central line** (peripheral max 10–20 mmol/h in HF).
- Target: K^+ >3.5 mmol/L urgently, then >4.0–4.5 mmol/L.
- Switch to oral once stable.
- Continuous ECG monitoring + K^+ recheck every 1–2 hours. Withold spironolactone/ramipril.

Further Information:

Despite careful replacement, she develops recurrent non-sustained VT. Her repeat ABG shows pH 7.35, HCO_3^- 20 mmol/L.

2e. Discuss the potential life-threatening complications of both hyperkalaemia and hypokalaemia in this specific patient. What is your management plan and the indications for urgent dialysis?

Answer

2e.

Complications:

- **Hyperkalaemia**: Bradyarrhythmias, sine wave, VT/VF, asystole — worsened by heart failure and possible digoxin use (if on it).
- **Hypokalaemia**: Torsades de pointes, VT, muscle weakness, respiratory failure — particularly dangerous with low EF and low Mg^{2+}.

Management & Disposition:

- Admit to ICU if unstable with continuous cardiac monitoring.
- Serial K^+, Mg^{2+}, ABG, ECG q1–2h.
- Indications for urgent dialysis: Refractory hyperkalaemia, persistent severe acidosis, fluid overload with pulmonary oedema, or oliguria.

3. Acute Decompensation in a Young Child

Scenario:

A 6-year-old boy (weight 22 kg) is brought to the ED by his parents with a 3-days history of polyuria, polydipsia, lethargy, and vomiting. He has no known medical history but has been unwell with a recent viral illness.

Vital signs: HR 138 bpm, BP 92/55 mmHg, RR 32/min with deep sighing respirations, SpO_2 96% on air, capillary refill 3 seconds, GCS 14 (irritable). Bedside glucose: **32 mmol/L**. Blood gas: **pH 7.12,** pCO_2 22 mmHg, HCO_3^- 8 mmol/L, lactate 2.8 mmol/L. **Ketones: 5.2 mmol/L**.

3a. Interpret the blood gas and outline the key pathophysiological mechanisms linking insulin deficiency to the observed acid-base, fluid, and electrolyte disturbances in this child.

Answer

3a.

This is **severe DKA** (pH <7.20, HCO_3^- <10, ketones >3).

Pathophysiology:

Absolute insulin deficiency → hyperglycaemia → osmotic diuresis → profound dehydration + electrolyte loss (Na, K, PO_4)

Lipolysis → ketone production → high anion gap metabolic acidosis with respiratory compensation (Kussmaul breathing). Potassium is initially high due to extracellular shift from acidosis/insulin deficiency despite total body depletion.

3b. What is the most feared complication suggested by the new clinical changes? List key risk factors in paediatric DKA and discuss how your initial fluid management may influence this risk.

3b.

Cerebral oedema (most serious complication in paediatric DKA, mortality ~25% among those affected).

Risk factors: Young age (<10 years), new-onset T1DM, severe acidosis, high urea, low pCO_2, rapid fall in corrected sodium/osmolality, bicarbonate use, excessive or hypotonic fluids.

Fluid management should be cautious: 10–20 mL/kg isotonic bolus only if shocked; replace deficit + maintenance over 48 hours (avoid rapid osmolar shifts).

3c. Explain the mechanism behind the drop in potassium. Describe your repletion strategy, including timing relative to insulin therapy.

Answer

3c.

Mechanism:

Correction of acidosis + insulin therapy drives potassium intracellularly; **unmasking of total body depletion** from osmotic diuresis.

Strategy:

- Do **not** start insulin if K^+ <3.5 until corrected.
- Add 40 mmol/L KCl to fluids
- Maximum peripheral infusion 0.5 mmol/kg/h with cardiac monitoring.
- Frequent rechecks (q1–2h).

Further Information:

You start a insulin infusion. Six hours into the treatment, the child develops recurrent vomiting, hypertension (BP 118/82), and unilateral pupil dilatation. PICU team is contacted, but transfer will take 45 minutes.

3d. Outline your immediate management priorities at this stage, including specific interventions for the suspected complication.

Answer

3d.

Suspect **cerebral oedema** → elevate head, restrict fluids to maintenance rate, give **mannitol 0.5–1 g/kg IV** or **3% hypertonic saline** (2–5 mL/kg).

Avoid further rapid osmolar changes; monitor corrected Na closely.
Continue insulin at 0.05–0.1 unit/kg/h (after fluids started).
PICU admission/transfer, neurological monitoring, possible intubation if deteriorating.

