

FEx Net 2022 Collated SAQs: Clinical Chemistry

Answers

1. **ABG**
2. **ABG**
3. **ABG**
4. **ABG**
5. **ABG**
6. **Collapse**
7. **Collapse**
8. **Confusion**
9. **Confusion**
10. **Confusion**
11. **LFTs**
12. **Seizure**
13. **VBG**
14. **VBG**
15. **VBG**
16. **VBG**
17. **Vomiting**
18. **Vomiting**
19. **Vomiting**
20. **Vomiting**

Question 1

1. Hyponatremia – moderately low; corrected for glu – 127 (1 mark)

- Likely GI losses or other cause (1 mark)

K 3.5 – corrected for pH => 2.5 (1 mark)

- GI losses likely (1 mark)

Hypochloremia - ??? (1 mark)

- Vomiting, electrical neutrality (1 mark)

Ionised Ca – severe hypocalcaemia (1 mark)

- Pancreatitis, renal failure, rhabdo a possibility (1 mark)

2. 1 mark for comment, 2 marks of differentials

High anion gap metabolic acidosis (1 mark)

- Ketones – DKA, alcoholic (1 mark)
- Lactate (type A or B with liver failure) (1 mark)

Inadequate respiratory compensation (expect CO₂ to be lower) (1 mark)

- Decreased consciousness eg alcohol, head injury, other drugs (1 mark each)

Delta ratio $(42-12)/(22-11) = v.$ high almost (1 mark)

No differential acceptable

Lactate high

- Type A hypoperfusion (1 mark)
- Type B – liver failure (1 mark)

Hyperglycaemia

- DKA (1 mark)

Question 2

- 1- List 4 metabolic abnormalities including any calculations required. (4 marks)

Sever metabolic acidosis (Low HCo3)

Expected Resp compensation with low Pco2 but actual Pco2 very high

→ **Concurrent Resp acidosis**

Anion Gap: $\text{Na} - (\text{Cl} + \text{HCo}_3)$

$$136 - (90 + 9) = 36 \rightarrow \text{HAGMA}$$

Delta Ratio : $\Delta \text{AG} / \Delta \text{HCo}_3 = 25 / 15 = > 1$ Therefore Pure HAGMA

Urea/Creatinine ratio $18 / 0.12 = > 100$

→ **Pre-renal failure**

- 2- What's the A-a gradient for the above patient: (1 mark)

A-a gradient: $150 - 1.2 \times \text{PCO}_2 - \text{PO}_2$

$$150 - 86 - 60 = 0 \text{ Normal}$$

Hypoxia → Hypoventilation

- 3- List 5 potential causes for her presentation: (5 marks)

CNS Depression: Toxin (Benoz, opioid, alcohol, ect)

Head Injury

Sever sepsis

Airway obstruction: Asthma

Metabolic: DKA with cerebral oedema

Neuromuscular Weakness: Organophosphate Poisoning
Botulism

Question 3 (19 marks)

A 30 year old man presents to your ED with a 24 hour history of increasing weakness.

An ABG and electrolytes are performed.

ABG

FiO2	21%	
pH	7.26 mmHg	(7.35-7.45)
pCO2	29 mmHg	(35-48)
pO2	101 mmHg	(83-108)
HCO3	13 mmol/L	(21-28)
BE	-13 mmol/L	(-1.5-3.0)

ELECTROLYTES

Na	137 mmol/L	(136-146)
K	1.8 mmol/L	(3.9-5.2)
CL	116 mmol/L	(95-110)
Urea	7.8 mmol/L	(3.1-8.1)
Creat	86 mmol/L	(60-110)
Glc	4.0 mmol/L	(3.9-5.8)
CK	1975 U/L	(29-168)
Trop T	20 ng/L	(<30)

i. Describe and interpret his blood results (8 marks)

- **NAGMA**
 - $AG = Na - (Cl + HCO_3) = 137 - (116 + 13) = 8$
 - Causes of NAGMA include pancreato/uretero-enterostomies, small bowel fistulae, excess chloride, diarrhoea, carbonic anhydrase inhibitors, RTA, Addison's.
- **Appropriate respiratory compensation**
 - Expected $CO_2 = 1.5 \times \text{bicarb} + 8 = 28$
- **Severe hypokalaemia** likely cause of weakness - ? secondary to chronic diarrhoea, may be hereditary
- **Mild elevation in CK** – likely just dehydration, could be due to: infection, hypokalaemia, metabolic (thyroid storm, pheochromocytoma). Not severe rhabdomyolysis

ii. List your 3 immediate management priorities (3 marks)

- Move to monitored bed. Need for ECG
- Needs potassium replacement in monitored environment, likely to need central line for IV potassium replacement. Need to contact HDU
- Need for IVF as likely element of pre-renal failure in view of mild CK rise

