## VAQ 2008.2.3

An 83 year old woman presents with a three day history of malaise and polyuria. She has a past history of Type 1 Diabetes and Hypertension. Her observations are:

| | | |
|---|---|---|
| HR | 100 | /min |
| BP | 183/65 | mmHg supine |
| GCS | 14 | M6, V5, E3 |
| $O_2$ saturation | 100% | $O_2$ 6 L/min via Hudson mask. |

| | | | Reference Range |
|---|---|---|---|
| Na | 125 | mmol/L | (135-145) |
| K | 6.0 | mmol/L | (3.2-4.5) |
| Cl | 81 | mmol/L | (98-106) |
| Bicarbonate | 7 | mmol/L | (22-28) |
| Urea | 25.0 | mmol/L | (3.0-8.0) |
| Creatinine | 262 | µmol/L | (50-100) |
| Glucose | 54.5 | mmol/L | (3.5-5.5) |
| Osmolality | 337 | mmol/Kg | (275-295) |

This lady most likely has DKA, with a history of IDDM, polyuria, and lab evidence of hyperglycaemia and HAGMA. She will require treatment with fluids and insulin, with careful surveillance of serum potassium, and assessment for an underlying precipitant. High blood pressure with a wide pulse pressure is noted which may reflect an underlying precipitant or pre-existing condition.

**Na**

- moderate hyponatraemia
  - corrected for hyperglycaemia
  - Na + (delta Glu / 3) = 125 + 16 = 141
  - i.e. **pseudohyponatraemia**

**K**

- **moderate hyperkalaemia**
  - increased intake
    - endogenous
      - rhabdo/TLS
    - exogenous
      - potassium supplement – oral / IV
  - transcellular shift
    - **acidosis** (implied by low bicarbonate and clinical stem)
  - reduced output
    - **renal impairment**
      - acute implied by urea/creatinine
      - may have chronic impairment – age/hypertension
    - potassium sparing diuretics
      - thiazide/spironolactone (check meds)

**Cl**

- moderate **hypochloraemia**
  - corrected for hyperglycaemia – same as above
  - 81+16 = 97 = minimal change / pseudohypochloraemia
  - likely reflects passive shift to maintain electroneutrality
  - suggests against NAGMA (hyperchloraemia expected)

**Osmolarity**

- 1.8*Na+glu+urea+9 = 341 (using corrected sodium)
- *(there are at least 13 formulae for this – this is the most accurate (actually 1.86*Na, I use 1.8 for simplicity) but use whichever you like)*
- osmolar gap <10 suggests against toxic alcohols

**Bicarb**

**severe depletion**

**implies metabolic acidosis**

**AG = 125-81-7 = 37 = high AG**

**HAGMA**

C – cyanide – not suggested

U – uraemia present, likely some contribution

T – toluene – expect hypokalaemia, stem not suggestive

E – ethanol – not suggested

**D – DKA – overwhelmingly most likely**

I – iron/isoniazid – not suggested

M – methanol – not suggested

P – propylene glycol – not suggested

L – lactate – may contribute, check level

E – ethylene glycol – not suggested

S – not suggested

**Urea**

**moderately raised**

pre/postrenal azotaemia – likely **hypovolaemia** and prerenal azotaemia

protein load (GI bleed) – not suggested

**Creatinine**

**moderately raised**

ketone interference with assay likely (and consequent urea:creatinine higher and more suggestive of prerenal azotaemia)

**renal impairment**

**acute – likely from stem**

**chronic**

**Glucose**

**severe hyperglycaemia**

**insulin deficiency**

**absolute – IDDM (known)**

relative – NIDDM

consider HHS with extremely high glucose but short history/acidaemia not consistent

**Osmolality**

**hyperosmolar**

**haemoconcentration**

consistent with DKA and osmotic diuresis

**Interpretation**

**Overall most in keeping with DKA**

**hyperkalaemia** expected to resolve with insulin treatment – usually low total body potassium

ECG changes of hyperkalaemia may indicate specific treatment required

**DKA is a life threatening condition requiring urgent treatment**

initial insulin administration and fluid therapy

controlled correction of metabolic abnormalities over 2-3 days is safest in elderly patient to avoid fluid overload

cerebral oedema

underlying precipitant should be actively sought