



Severe muscle fasciculations and tremor in a cat with hypochloraemic metabolic alkalosis secondary to duodenal obstruction

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Alison Jukes, Marcus Gunew and Rhett Marshall

Abstract

Case summary An 18-month-old, female spayed, Australian Mist cat presented with a 24 h history of muscle tremors and inappetence progressing to collapse with generalised muscle fasciculations. The cat was diagnosed with a hypochloraemic metabolic alkalosis due to a duodenal foreign body found to be a trichobezoar at coeliotomy. The cat made a complete recovery after enterotomy to remove the trichobezoar, with cessation of neuromuscular clinical signs and normalisation of its electrolyte and acid–base imbalances.

Relevance and novel information Muscle fasciculations and tremors in cats can be caused by intoxications, metabolic derangements, encephalomyelitis, feline hyperaesthesia syndrome and cerebellar diseases. The presenting clinical signs of severe muscle fasciculations and tremors have not previously been reported in association with an intestinal obstruction in the cat.

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Introduction

The most common causes of metabolic alkalosis in dogs and cats are respiratory disease, proximal gastrointestinal (GI) tract obstruction, diuretic administration and renal disease.¹ GI foreign bodies are common in feline practice and often present with vomiting, anorexia, depression and abdominal pain. Clinical signs of metabolic alkalosis usually reflect those of the underlying disorder.²

Important diagnostic possibilities for muscle tremors and fasciculations in cats include intoxications (organophosphates, carbamates, organochlorines, strychnine, pyrethrins and pyrethroids, mycotoxins), metabolic derangements (hypoglycaemia, hypocalcaemia, hypokalaemia, hepatic encephalopathy, uraemia), encephalomyelitis, feline hyperaesthesia syndrome and degenerative and inherited cerebellar diseases.^{3,4} This case report presents a novel clinical presentation of a feline GI obstruction in which the initial clinical signs were severe muscle tremors and fasciculations reminiscent of permethrin intoxication but demonstrated subsequently to be related to

hypochloraemic metabolic alkalosis due to complete proximal intestinal obstruction.

Case description

An 18-month-old, female spayed, Australian Mist cat (5.4 kg) presented to a first-opinion branch practice with a 24 hrs history of lethargy, inappetence, muscle tremors on handling and a single vomit. The cat was housed indoors predominantly with no known exposure to toxins.

On physical examination the cat was quiet but alert with normal thoracic and cardiac auscultation (heart rate 196 beats per min, respiratory rate 36 breaths per min), marked dehydration (approximately 10%) and was tense and painful on cranial abdominal palpation. The cat was

The Cat Clinic, Mt Gravatt, Australia

Corresponding author:

Alison Jukes BVSc, MANZCVS, The Cat Clinic, 189 Creek Road, Mt Gravatt 4122, Australia
 Email: alison.alick@gmail.com



normothermic (rectal temperature 38.4°C). Neurological and musculoskeletal examination revealed mild tetraparesis with equivocally slow proprioception, occasional muscle tremors and normal cranial nerve function.

Methadone (Methadone; Ilium) was administered subcutaneously (1.2 mg; 0.2 mg/kg) for sedation and analgesia and blood for serum biochemistry and haematology was obtained by jugular venepuncture and sent to an external laboratory for urgent analysis. A few minutes later the cat became progressively more lethargic with worsening muscle tremors so the cat was transferred immediately to a referral hospital. Thirty minutes later, on presentation at the referral hospital, the cat was laterally recumbent and non-responsive with generalised muscle fasciculations and limb rigidity, tachycardia and hyperthermia (rectal temperature 40.9°C) (see video in the supplementary material).

Intravenous (IV) fluid therapy was started using compound sodium lactate (Hartmann's solution; Baxter Viaflex) at 10 ml/kg/h and blood was collected for urgent in-house analysis of serum biochemistry and haematology. While awaiting blood test results, the cat was administered diazepam (two doses of 2.5 mg IV, 10 mins apart; Pamlin, Ceva). As IV diazepam did not impact on the cat's signs, intermittent boluses of alfaxalone (Alfaxan; Jurox) totalling 10 mg were administered over 40 mins, with little improvement in muscle fasciculations. Jugular venous blood gas analysis was then performed and methocarbamol (250 mg; Robaxin, Bomac Laboratories) was administered per rectum, which resulted in a

gradual reduction in muscle tremors, fasciculations, and heart rate and body temperature.

Blood test results revealed severe hypochloraemia, hyponatraemia, increased bicarbonate concentration, azotaemia, mild hyperproteinaemia, haemoconcentration and normal total calcium (Table 1). Venous blood gases (performed in-house after 1 h on IV fluids) revealed a mixed acid-base disturbance, with an alkalaemia (pH 7.42; reference interval [RI] 7.24–7.40), metabolic alkalosis (HCO_3 31.2 mmol/l; RI 22.0–24.0 mmol/l) and compensatory respiratory acidosis (PCO_2 53.0 mmHg; RI 34.0–38.0). Base excess and ionised calcium were not measured. At this point the veterinary team were still unclear why these changes were causing the cat's clinical signs, so imaging studies were performed to investigate the hypochloraemic metabolic alkalosis, as proximal GI obstruction seemed a likely possibility.