- iii. An internal jugular central venous catheter is placed to facilitate potassium replacement. List 4 early and 4 late complications of internal jugular central venous catheter placement (8 marks)

Immediate/Early

- pneumothorax
- failure to locate vein
- accidental arterial puncture
- haemothorax
- haematoma
- arrhythmia
- thoracic duct injury
- guide wire embolus
- air embolus
- haemopericardium and tamponade
- pneumothorax
- catheter blockage
- chylothorax
- catheter knots

Late

- infection (no difference in the rate of catheter-related bloodstream infections between the IJ, SC and Femoral sites -> 2.5 infections/ 1000 catheter days)
- catheter fracture
- vascular erosion
- vessel stenosis
- thrombosis
- osteomyelitis of clavicle

Question 4 (12 marks)

A 78-year-old woman presents to your tertiary hospital with nausea, vomiting, loose bowel motions and malaise. She has a background of atrial fibrillation, hypertension and type II diabetes. Her current medications include: Candesartan 16mg daily, apixaban 2.5mg bd, dapagliflozin 10mg od, lercanidipine 10mg daily, bisoprolol 10mg daily, simvastatin 40mg daily and linagliptin-metformin 2.5mg-1g I bd. She has not taken any over the counter medications.

Her observations on arrival are: BP 141/72, HR 90, RR 28, saturations 93% on RA, temperature 35.2 degrees Celsius. She is alert and oriented with a clear chest and soft-non-tender abdomen. Arterial blood gas and renal function are performed; the results are shown below:

FiO2	0.21	
pH	6.75	(7.35-7.45)
pCO2	24mmHg	(32-48)
pO2	86 mmHg	(83-108)
Bicarbonate	3 mmol/L	(22-32)
Sodium	132 mmol/L	(135-145)
Potassium	6.4 mmol/L	(3.5-5.2)
Chloride	102 mmol/L	(95-110)
Glucose	15.7 mmol/L	(3.0 -7.8)
Lactate	15.0 mmol/L	(0.5-2.2)
Urea	14.6 mmol/L	(3.0-8.0)
Creatinine	237 µmol/L	(40-90)

a). Analyse the above results, listing the major abnormalities and their clinical implication. Include calculations where relevant. (7 marks)

Abnormality	Interpretation
Profound acidaemia	Metabolic with bicarbonate ⁻ of 3
Mildly low pCO2 (24)	Expected CO2= $1.5 \times \text{HCO}_3^- + 8 = 12.5$. inadequate respiratory compensation
High anion gap	$= \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) = 27$ Delta ratio = change in AG/change in HCO3 ⁻ = $15/21 = 0.71$. Predominantly HAGMA + element of NAGMA HAGMA due to combination of metformin, lactate and uraemia.
Hyperkalaemia	corrected to ~3.4 (Correcting for pH = ~0.5 K ⁺ change for every 0.1 of pH) Predominantly due to acidaemia, ± contributed to from AKI
Elevated lactate	Type B – in this setting most likely B2 (drugs) secondary to metformin

Mild hyponatraemia	Correcting for hyperglycaemia = $132 + (15.7-5) \cdot 3 = 135 = \text{in normal range}$
Urea 14.6 and creatinine 237 = acute renal failure	Urea : creat ratio = 60 = acute renal injury (not just pre-renal) No prior history of renal failure and current medication list suggest AKI

b). Give the most likely underlying aetiologies of the major acid-base abnormality. (1 mark)

Metformin-associated lactic acidosis + Acute renal failure impairing excretion of metformin and also contributing to acidaemia

c). List your management priorities. Give drug doses and end points where relevant. (4 marks)

Correct critical acidaemia: initially 1mEq/kg 8.4% sodium bicarb (50-100 mL)
Urgent haemodialysis, likely in ICU
Fluid resuscitation: start with 1L Hartmann's and reassess perfusion/urine output.
Treat underlying precipitant: hypothermic – culture and consider empiric antibiotics such as piperacillin/tazobactam 4.5g IV.
Oxygen, titrate to sats >94%
Treat hyperkalemia (relative hyperkalemia is secondary to acidosis therefore correction of acidosis should be priority)

Question 5

Variable	Formula Used	Result <i>Not required for marks as this is not a maths test</i>	Clinical Implication in this case
Expected CO ₂	$1.5 \times \text{HCO}_3 + 8$	24.5	Severe Respiratory acidosis is present
A-a gradient	Alveolar O ₂ = $713 \times 1 - 60/0.8$	<i>Alveolar = 640 gradient = 440.</i>	Marked A-a gradient – implies ineffective gas transfer – shunt/V/Q mismatch
Anion Gap	$\text{Na} - (\text{Cl} + \text{HCO}_3)$	28	HAGMA present (likely ketones given lactate not too high, could be renal failure or toxic alcohols)
Delta Gap	Change in AG/ change in HCO ₃	$28-12/24-11 = 16/13 = 1$	Pure HAGMA
Corrected Na ⁺	$\text{Na} + (\text{Gluc}-5)/3$	$125 + 10 = 135$	No hyponatraemia present

