VAQ 2010.1.7 (ABG)

A 45 year old woman with a past history of depression presents to your emergency department with 2 weeks of nausea, weakness and lethargy. There has been no vomiting or diarrhoea.

Serum biochemistry and arterial blood gas

			Reference Range
FiO_2	0.21		
pH	7.60		7.35-7.45
pCO_2	41	mmHg	35-45
pO_2	80	mmHg	80-95
Bicarbonate	40	mmol/L	22-28
Base excess	16		-3 - +3
O_2 saturation	96	%	> 95
Na^+	119	mmol/L	134-146
K^+	2.1	mmol/L	3.4-5
Cl^-	67	mmol/L	98-106
Glucose	6.4	mmol/L	3.5-5.5

Describe and interpret her results (100%)

These blood show considerable metabolic derangement with a metabolic and respiratory alkaloses and marked hyponatraemia, hypokalaemia and hypochloraemia (consistent with symptoms) with normal blood sugar.
Likely pathology is external Loss of H+ and all electrolytes (too profound to just be internal redistribution), given age and history most likely diuretic abuse i.e renal loss.
Other possible causes: anorexia / starvation, hidden vomiting (as stem states no GI loss), ion transport kidney diseases (unlikely new presentation at this age), SIADH - drug induced (antidepressants, SSRI).
Hyperadrenalism is not supported by serum sodium.

Marked hyponatraemia
- salt>water losses
- water gains
  - hypervolaemic
    - polydipsia / iatrogenic (not suggested by history)
    - oedema states
  - euvolaemic
    - SIADH, polydipsia
  - hypovolaemic – most likely from history given
    - losses replaced by hypotonic fluid
    - diuretic abuse
    - low dietary salt intake (difficult on Western diet)
- hypertonic losses
  - vomiting (not supported)
  - diarrhoea (not supported)
  - burns (not supported)

Marked hypokalaemia
- inadequate intake
- dietary
- excess excretion
- diuretics
- renal tubular pathology (unlikely demographic)
- kaliuretic drugs
- transcellular shift
- insulin / beta adrenergic use
- alkalosis (present)

Severe hypochloraemia
- usually passive shift to maintain electroneutrality
- chlorine losses usually relate to water losses
- strong association with metabolic alkalosis, diuretic related losses

Acid-base
A – alkalaemia pH 7.6
R – pCO_2 normal suggests metabolic alkalosis
M – high bicarbonate, strongly positive BE supports metabolic alkalosis
A – n/a
D – n/a

A – expected pCO_2 in metabolic alkalosis = 0.7x 40+20 = 48 – lower than expected suggests incomplete compensation / co-existent respiratory alkalosis

metabolic alkalosis
- primary hyperadrenalism (Conn syndrome) – expect high Na
- alkali gain
- milk alkali syndrome
- iatrogenic (e.g. sodium bicarbonate administration) not supported by history
- acid loss
    - GI - vomiting (not supported by history unless concealed), NG suction
    - renal

marked hypokalaemia (evident on bloods)
- carbonic anhydrase inhibitors

Respiratory alkalosis (or poorly compensated metabolic alkalosis)
- hyperventilation
- pain (not supported by history)
- anxiety
- hypoxaemia (pCO_2 lower end of normal but unlikely to give significant added hypoxaemic drive)
- salicylates (no metabolic acidosis)

A-a gradient
- expected pO_2 = 150 – 41×1.25 = 98, measured 80, gap 18
- expected age/4+4 = 15 essentially normal gap

Overall likely considerations include
- diuretic abuse
- anorexia
- concealed vomiting
- milk-alkali syndrome