Thoracic and abdominal radiographs demonstrated gastric dilation. Abdominal ultrasound revealed a dilated stomach filled with fluid containing hyperechoic particulate material and an obstruction in the proximal duodenum. Both kidneys had corticomedullary rim signs. The liver was ultra sonographically normal.

Anaesthesia was induced using diazepam and alfaxalone, and maintained using isoflurane (Isothesia; Henry Schein) delivered via a Bain system. A coeliotomy was performed. Hartmann's solution was administered intraoperatively and during recovery. A trichobezoar was removed from the proximal duodenum via an enterotomy. Recovery was uneventful and the muscle

Table 1 Laboratory measurements at admission and 4 months after discharge.

Analyte (units)	Reference interval	Admission	Follow up 4 months later
RBCS ($\times 10^{12}/l$)	4.9–10.0	10.7	7.8
Haemoglobin (g/l)	77–156	159	119
Sodium (mmol/l)	144–158	143	150
Potassium (mmol/l)	3.7–5.4	4.8	4.7
Chloride (mmol/l)	106–123	74	117
Bicarbonate (mmol/l)	12–24	45	23
Glucose (mmol/l)	3.2–7.5	11.5	3.8
Urea (mmol/l)	5.0–15.0	22.5	8.9
Creatinine (mmol/l)	0.08–0.20	0.29	0.16
Calcium (mmol/l)	2.1–2.8	2.4	2.5
Phosphate (mmol/l)	1.0–2.3	3.8	1.9
Total protein (g/l)	60–84	89	68
Albumin (g/l)	25–38	41	34
ALP (IU/l)	5–50	23	25
AST (IU/l)	2–62	52	116
ALT (IU/l)	19–100	58	134
CK (IU/l)	64–400	512	722
USG	1.035–1.060	Not performed	>1.050

RBCS = red blood cells; ALP = alkaline phosphatase; AST = aspartate transaminase; ALT = alanine transaminase; CK = creatine kinase; USG = urine specific gravity. The most dramatic perturbations are underlined. Abnormal findings are in bold.

fasciculations had ceased completely. Postoperatively, the electrolytes were improved (sodium 149 mmol/l; potassium 3.5 mmol/l; chlorine 96 mmol/l). The cat became fractious to handle subsequently and further blood tests were not able to be performed. The day following surgery the cat's appetite had returned and there were no neuromuscular signs. The cat had an unremarkable recovery and no clinical abnormalities were evident at suture removal 10 days later.

The cat was reviewed 4 months postoperatively. Its owner reported it had been normal since discharge. Physical examination was unremarkable, the cat had gained almost 1.0 kg, and renal analytes, haematology and urine specific gravity were within RIs (Table 1). The cat had developed a mild elevations in aspartate transaminase (116 IU/l [RI 2–62 IU/l]) and alanine transaminase (134 IU/l [RI 19–100 IU/l]) for which the owner declined further investigations. Retesting of liver parameters 7 months postoperatively was unremarkable and the cat remained clinically normal 18 months after surgery.

Discussion

There have been no reported cases in the feline literature of hypochloraemic metabolic alkalosis due to GI obstruction presenting with disorders of involuntary movement such as generalised muscle fasciculation or tremors.

Tremors or fasciculations may be of muscle or neuronal origin.³ Initially, the cause of this cat's muscle tremors and fasciculations were thought to be due to an intoxication, owing to the young age of the cat, the acute onset of the problem and the lack of other specific clinical signs apart from a single vomit. The initial blood tests were sent to an external laboratory and were not available when the clinical signs worsened, so blood was taken urgently for in-house testing and symptomatic treatment for a presumed intoxication with IV diazepam, IV alfaxalone and methocarbamol per rectum was started while awaiting in-house laboratory data. Only the rectal methocarbamol provided symptomatic relief.

Metabolic alkalosis with concurrent high duodenal obstruction led to a decision to perform an urgent coeliotomy, despite us not having a clear understanding of the cause for the neuromuscular signs. Resolution of neuromuscular signs postoperatively in conjunction with marked improvement of electrolyte and acid–base status suggested the clinical signs were due to the electrolyte abnormalities and acid–base derangements.