1 mark total for formula and result, 1 mark for clinical implication

2. Goals:

Achieve synchrony – sedate further +/- drug paralyse patient (2 marks)

Improve Ventilation – increase RR to 16-20 (1 mark)

Improve Oxygenation –

increase PEEP to 10-15cm H₂O (1 mark)

Tidal volume to 480-500mls (6mls/kg) (1 mark)

Question 6 18 marks)

A 67 year old male patient was brought into the ED by his family after he collapsed at home. His past medical history includes dialysis dependent chronic renal failure (secondary to diabetic nephropathy), hypertension, ischaemic heart disease and congestive cardiac failure.

His initial vital signs in ED are as follows:

GCS 15

BP 80/50

P 80

RR 30

SpO₂ 90% on air

(a) List 4 specific point of care / bedside tests which are indicated in this patient and state your justifications (8 marks)

Blood glucose level - hypoglycaemia

Serum K⁺ - hyperkalaemia

ECG – dysrhythmias secondary to hyperkalaemia

Bedside U/S – pericardial tamponade, pulmonary oedema

CXR – pulmonary oedema

Blood gas – acid-base status → expect metabolic and respiratory acidosis

Lactate – hypoperfusion, sepsis

Hb – critical anaemia

(b) An ECG is performed. List 3 diagnostic abnormalities in his ECG and state your unifying diagnosis (4 marks)

Peaked T waves

Abnormally wide QRS

Superior axis

Sinoventricular rhythm / sine-wave morphology

Diagnosis : critical hyperkalaemia

(c) In view of the ECG findings, state 3 priorities of treatment to address this specific abnormality and the critical interventions you would institute to achieve these goals (6 marks)

Stabilise myocardium : calcium chloride or calcium gluconate

Move K⁺ from extracellular to intracellular space : salbutamol, insulin / dextrose, bicarbonate

Remove K⁺ : haemodialysis

Pass marks a 7/8 b 3/4 c 4/6 total 14/18

Question 7

1.

Hx organ failure – heart, renal, liver – causes hypervolemic hypoNa

Drug history – drugs that cause SIADH, diuretics

Mental health history – drugs that cause SIADH, water intake for psychogenic polydipsia

Intercurrent illnesses – eg sepsis, pancreatitis, gastroenteritis– causes of hypovolemic hypoNa

Malignancy – SIADH

(other things may ne acceptable)

Pass 3 of 5

2.

Urea/creat – renal failure

BSL – cause of dilutional hyponatremia

TFTs- hypothyroidism

CXR – lung malignancy – SIADH

LFTs – liver failure

Urine Na – elevated in SIADH >20-40

Serum K – for typical low Na / high K ratio in Addison's disease

(1 mark for test plus reason no ½ marks)

Pass 3 of 5

3.

Midazolam / diazepam 5mg bolus aiming to terminate seizure, repeat if needed

3% saline bolus – approx. 2mL/kg bolus aiming for seizure termination and small rise in Na (about 5)

1 mark for drug, 1 mark for rest

Pass 2 of 4

TotAL PASS 8/14 corrects to 5.5/10

Question 8 (12 marks)

A 13 year old girl presents to the emergency department with confusion and abdominal pain. She has a background of type 1 diabetes requiring insulin treatment. She has had 3 days of vomiting and 2 days of diarrhoea prior to this presentation. It is unknown if she has been taking her insulin during this current illness. She is afebrile, has a heart rate of 124, and a blood pressure of 120/70 with a central capillary refill time of 4 seconds.

Her initial venous blood gas results on room air are:

pH	6.93	Na	124 mmol/L
pCO ₂	9 mmHg	K	6.2 mmol/L
pO ₂	44.9 mmHg	Cl	79 mmol/L
HCO ₃	2 mmol/L	Glc	43.5 mmol/L
BE	-25.5 mmol/L	Hb	162 g/L

- a. Quote four (4) appropriate formulae that assist the analysis the above blood results.

You are not required to insert the appropriate values or calculate results. **(4 marks)**

Formula 1: Anion Gap = $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$. Will also accept $(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$

Formula 2: Corrected $\text{Na}^+ = [\text{Na}^+] + [\text{Glucose}]/3$

Formula 3: Metabolic acidosis expected change in $\text{pCO}_2 = (1.5 \times \text{HCO}_3^-) + 8$

Formula 4: Serum K decreases by 0.3mEq/L for every 0.1U increase in pH above normal.

