CASE REPORT

Metabolic alkalosis and myoclonus

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This is the first case reported of vomiting-induced metabolic alkalosis associated with myoclonus. The report describes an unusual presentation of myoclonus secondary to acid-base disturbance caused by recreational drug-induced vomiting. The severe derangement of hyponatraemia, hypokalaemia, and alkalosis appears to have been reasonably well tolerated due to the gradual onset and relatively long history. The causes, mechanism, and management of metabolic alkalosis are discussed.

Recreational drug use is becoming increasingly common among the patient population with as many as 20% of all acute medical admissions identified as abusing such substances.[1] This constitutes a major burden on acute medical services and is responsible for a wide range of clinical presentations.

Prompt and accurate diagnosis is essential in order to treat the patient effectively but can be particularly difficult in patients unable to communicate due to an altered level of consciousness. Toxin and drug ingestion should always be considered in unusual presentations of illness and more than one metabolic derangement can coexist. This case illustrates the necessity of considering a broad differential diagnosis in an unusual presentation.

CASE REPORT

A 29 year old man with a one week history of constipation and vomiting was admitted as an emergency with symptoms of perioral paraesthesia and "muscle twitching".

On questioning he admitted to smoking heroin and cocaine on a daily basis but denied the use of intravenous drugs or having ingested other substances. He had no abdominal pain or focal symptoms and took no other medications.

His past medical history included two similar admissions with vomiting, dehydration, hypokalaemia, and hyponatraemia. Endoscopy performed after the first admission three years previously demonstrated grade 1 oesophagitis and gastroparesis. One year later he was admitted with a first seizure and dehydration with acute renal failure (urea 40 mmol/l, creatinine 320 µmol/l). Metabolic abnormalities this time included hypokalaemia (potassium 1.9 mmol/l), hyponatraemia (sodium 115 mmol/l), and metabolic alkalosis (pH 7.58 kPa, carbon dioxide pressure 8.2 kPa, oxygen pressure 7.9 kPa, bicarbonate 57 mmol/l, base excess 29 mmol/l). All other investigations including viral serology and a computed tomography of his head were normal. After intravenous rehydration with normal saline he quickly improved and was discharged within a few days but failed to attend for subsequent outpatient follow up.

On the current admission, examination revealed that he was dehydrated, uncooperative, and mildly confused with a Glasgow coma scale score of 14. He was afebrile with a respiratory rate of 10 breaths/min and an oxygen saturation of 88% on air with no focal chest signs. Cardiovascular and abdominal examinations were unremarkable. Chvostek's sign was negative. Neurological examination revealed marked myoclonus predominantly of the upper limbs with an approximate five second frequency but no other focal neurological signs or meningism. Results of investigations are listed in tables 1 and 2 and box 1.

Table 1 Biochemistry on admission

Investigation	Patient's results	Normal range
Day 1		
Sodium (mmol/l)	127	133–144
Potassium (mmol/l)	2.1	3.3–5.3
Urea (mmol/l)	28	2.5–6.5
Creatinine (µmol/l)	493	60–210
Chloride (mmol/l)	65	95–105
Phosphate (mmol/l)	1.41	0.8–1.4
Albumin (g/l)	41	35–55
Alanine aminotransferase (IU/l)	8	2–53
Alkaline phosphatase (IU/l)	46	40–130
Bilirubin (mmol/l)	23	3–17
Adjusted calcium (mmol/l)	2.24	2.1–2.6
Magnesium (mmol/l)	0.88	0.75–1.05
Creatine kinase (IU/l)	169	25–200
Glucose (mmol/l)	5.1	3.3–6.0
9 am cortisol (nmol/l)	245	140–690
Serum osmolality (mosmol/l)	286	278–305
C-reactive protein (mg/l)	36	0–10
Day 2		
Sodium (mmol/l)	136	133–144
Potassium (mmol/l)	3.5	3.3–5.3
Urea (mmol/l)	19.9	2.5–6.5
Creatinine (µmol/l)	255	60–210

Table 2 Blood gases

Investigation	Patient's results		Normal range
	On admission	4–6 hours later	
pH	7.58	7.47	7.35–7.45
Oxygen pressure (kPa)	8.25	15.0	11–14
Carbon dioxide pressure (kPa)	6.74	5.05	4.7–6.0
Bicarbonate (mmol/l)	46	50	22–30
Base excess (mmol/l)	21	28	±2

The initial diagnosis was severe dehydration secondary to vomiting (probably induced by opiates and related constipation). The marked metabolic alkalosis was thought to be due to vomiting of hydrogen ions and loss of chloride with compensatory renal bicarbonate retention. The myoclonus was considered to be due to marked alkalosis and reduced ionised serum calcium (possibly accentuated by coexisting uraemia). Hypoxia was consistent with respiratory compensation by hypoventilation with hypercapnia.