Metabolic alkalosis can occur owing to loss of chloride-rich fluid (via the GI tract or kidneys) or through excessive administration of alkali.² Most causes of acute metabolic alkalosis occur with a decrease in plasma chloride concentration resulting in an increase in strong ion difference (SID).⁵ Increased SID can also result from decreased water content of plasma (concentration alkalosis).⁶ The kidneys respond to a hypochloraemic metabolic alkalosis by reducing the SID. This is achieved by

increasing chloride reabsorption, and this means producing tubular reabsorbate with a lower than normal SID (allowing some Na⁺ to escape into the urine without accompanying Cl⁻).⁶ If a cat is hypochloraemic and volume depleted (as in the present case), the kidneys will conserve Na⁺ and hypochloraemia is perpetuated.^{2,6}

Resolution of metabolic alkalosis depends on correcting the underlying disorder. The kidneys respond to hypochloraemia by increasing chloride reabsorption (in preference to bicarbonate) and conserving chloride ions until plasma levels are near normal.⁷ In patients with normal renal function that have had correction of the underlying cause of the hypochloraemic metabolic alkalosis, chloride replacement should resolve the alkalosis.^{6,9} Administration of 0.9% NaCl is the fluid of choice for correcting hypovolaemic hypochloraemic metabolic alkalosis,^{6,9} and, in retrospect, would have been a better IV fluid choice for our cat.

Gastric fluid is high in chloride, hydronium ions, sodium and potassium. Hypochloraemic metabolic alkalosis is common in dogs with GI foreign bodies and functional pyloric outflow obstruction.¹⁰ In a recent study examining metabolic alkalosis in dogs and cats, 87% of cats with a metabolic alkalosis were hypochloraemic.¹ In an experimental study of complete obstruction of the ileum in dogs, it was found that bowel proximal to obstruction had net water, sodium and potassium secretion (chloride was not measured), resulting in sequestration of fluid and electrolytes into the lumen of the intestine.¹¹ This mechanism likely played a role in the level of dehydration and hypochloraemia seen in our cat, despite only one witnessed episode of vomiting.

Clinical signs of hypochloraemic metabolic alkalosis in humans include apathy, confusion, seizures, cardiac arrhythmias and neuromuscular irritability, including myoclonus and tetany.^{9,12} Muscle twitching and seizures have also been reported anecdotally in dogs with metabolic alkalosis.² Lowered ionised calcium concentration is thought to be the mechanism that leads to these neuromuscular clinical signs.^{2,12} Hypochloraemic metabolic alkalosis and hypocalcaemia with associated synchronous diaphragmatic flutter is also reported in horses after endurance events.¹³ Alkalosis causes increased negative charges on plasma proteins, which, in turn, increases calcium binding.² The resultant decreased ionised calcium concentration increases the excitability of the muscle cell membrane leading to repetitive firing, muscle tremors and fasciculations (due to spontaneous firing of motor unit potentials).² Experimentally, however, neuromuscular irritability and hypocalcaemia, as seen with metabolic alkalosis, is not completely explained by alkalaemia.^{9,14}

In a study of dogs and cats with metabolic alkalosis, ionised hypocalcaemia was noted in 30% of 319 dogs but only one of 30 cats, although not all patients were

alkalaemic.¹ Hypocalcaemia and muscle fasciculation has been reported in a young cat given therapeutic administration of bicarbonate for salicylate intoxication,¹⁵ while lowered ionised calcium concentrations were reported experimentally after single doses of sodium bicarbonate administered to clinically normal cats.¹⁶ Unfortunately, ionised calcium was not measured in this case, although the total calcium was within the RI.

The cat's azotaemia and hyperphosphataemia were considered to be most likely pre-renal owing to the extent of dehydration and haemoconcentration and likely reduced renal perfusion. Although urinalysis was not performed at the time of crisis, renal analytes and urinalysis were completely normal 4 months later. This cat's acute hyperphosphataemia may also have contributed to suspected low ionised calcium concentration by mass law interactions.¹⁷

Metabolic alkalosis in association with disorders of involuntary movement, including myoclonus, muscle twitching or tetany, has been reported sporadically in human patients. Upper limb myoclonus was reported in one patient with chronic vomiting and hypochloraemia in association with illicit drug use,¹⁸ as well as two other patients in association with the use of dietary supplements containing high levels of sodium bicarbonate or liquorice extract.^{19,20} A further case of metabolic alkalosis with low ionised calcium was reported in a 35-year-old man due to baking soda ingestion, resulting in facial twitching and increased muscle tone in his upper limbs.²¹ Clinical signs resolved in all these patients after withdrawal of the causal agent and appropriate fluid therapy.

Conclusions

This case report demonstrates an unusual clinical presentation of a common feline condition. While an intoxication causing the cat's clinical signs was not definitively excluded and, unfortunately, ionised calcium was not measured, the absence of neuromuscular signs after surgery and rapid normalisation of electrolytes suggested the metabolic derangements secondary to the duodenal foreign body were the underlying cause of the spectacular neuromuscular presentation.

Supplementary material Video 1: Generalised muscle fasciculations in the cat on presentation to referral hospital.

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