Formula 5: Delta ratio, Delta Gap = Anion Gap – Baseline Gap (i.e. 12)

Delta ratio = (Delta Gap (Increase in Anion Gap) / Delta HCO_3^- (Decrease in bicarbonate))

In DKA, ratio should be close to 1, due to loss of ketone acids in urine.

- b. State four (4) important principles regarding your fluid and electrolyte treatment of this patient while in ED, and give a justifying statement (which may include doses) for why it is an important principle. **(8 marks)**
- c. Rehydrate over 48 hours.
- a. Reason: patient 10% dehydrated and thus needs to be rehydrated slowly to ensure no rapid fluid/electrolyte shifts within body compartments. Fluid

rehydration should include maintenance plus rehydration deficit replacement. Small bolus is OK.

- d. Start insulin infusion early (without bolus).
 - a. Reason: need to start shifting glucose back into cells early in order to correct the acidosis (recommended rate $0.1\text{u/kg/hr} = 4\text{u/hr}$)
- e. Monitor glucose fall closely.
 - a. Reason (any of the following will be accepted): Often rapid falls in glucose are due to rapid fluid rehydration and thus the fluid rehydration rate may need to be slowed.
 - b. Theoretical rapid glucose fall may result in cerebral oedema.
- f. Monitor potassium closely.
 - a. Reason: fall in potassium will occur due to fluid rehydration plus addition of insulin. Need to ensure not too low to ensure cardiac activity is not compromised.

Pass marks a 3/4 b 6/8 total 9/12

Question 9

Answers:

1. Give 8 possible causes for his confusion. (8 marks) 6/8

Acute alcohol intoxication
 Alcohol withdrawal
 Post-ictal state (numerous causes)
 Head trauma – SDH/EDH/ICH
 Hepatic encephalopathy
 Infection – meningitis, encephalitis
 Korsakoff's syndrome – from severe vitamin deficiency
 Wernicke's encephalopathy
 Co-ingestion of other drugs of abuse, eg: opiates
 Thrombotic stroke, eg secondary to AF

Some bloods are taken as part of his diagnostic work-up:

2. Describe the Acid-base status above (including calculations), and give 3 relevant differentials for same. (8 marks) 6/8

Metabolic acidosis (Low bicarb)
 Cannot comment on respiratory component (not provided)
 $\text{Anion Gap} = 133 - (87 + 10) = 36 = \text{HAGMA}$

$\text{Gap:Gap} = \frac{(36-12)}{(24-10)} = 1.7 = \text{Pure HAGMA}$

$\text{Osmolar gap} = 298 - (1.86 \times 133 + 8.0 + 6.0 + 9) = 28$, therefore raised osmolar gap

Differentials:
 Alcoholic keto-acidosis
 Starvation keto-acidosis
 Consider poisoning with ethanol, ethylene glycol, methanol given high osmolar gap
 Renal failure
 Lactic acidosis
 Salicylate poisoning

Note: No marks for suggesting DKA

3. Describe and interpret the patient's LFT's. (6 marks) 3/6

Raised aminotransferases largely suggest alcoholic hepatitis:
 AST:ALT ratio approximately 2

AST 5-10 x normal

Markedly elevated GGT also supports this

ALP also normal which supports this

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Largely unconjugated hyperbilirubinaemia ?need to consider haemolysis

Albumin normal suggesting synthetic function in tact

Would need to see Coag profile also

Passmark: 15 /22 Adjust to ../10 please

Question 10

1.

Medications – new meds or changes in dose of drugs that are associated with hyponatraemia (ie thiazides, SSRIs, anticonvulsants)

GIT illness with volume loss – could produce hypovolaemic hyponatraemia

Hx of liver disease (or surrogate ie increasing abdominal distention – hypervolaemic hyponatraemia

Hx of CCF – hypervolaemic hyponatraemia

Hx excessive water intake – ie psychogenic polydipsia

Hx malignancy (lung or brain) – risk for development of SIADH

Hx of weight loss, nightsweats etc – possible undiagnosed malignancy

Hx of previous hyponatraemia

Really anything here could be ok ie Hx of thyroid dysfunction

Alcohol intake – seeking potomania

2.

Volume status (ie accept one of – peripheral oedema, ascites, pulmonary congestion) as evidence of hypervolaemic hyponatraemia

ALSO accept one of significant dehydration ie tachycardia, tachypnoea, dry mucous membranes etc as evidence of possible hypovolaemic hyponatraemia

Pulmonary findings of malignancy ie focal consolidative signs/crackles, unilateral effusion or better answers to be more specific to pulmonary malignancy ie unilateral hand wasting, HPOA, brachial plexopathy or Horner's syndrome to suggest apical lung malignancy

Goitre or other signs of thyroid disease (exophthalmus, tremor)

3.