4. Acute Deterioration in an Elderly Patient with Chronic Lung Disease

Scenario:

A 78-year-old man with known severe **COPD** (on home oxygen 2L/min) presents to the AED with increasing SOB and drowsiness for 24 hours following a presumed chest infection. He is a heavy ex-smoker.

Vital signs: RR 26/min, SpO_2 88% on 4L oxygen via nasal cannula, BP 148/82 mmHg, HR 112 bpm, GCS 13 (confused). Arterial blood gas on **4L O_2: pH 7.28**, pCO_2 10kPa, pO_2 9kPa, HCO_3^- 36 mmol/L, lactate 2.1 mmol/L.

4a.

Interpret the blood gas. What type of respiratory failure is this, and what are the key pathophysiological mechanisms contributing to his current decompensation?

Answer

4a.

This is **acute on chronic Type 2 (hypercapnic) respiratory failure** with acute respiratory acidosis on background chronic compensated respiratory acidosis (raised bicarbonate).

Pathophysiology: Chronic airflow limitation and V/Q mismatch in COPD lead to CO_2 retention. Acute infection increases dead space, CO_2 production, and work of breathing, culminating in respiratory muscle fatigue and alveolar hypoventilation.

Further information: He is started on controlled oxygen therapy and bronchodilators. You initiate non-invasive ventilation (NIV). After 90 minutes, he becomes **more drowsy (GCS 10)** with shallow breathing. Repeat ABG: pH 7.22, pCO_2 12.3kPa, HCO_3^- 38 mmol/L. He is now **hypotensive** (BP 92/58 mmHg).

4b. What is happening with his respiratory status? Outline the indications for escalation of therapy and the key settings you would use on NIV (or reasons to abandon it).

Answer

4b.

He is **failing NIV** (worsening hypercapnia, acidosis, and reduced GCS).

Indications for **intubation**: Persistent pH <7.25, deteriorating mental status, haemodynamic instability.

NIV settings (if attempting further): IPAP 16–20 cmH_2O, EPAP 5–7 cmH_2O, titrated to patient comfort and pCO_2 reduction. However, early intubation is appropriate here.

Further information: He is successfully intubated and mechanically ventilated. Plateau pressure is 34 cmH_2O. Repeat ABG shows **pH 7.19, pCO_2 11.7kPa** despite optimisation of ventilation.

4c. Discuss the concept of permissive hypercapnia in this patient. What ventilator adjustments and adjunctive measures would you implement to manage the persistent hypercapnia whilst avoiding ventilator-induced lung injury and dynamic hyperinflation?

Answer

4c.

Permissive hypercapnia: Strategy of accepting elevated pCO_2 (up to 10.6 - 13.3 kPa) to avoid high ventilator pressures and allow lung protection, as long as pH remains >7.20–7.25. It reduces risk of barotrauma in COPD patients.

Ventilator adjustments and measures:

- Low tidal volume (5–7 mL/kg ideal body weight).
- Low respiratory rate (8–12 breaths/min) to maximise expiratory time and reduce auto-PEEP.
- I:E ratio of 1:3 or 1:4.
- Measure intrinsic PEEP (expiratory pause manoeuvre) and set extrinsic PEEP ~80% of auto-PEEP.
- Pressure-controlled ventilation mode preferred initially.
- Adjuncts: Deep sedation ± paralysis (to reduce patient-ventilator asynchrony), aggressive bronchodilators, steroids, and bicarbonate infusion if pH critically low (<7.15).
- Monitor for dynamic hyperinflation (high plateau pressure, hypotension from reduced venous return).

Further information: Six hours later, he develops **atrial fibrillation with rapid ventricular response and worsening shock.**

4d. Discuss potential complications of prolonged hypercapnic respiratory failure and mechanical ventilation in this patient. What is your comprehensive ongoing management and disposition plan?

Answer

4d.

Complications:

- Dynamic hyperinflation, barotrauma (pneumothorax), ventilator-associated pneumonia, critical illness polyneuropathy, difficult weaning, haemodynamic instability, and atrial arrhythmias (as seen).
- Hypercapnia effects: cerebral vasodilatation, somnolence, and acidosis-related myocardial depression.

Management & Disposition:

- Treat underlying infection and COPD exacerbation (antibiotics, steroids, bronchodilators).
- Rate control or cardioversion for AF with shock.
- Daily sedation breaks and spontaneous breathing trials.
- Early involvement of respiratory/ICU team; consider tracheostomy if prolonged weaning anticipated.
- ICU admission with goals-of-care discussion given advanced COPD.

