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Manual ventilation therapy and aggressive potassium supplementation in the management of respiratory failure secondary to severe hypokalaemia in a cat with exocrine pancreatic insufficiency

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Abstract

A domestic shorthair cat was referred for progressive muscle weakness and dyspnoea. The cat had a 2-month history of severe weight loss, small intestinal diarrhoea, polyphagia and polyuria/polydipsia. Biochemical analysis and venous blood gas evaluation revealed severe hypokalaemia [1.7 mmol/l; reference interval (RI): 3.5–5.1 mmol/l] and hypoventilation (partial pressure of carbon dioxide = 68 mmHg; RI: 34–38 mmHg). Aggressive potassium supplementation was initiated. The cat was manually ventilated until serum potassium increased to 3 mmol/l. A diagnosis of exocrine pancreatic insufficiency (EPI) was made based on clinical signs and serum feline trypsin-like immunoreactivity (0.1 µg/l; RI: 12–82 µg/l). Medical management of the EPI resulted in clinical recovery.

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A 5-year old, spayed female domestic shorthair cat was referred for progressive muscle weakness, dyspnoea and a 24-h history of anorexia. The cat had a 2-month history of weight loss, persistent small intestinal diarrhoea with soft-to-liquid stools, polyphagia, and polyuria (PU)/polydipsia (PD). The cat was fed a standard commercial diet. A recent test (Snap test FIV/FeLV; Idexx) for feline leukaemia virus and feline immunodeficiency virus was negative. The owner did not report any previous illness. The cat was not vaccinated but had been treated for internal parasite (milbemycine/praziquantel) by the referring veterinarian 2 weeks before presentation.

On presentation, the cat was depressed and laterally recumbent. Its bodyweight was 2.2 kg (body condition score 1/9). She was bradycardic [100 beats per minute (bpm)], dyspnoeic, tachypnoeic (40 respirations per minute (rpm)) and hypothermic (36.4°C; reference interval: 38.1–39.2°C). The cat appeared significantly dehydrated. Generalised muscle weakness and cervical ventroflexion were noted. Cranial nerve examination and spinal reflexes were unremarkable. Systolic, diastolic and mean arterial blood pressure measured at the right forelimb using an oscillometric method (VetCare B Braun Smith Medical PM) were 137, 98 and 111 mmHg, respectively. An electrocardiogram showed sinus bradycardia. Pulse oximetry (veterinary pulse oxymeter VE-H100B; Kontron Medical) could not be assessed because the cat did not tolerate proper placement of the pulse oximetry probe.

A complete blood cell count (Table 1) revealed mild eosinopenia. Biochemical analysis (Table 1) showed severe hypokalaemia [1.7 mmol/l; reference interval (RI): 3.5–5.1 mmol/l], hyperglycaemia (15.6 mmol/l; RI: 4.2–11.0 mmol/l) and a mildly increased alanine transaminase (127 U/l; RI: 20–107 U/l). Total magnesium and total serum thyroxin concentrations were within normal limits (Table 1). Venous blood gas analysis (Vetstat; Idexx)

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Table 1 Results of the complete blood cell count, biochemical analysis, venous blood gas analysis, urinalysis, coagulation profile and intestinal function tests from a cat with hypercapnoeic respiratory failure and exocrine pancreatic insufficiency. Abnormal values are in bold

Variable	Results	Reference interval
Complete blood cell count:		
Haemoglobin	10.3 g/dl	9.8–16.9 g/dl
HCT	29.1%	29–48%
MCHC	35.4 g/dl	32.9–39.1 g/dl
MCV	42.3 fl	33.6–48.3 fl
White blood cells	10.56 × 10^3/µl	3.70–18.66 × 10^3/µl
Neutrophils	7.68 ×10^3/µl	1.45–9.62 × 10^3/µl
Lymphocytes	2.28 × 10^3/µl	1.18–10.36 × 10^3/µl
Monocytes	0.52 ×10^3/µl	0.09–0.82 × 10^3/µl
Eosinophils	**0.08 × 10^3/µl**	0.16–1.81 × 10^3/µl
Platelets	400 × 10^3/µl	175–500 × 10^3/µl
Biochemical analysis:		
Creatinine	**55 µmol/l**	80–229 µmol/l
Glucose	**15.6 mmol/l**	4.2–11.0 mmol/l
Total protein	64 g/l	55–71 g/l
Albumin	35 g/l	27–39 g/l
ALT	**127 U/l**	20–107 U/l
ALP	42 U/l	23–107 U/l
Ionised calcium	1.31 mmol/l	1.1–1.4 mmol/l
Total magnesium	21 mg/l	15–30 mg/l
Sodium	154 mmol/l	148–157 mmol/l
Potassium	**1.7 mmol/l**	3.5–5.1 mmol/l
T4	26.5 nmol/l	15–55 nmol/l
Venous blood gas analysis:		
pH	**7.13**	7.24–7.40
PCO_2	**68 mmHg**	34–38 mmHg
HCO_3^-	**20.7 mmol/l**	22–24 mmol/l
Anion gap	23 mEq/l	17–31 mEq/l
Urinalysis:		
Specific gravity	1.030	1.030–1.060
pH	**5**	5.5–8.0
Proteins	**+**	–
Glucose	**++++**	–
Fractional excretion of potassium	4%	⩽24%
Coagulation profile:		
Prothrombin time	10.7 s	9.8–12.4 s
Activated partial thromboplastin time	15.4 s	12.8–20.3 s
Exocrine pancreatic and intestinal function tests:		
Serum fTLI	**<0.1 µg/l**	12–82 µg/l
Folate	13.8 ng/ml	10–25 ng/ml
Cobalamin	**<150 ng/l**	300–1500 ng/l