TFTs – hypernatraemia causally linked to SIADH

Other electrolytes ie if K, Cl also low suggestive of potomania

(note not accepting osmolality or BSL as not useful in determining cause)

BNP would be weak here

Renal function and/or LFTs likely acceptable if justified through cause of hypervolaemic hyponatraemia (not just Chem20)

Urine osmol/Na – useful in identifying sodium wasting of SIADH

Cortisol – if suspected hypoadrenalism

CXR – seek malignancy/ other pulmonary process (abscess/empyema) – must state one path process

Other – CT Head – malignancy/abscess/ infarct

Sputum cytology – must state seek small cell lung cancer

4.

Comminuted fracture Rt femur

- Intertrochanteric fracture
- Spiral fracture of proximal femoral shaft with shortening and displacement (one mark for description – displacement/angulation)

5.

Lignocaine - accept 3-5mg/kg

Bupivacaine – accept 2-2.5mg/kg

6.

Open disclosure of events around fall and femoral fracture

Apologise for events and injuries sustained in hospital

Explain that incidents will be investigated through hospital processes

Ensure understanding of severity of father's presentation – severe hyponatraemia

Explanation of need for fixation of femoral fracture

Seek clarification around father's history and premorbid level of function

Seek clarification around pre-existing documentation around advanced health directives etc

Question 11

1.

Main abnormality: HAGMA

Justification: $AG = 135 - (102) = 33$

Most likely cause: Lactate (liver failure – impaired Cori cycle) or toxins

2.

Main abnormality: acute renal failure (intrinsic)

Justification: urea: creat ratio <100

Most likely cause: hepatorenal syndrome or toxins (CK, Mb etc)

3.

Main abnormality: Synthetic liver failure

Justification: Albumin and globulin both decreased

Most likely cause: Acute fulminant liver failure

4.

Main abnormality: Acute severe hepatitis

Justification: ALT/AST elevated $\gg$ membrane bound enzymes (GGT/ALP)

Most likely cause: Toxic hepatitis (paracetamol toxicity)

5.

Glucose – in liver failure exclude as cause of decreased consciousness

Paracetamol level – detectable paracetamol (>10) supports paracetamol toxicity as cause

Hepatic USS – Excludes structural cause ie CBD obstruction, PSC

Hepatitis serology

Question 12

A normally well 24 year old man is brought to your ED with increasing confusion and a seizure. A blood gas shows the following.

pH	7.31		
PCO ₂	51	mmHg	(35-45)
HCO ₃	22	mmol/L	(22-28)
Na	109	mmol/L	(135-145)
K	4.1	mmol/L	(3.5-4.2)
Cl	91	mmol/L	(95-110)
Glucose	10	mmol/L	(3.9-5.8)

- i. Detail the specific emergency treatment required and provide treatment aims/endpoints (3 marks)

- Hypertonic saline
- 3mls/kg of 3% - or sensible dose
- Over 30 mins - or sensible rate
- Clinical improvement - cessation of seizures and clinical recovery
- Raising serum Na and serum osmols by 2-3mmol/L

- ii. List 3 risk factors for the development of Osmotic Demyelination Syndrome (ODS) and write brief explanatory notes (6 marks)

Candidate number _____

- Serum sodium at presentation – most cases of ODS occur when initial [Na] < 105, and unusual for ODS to occur if initial Na > 120 mmol/L
- Duration of hyponatraemia – ODS rarely occurs if hypoNa has developed over only a few hours or up to a day (e.g. following ingestion of large volumes of water)
- Overly rapid correction – the increase over 24 or 48 hours appears more important than the hour-by-hour change. Safe 24 hour rise generally said to be about 5 mmol/L per day
- Other risk factors – especially groups who can develop ODS with less severe hypoNa and slower rates of correction
 - Alcoholism
 - Malnutrition
 - Liver disease
 - Hypokalaemia

iii. Hypertonic saline is also used in the management of raised intracranial pressure in traumatic brain injury (TBI). What dose/regime would you use and what target endpoints would guide your therapy (4 marks)

- Hypertonic saline 3% 150 mL bolus IV or 7.5% 75 mL bolus IV. Can repeat q 2-4 hours
- Targets/endpoints
 - ICP < 20 mmHg
 - Osmolality 300-320 mOsm/L
 - Sodium 145-150 mmol/L

iv. Briefly outline the advantages/disadvantages of hypertonic saline versus mannitol in TBI (3 marks)

- No good quality evidence of improved neuro or mortality outcomes for either drug
- HTS more effective in lowering ICP (failure rate of 16% for HTS vs 35% for mannitol)
- HTS has a more prolonged effect on ICP
- HTS “haemodynamically stable” compared to mannitol which causes osmotic diuresis → hypotension

Question 13

1.