5. A Burn Patient

Scenario:

A 48-year-old man (weight 82 kg) is rescued from an **enclosed factory fire.** He has deep burns involving the face, neck, chest, and both upper limbs (estimated **35% TBSA).** He was trapped for 30 minutes. He has soot in the nostrils, hoarse voice, and cherry-red skin.

Vital signs: **BP 92/58 mmHg, HR 138 bpm, RR 34/min, SpO_2 94% on 15L** non-rebreather, **GCS 13** (agitated and confused). Initial ABG on arrival: **pH 7.08**, pCO_2 3.4kPa, HCO_3^- 8 mmol/L, pO_2 11.3kPa (on high FiO_2), lactate 14.2 mmol/L, base excess -22.

5a. Interpret the blood gas and explain the likely acid-base disorder. What toxicological emergency should you suspect in this scenario, and how would you confirm and treat it?

Answer

5a.

ABG: Severe **high anion gap metabolic acidosis** (lactate-driven) with respiratory compensation.
Suspected toxicity: **Cyanide poisoning** (common in enclosed fires with burning plastics/synthetics). Clinical clues: persistent lactic acidosis despite resuscitation, altered mental status, and cherry-red appearance.

Treatment:

- 100% oxygen.
- Hydroxocobalamin 5 g IV (repeat if needed).
- Sodium thiosulfate as alternative/second line.
- Supportive care with aggressive fluids and vasopressors.

Further information: You suspect **cyanide poisoning** and administer hydroxocobalamin. You start Parkland-guided fluid resuscitation with balanced crystalloid. After 4 hours, he has received 3.8 L of fluid. His airway is deteriorating (increasing stridor and swelling). **BP is 78/45 mmHg**, lactate remains high at 11.8 mmol/L, and he is becoming obtunded **(GCS 9).**

5b. Outline your fluid resuscitation strategy so far and any necessary adjustments. Why is this patient at high risk of both under- and over-resuscitation?

Answer

5b.

Fluid strategy: Parkland formula = 4 mL × 82 kg × 35% = **11,480 mL** in 24 hours (first half in the first 8 hours ≈ 720 mL/h). Use balanced crystalloid; consider increasing volume by 30–50% due to inhalation injury + cyanide.

Risks:

- **Under-resuscitation**: Ongoing capillary leak + cyanide-induced mitochondrial dysfunction → persistent shock and lactic acidosis.
- **Over-resuscitation**: Massive fluid shifts leading to oedema, compartment syndromes, and pulmonary oedema.

Adjustments:

Titrate to urine output (0.5–1 mL/kg/h), lactate clearance, and haemodynamic response. Early vasopressors if fluid unresponsive.

Further information: You decide to secure the airway. The patient is now hypoxic and acidotic with **pH 7.05.** You prepare for **rapid sequence intubation.**

5c. What is the major contraindication regarding the use of suxamethonium (succinylcholine) in this patient? What alternative agents would you choose for induction and paralysis, and why?

Answer

5c.

Contraindication of suxamethonium:

Burn patients are at high risk of **hyperkalaemic cardiac arrest** due to upregulation of extrajunctional acetylcholine receptors. This risk starts as early as 24 hours post-burn but can occur earlier in major burns with muscle injury.

Preferred agents:

- Induction: Ketamine (haemodynamically stable choice) or etomidate.
- Paralytic: Rocuronium (1.2 mg/kg) — non-depolarising agent, safer in burns. Prepare for difficult airway (burned face/neck).

Further information: He is successfully intubated. At 14 hours post-burn, despite ongoing resuscitation, he develops **abdominal distension with bladder pressure 29 mmHg, anuria, and worsening hyperkalaemia (K^+ 6.4 mmol/L).**

d. Discuss the interplay between fluid overload, cyanide toxicity, and acid-base disturbance contributing to his current deterioration. Outline your ongoing management and escalation plan.

Answer

5d.

Interplay:

Cyanide causes profound lactic acidosis (inhibits cellular respiration). Fluid resuscitation is essential but leads to oedema and abdominal compartment syndrome, worsening AKI and hyperkalaemia. Acidosis further exacerbates hyperkalaemia.

Ongoing management:

- Restrict crystalloids, consider albumin.
- Vasopressors/inotropes.
- Renal replacement therapy for hyperkalaemia, acidosis, and anuria.
- Decompressive laparotomy if compartment syndrome confirmed.
- Transfer to Burns ICU. Continuous monitoring of lactate, electrolytes, and compartment pressures.