Box 1: Other investigations

- Blood cultures: no growth.
- Normal chest radiograph.
- Electrocardiogram: sinus rhythm 80 with prolonged QTc 521.
- Urine toxicology: positive for morphine, cannabis, cocaine, benzodiazepines, and paracetamol.
- Serum paracetamol and salicylates not detected.
- No myoglobinuria.
- Urine trace intact red cells, trace ketones, protein 1 g/l, white cells 15/µl.
- Urine microscopy: no casts, white cells 80/µl, no red blood cells, no organisms.

The degree of alkalosis prompted concern about ingested substances such as caustic soda. This and other agents were denied when the patient was more alert and cooperative.

Treatment was with intravenous normal saline rehydration, careful potassium replacement, and fluid balance monitoring. After infusion of nine litres of fluid the alkalosis and renal impairment improved. The myoclonus resolved and he was discharged approximately 24 hours later with a creatinine of 255 µmol/l and bicarbonate 32 mmol/l.

DISCUSSION

This is the first case report in the literature of vomiting induced metabolic alkalosis causing myoclonus. A case report from Japan describes a case of metabolic alkalosis and myoclonus induced by ingestion of antacid (containing sodium bicarbonate) in a man with pre-existing cerebrovascular disease.[2]

Neuromuscular symptoms of metabolic alkalosis include paraesthesias, muscle twitching, and myoclonus and may be partly due to decreased serum ionised calcium concentrations.[3] Chvostek's sign is usually negative.[4]

Symptoms of muscle twitching and tetany appear to be more common than myoclonus. Tetany is thought to be due to decreased serum ionised calcium concentration[5] and also has been demonstrated to be due to an increase in pH dependent myofibrillar calcium sensitivity.[6]

Hypoxia results from left shift of the oxygen-haemoglobin dissociation curve[3] and compensatory hypoventilation. As in other cases, coexisting factors such as presumed decreased glomerular filtration rate, volume contraction, hypokalaemia, and hypochloraemia help maintain metabolic alkalosis.[7]

Metabolic alkalosis has been the subject of a recent review.[7] The kidney preserves normal acid-base balance by bicarbonate reclamation (mainly in the proximal tubule) and bicarbonate generation (predominantly in the distal nephron). In metabolic alkalosis either gain of base or loss of acid occurs. Acid may be lost via the gastrointestinal tract or kidney. Excess base may be gained by bicarbonate, lactate, acetate, or citrate administration. Clinical states associated with metabolic alkalosis are vomiting, mineralocorticoid excess, the andrenogenital syndrome, liquorice ingestion, diuretic administration, and Bartter's and Gitelman's syndromes.[7]

Learning points

- Recreational drug use is increasing and causes a wide range of clinical pathology.
- Toxin and drug ingestion is a common presentation to the acute medical services and should always be considered in unusual presentations of illness.
- Causes of hypokalaemia and alkalosis include adrenal tumours, ACTH excess, diuretics, laxative abuse, and villous adenoma.
- Combined hypokalaemia with hyponatraemia occurs in very few conditions such as increased antidiuretic hormone with raised ACTH, vomiting, and diuretics.

The combination and degree of metabolic derangement in this case (of hyponatraemia, hypokalaemia, and alkalosis) is unusual and may have been reasonably tolerated as it developed over a number of days. Causes of hypokalaemia and alkalosis include adrenal tumours, adrenocorticotrophic hormone (ACTH) excess, diuretics, laxative abuse, and villous adenoma. Hypokalaemia is caused by the above and in addition by Bartter's, Conn's, and Cushing's syndromes. Combined hypokalaemia with hyponatraemia occurs in very few conditions such as increased antidiuretic hormone with raised ACTH, vomiting, and diuretics.

Treatment is aimed at reversing the underlying cause. The importance of conservative management of carbon dioxide retention has been emphasised in a case of profound alkalosis (caused by gastric outlet obstruction) as hypercapnic respiratory compensation was considered life saving.[8]

Treatment consists of reversing contributory factors and in severe cases administration of carbonic anhydrase inhibitors, acid infusion, and low bicarbonate dialysis.[7]

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