Marked Respiratory Alkalosis (CO2 19)

Differential:

Pain

Hypoxia

Anxiety

Sepsis

2.

HAGMA

HAGMA – Anion Gap = $137 - (98 + 18) = 21$

Delta ratio $9/6 = 1.5$

(for this will accept both conclusions of pure HAGMA and possible concomitant met alkalosis from vomiting)

Lactic acidosis

- Hypoperfusion
- Liver failure

With low Ca, pancreatitis a big DDx

3.

CXR	?Subdiaphragmatic free air indicating perforated viscus
Lipase	Evidence of pancreatitis
Chem20	Exclude renal failure given marked hyperlactataemia, exclude evidence of fulminant hepatitis
COAGs	Synthetic liver failure (staging of severity of liver disease)
Many others likely acceptable	

Question 14

A 32 yr old Aboriginal woman presents to the ED with lethargy. She is previously well, and has not been seen by medical services for the previous 10yrs,

Her VBG is attached.

What is the most likely cause for the abnormalities on the VBG? Describe and interpret the abnormalities (5 marks)

Table might look something like this:

pH	Compensated metabolic acidosis. Expected $p\text{CO}_2 = 8 + (1.5 \times 12) = 26$ (about right) Anion gap = 30 – profoundly raised Delta ratio = $\text{AG} - 12 / 24 - \text{Hco}_3 = 18 / 12 = 1.5 = \text{RAGMA}$ i.e widened anion gap probably from uraemia (consider ketosis)
K	Mild hyperkalaemia ?artefactual/ ?renal failure
GLuc	Mild hyperglycaemia from untreated DM
Ca	Hypocalcaemia from renal failure
Hb	Anaemia would be consistent with renal failure ? other causes/ acute blood loss etc
Summary	Most likely cause is renal failure with uraemia, hyperkalaemia, hypocalcaemia and anaemia. In the setting of untreated diabetes.

Her creatinine is 1842 with a urea of 62

List 4 possible causes for these findings in a 32 yr old Aboriginal woman, with the most common cause first. (3 marks)

(3 points; at least one bold to pass)

Post strep GN
DMII nephropathy
 Other forms of GN
 Causes of ARF

She becomes agitated and appears to be getting ready to leave the ED. What would be some strategies to stop her leaving? (4 marks)

(4 points; bold required, need at least 3)

Get ALO/ILO
 Create rapport by asking where she is from, introducing oneself
 Explore: try to find why she wants to leave, can we assist with that, what are her concerns, what is her understanding of what is going on
 Appropriate body language
 Do not force eye contact
 Gender preference for doctor

Question 15

A 75yo man presents to your ED with a 2 day history of lower abdominal pain, fevers and malaise. You are unable to obtain more information.

Vital signs:

HR 110
RR 24
Temp 38.2
BP 95/55
GCS E3V4M6 13

A Venous Blood Gas and basic bloods are performed:

pH 7.12
pCO₂ 34
HCO₃ 13
Hb 124
Na 138
K 6.2
Cl 110
Creat 443

FBC:

Hb 121
WCC 15
Plt 4

Chemistry:

Urea 20
Creat 465
ALP 174
GGT 86
ALT 97
AST 372
LDH 1180

His previous liver and renal function tests have all been normal

Questions:

1. For the venous blood gas, calculate, showing your workings, the anion gap (2 marks)

$$\begin{aligned}\text{Anion Gap} &= \text{Na} + \text{K} - \text{HCO}_3 - \text{Cl} \\ &= 138 + 6 - 13 - 108 \\ &= 144 - 121 \\ &= 23\end{aligned}$$

Therefore, this is a HIGH Anion Gap Metabolic Acidosis

2. List 3 likely causes of the metabolic derangement in this patient (3 marks)

Lactic acidosis from shock – tissue hypoperfusion and possible sepsis

Uraemia/renal failure – pre-renal acute kidney injury

Other toxic effect of overdose, such as Metformin or salicylates etc (less likely)

3. What is the MOST likely unifying diagnosis in this patient, and what are the supporting features? (4 marks)

Sepsis with multi-organ failure, likely urinary source

Supporting features:

Evidence of multi-organ failure

Deranged LFTs

Haemodynamic compromise

Renal Failure

Thrombocytopenia – likely DIC

Elevated WCC – supports infective process/sepsis

Likely urinary source – lower abdominal pain, renal failure

A single slice of the patient's CT scan is shown. List 1 positive and 2 negative features, relevant to this patient's presentation (3 marks)

Positive Feature

Radio-opaque density in distal ureter at VUJ likely to represent stone – some evidence of dilated ureter proximal to this

Negative features

Aorta normal calibre – unlikely AAA

No evidence free gas or pneumatosis coli – unlikely ischaemic gut/perforation

Must get stone for full marks

Any other reasonable negative feature acceptable

4. List your 3 main management priorities, with justification, for this patient (6 marks)

Likely urosepsis with infected obstructed kidney

1. Fluid resuscitation/inotropic support aim MAP >65 in patient with severe sepsis
2. Broad spectrum ABs – early administration reduces mortality
3. Urgent operative intervention with Urology to insert stent/relieve obstruction – source control

Question 16

An 89 yr old lady presents with generalised weakness ,nausea and diarrhoea . She had a fall 3 days earlier and had broken her humerus, and was discharged for orthopaedic follow up as an outpatient. She appears dehydrated, BP 140/80, pulse 80 regular, and is afebrile.

Her venous blood gas is below :

Venous Blood pH POCT	7.073
Venous Blood pO2 POCT	23.8 mmHg
Venous Blood pCO2 POCT	48.2 mmHg
Venous Blood O2 Saturation POCT	33.2 %
Venous Blood HCO3 POCT	13.4 mmol/L
Venous Blood Base Excess POCT	-14.8 mmol/L
Venous Blood Oxyhaemoglobin POCT	32.8 %
Venous Blood Inspired Oxygen POCT	21 %
Venous Blood Haemoglobin POCT	97 g/L
Venous Blood Reduced Haemoglobin POCT	66.0 %
Venous Blood Methaemoglobin POCT	0.4 %
Venous Blood Carboxyhaemoglobin POCT	0.8 %
Venous Blood Creatinine POCT	179 umol/L
Venous Blood Sodium POCT	126 mmol/L
Venous Blood Potassium POCT	3.2 mmol/L
Venous Blood Chloride POCT	100 mmol/L
Venous Blood Calcium Ionised POCT	1.25 mmol/L
Venous Blood Glucose POCT	5.6 mmol/L
Venous Blood Lactate POCT	1.1 mmol/L

(a) Please interpret the blood gas results, what are the likely causes of her acidosis ? (5 marks)

Answer :

Anion gap : $126 + 3.2 - 13.4 + 100 = 15.8$, therefore mildly raised anion gap (however, ?unlikely that this is the only cause of severe acidosis, ?co-existent non-anion gap metabolic acidosis)

Likely acute on chronic renal failure (causing mild HAGMA + also co-existent diarrhoea causing low potassium + normal anion gap metabolic acidosis)

(b) List 5 further investigations you feel are indicated, justify your responses ? (5 marks – need to justify, otherwise no mark for that particular answer).

Further Ix of fall : CT brain – as lethargy and vomiting

Ix of hyponatraemia : - formal UEC, serum and urine osmo , urine sodium

Ix of diarrhoea : ?stool culture .

MSU

CXR

ECG – hypokalaemia

Question 17 (15 marks)

A 40 year old developmentally delayed patient is brought to your ED with vomiting. She had PEG inserted 4 weeks ago for feeding. On arrival her GCS is 13 (E4 V3 M6) which is normal for her according to the carers. Sats 95%, RR 13, P 90 BP 140/70, she weighs approximately 40kg. She is on no regular medications.

A 12 LEAD ECG IS SHOWN IN THE PROPS BOOKLET, PAGE XX

- i. Please describe the ECG and provide 3 possible diagnoses (5 marks)

SR rate approx 80

Diffuse ST depression (all chest leads, inferior and lateral) with T wave inversion

Prominent U waves

(pseudo) Prolonged QT 480 uncorrected, 550 corrected (do not need values)

Differential:

Hypokalaemia – pathognomic

Ischaemia (but unusual as widespread ST depression)

Any cause of long QT – drugs (antis) for example. Should name a drug

- ii. Her clinical exam is unremarkable other than dry mucus membranes, bloods including a VBG are taken. The ambulance crew have NOT placed an IVC. Describe and interpret the VBG and provide a likely differential (5 marks)

pH 7.44 (7.35 – 7.45)

pCO₂ 47 mmHg (35 – 45)

HCO₃ 42 mmol/L (22 – 28)

BE xxxx (-3 - + 3)

K 2.0 mmol/L (3.4 – 5)

Na 155 mmol/L (134 – 146)

Cl 129 mmol/L (98 – 106)

Glu 7.4 mmol/L 3.5 – 5.5)

Severe / moderate respiratory acidosis and severe metabolic alkalosis with normal pH. (alkalosis likely pathological process as much more severe)

Severe hypernatraemia

Severe hypokalaemia – life threatening

Differential:

Conns / hyperaldosteronism

Cushings

PEG feed error

Dehydration (severe)

(Note: administration of chloride or sampling error unlikely given NO ivc present)

iii. Outline your immediate management of this case (5 marks)

Ensure patient monitored – risk of VT / VF

Potassium replacement – likely need CVC – 10mmol /hr periph, higher if central access

Check urinary Cl – high in mineralocorticoid excess, low in dehydration (former resistant to saline, latter saline is treatment)

Check Mg –often severely deplete with hypokalaemia

Check serum Cl – high in mineralocorticoid excess

Monitor EUC – 4 hrly

High risk for deterioration – discussion with family / next of kin regards treatment limitations

Disposition – monitored bed, likely HDU / ICU

Question 18

A 62 year old male with a history of chronic pancreatitis presents to the Emergency Department with several days of nausea and vomiting. His biochemistry profile is attached.

Interpret the abnormalities in the results(3 marks)

a) Severe metabolic alkalosis with respiratory compensation

High anion gap (approx. 31) metabolic acidosis

Profound hypochloraemia

Gastric losses and fluid depletion causing chloride loss and metabolic alkalosis

Metabolic acidosis secondary to renal failure (acute? Acute on chronic?) +/- sepsis from pancreatitis and/or gastro-enteritis

CO₂ retention as compensation for severe metabolic alkalosis

Give likely underlying causes for the main metabolic acid-base disturbances (3 marks)

a) Causes of a hypochloraemic metabolic alkalosis:

Recurrent vomiting

Gastric acid loss,

Diuretic-induced alkalosis (loop or thiazide diuretics)

Posthypercapnic metabolic alkalosis.

CLD, cystic fibrosis, and laxative abuse are also potential causes.

Describe the clinical features of severe hypocalcaemia (2 marks)

a) Carpopedal spasm, tetany, seizures, decreased cardiac function, or prolonged QT interval

Outline your management of symptomatic hypocalcaemia (2 marks)

a) Intravenous calcium replacement with either 10% calcium gluconate or 10% calcium chloride, followed by a calcium infusion.

Hypomagnesaemia should also be treated, with IV magnesium supplementation

Question 19

A 60 yr old man with a past history of type 2 diabetes, presents after being found on the floor by his elderly mother . He has recently been unwell with vomiting for the last 3 weeks. 12 marks

His vital signs are as follows :

BP 65/30 , GCS 13, pulse 120 regular, oxygen saturations 100% on 15 L NRM.

His arterial blood gas is attached.

Interpret the abnormalities in the above results (4 marks)

Severe metabolic acidosis

Some respiratory compensation (not fully compensated, expected CO₂ 13)

Hyperglycaemia , ?DKA

Renal impairment (Cr 246)

Severe metabolic acidosis , multifactorial : likely sec DKA, ARF .

How would you initially manage these abnormalities ? (3 marks)

IV fluid bolus -500ml – 1 L initially, then repeat as needed (note severe hypotension)

May need inotropic support

Insulin infusion 5 units/ hr, aim slow reduction of BSL

K⁺ replacement once K⁺ < 4

The patient's GCS deteriorates to 5 and he begins vomiting, what is your plan with regards to intubating this patient, and what ventilation strategy would you employ ? (5 marks)

- prepare for possible arrest
- have fluids and inotropes running prior to intubation
- most senior doctor intubating, video larygscopy if available
- drugs : ketamine 1mg/kg, suxamethonium
- high ventilation rate, as need to maintain low CO₂ to avoid worsening acidosis.
- early referral to ICU for management of ventilation on ICU ventilator.

Question 20

- 1. Describe the 4 key abnormalities shown in this blood gas. Include any calculations performed in your answer. (4 marks)**

Severe metabolic acidaemia. HAGMA. With appropriate respiratory compensation. (need to add calculation for resp compensation)

Severe hyponatraemia however corrected for glucose Na equals $106 + (82-5)/3 = 132$ which is close to normal range

Severe hyperkalaemia. Expected K given acidaemia $4 + 2.5 = 6.5$

Extreme Hyperglycaemia at 82 - consistent with DKA or a HHS pattern in a Type 2 diabetic/

- 2. What is the likely diagnosis? (2 Marks)**

Given the extremely high glucose it is likely HHS in a T2DM but DKA is also possible.

- 3. List 5 immediate management priorities for this patient: (5 Marks)**

1. Fluids- This patient is likely to be severely dehydrated. Aim correction over next 24 hours. Example N/S 500ml/hr for first 2 hours. Then 250ml/hr next 4 hours.
2. Insulin- 0.05-0.1units/kg/hr. No bolus.
3. Electrolytes- Watch K, will need to add extra potassium as pH improves and blood K levels drop.
4. Seek and treat precipitant of current state
5. Anticipate and treat complications eg cerebral oedema.
6. Disposition to ICU as requiring close observation and possibility for deterioration. Other answers at examiners discretion.