HCT = haematocrit; MCHC = mean corpuscular haemoglobin concentration; MCV = mean corpuscular volume; ALT = alanine transaminase; ALP = alkaline phosphatase; pCO_2 = partial pressure of carbon dioxide; HCO_3^- = bicarbonate ion; fTLI = feline trypsin-like immunoreactivity

(Table 1) revealed a mixed acid-base abnormality [pH = 7.13, RI: 7.24–7.40; partial pressure of carbon dioxide (PCO_2) = 68 mmHg, RI: 34–38 mmHg; bicarbonate ion (HCO_3^-) = 20.7 mmol/l, RI: 22–24 mmol/l; anion gap 23 mEq/l, RI: 17–31 mEq/l]. Urine analysis showed significant glucosuria (Table 1). Fractional excretion (FE) of

Table 2 Frequency of blood potassium measurements and progression of blood potassium values

Time post-admission (h)	Blood potassium level (mmol/l)
0	1.7
3	2.3
6	2.9
7	3.0
10	3.0
18	3.1
22	3.7
29	3.5
34	3.8

potassium (Table 1) was within normal limits (4%; RI: ⩽24%).[1] Thoracic radiographs and coagulation profile (Table 1) were unremarkable.

Initial therapy consisted of fluid therapy (sodium chloride 0.9%; B Braun Medical) and oxygen therapy via nasal flow-by. The venous hypercapnia was attributed to respiratory muscle weakness and hypoventilation caused by the hypokalaemia. The cat was sedated with diazepam (0.2 mg/kg IV; Valium Roche, Roche) and butorphanol (0.2 mg/kg IV; Dolorex, Intervet), and intubated after induction with alfaxalone (2 mg/kg IV; Alfaxan, Vetoquinol). Manual ventilation with 100% oxygen supplementation was performed using a Bain non-rebreathing circuit. Pressure was controlled manually as the circuit was not equipped with a manometer. Anaesthesia was maintained with a continuous rate infusion of alfaxalone (40 µg/kg/min). The ventilation rate was adjusted to maintain end-tidal CO_2 (ET-CO_2) between 35 and 45 mmHg. Venous blood gases were evaluated several times to assess $PvCO_2$ and identify any discrepancies between the $PvCO_2$ and the ET-CO_2. Inspired oxygen concentration was gradually decreased to 21% while maintaining oxygen saturation (SpO_2) >92%. Blood potassium was initially supplemented with 10% potassium chloride (Proamp; Aguettant) intravenously at a rate of 1 mEq/kg/h. After approximately 6 h blood potassium reached 2.9 mmol/l and potassium supplementation was changed according to a standard protocol described in the literature.[1] Blood potassium values remained within the normal reference interval from 22 h until discharge. The frequency of potassium measurements and the progression of blood potassium values are summarised in Table 2. After about 6.5 h of manual ventilation the cat was able to maintain its ET-CO_2 below 40 mmHg while breathing spontaneously. Manual ventilation was discontinued and the cat was extubated.

Causes for the severe hypokalaemia were explored. Abdominal ultrasonography revealed a diffusely thickened small intestinal muscularis layer. Pancreas and adrenal glands were unremarkable. Faecal flotation for intestinal parasites was negative. Serum was sent for feline trypsin-like immunoreactivity (fTLI), folate and cobalamin determination (Idexx Alfort). The cat was discharged 7 days after initial presentation with cobalamin supplementation (250 µg SC once a week for 6 weeks; Vitamin B_{12}, Laboratoire Gerda), fenbendazole (50 mg/kg PO q24h for 5 days; Panacur 250 Chien, Intervet), a highly digestible diet (prescription diet feline i/d; Hills) and potassium supplementation (2 mEq PO q12h for 12 days; potassium gluconate H3 Santé, Sirop). At recheck, 12 days after oral potassium supplementation was initiated, blood potassium was 6.6 mmol/l. At this point oral potassium supplementation was discontinued. Endoscopic evaluation of the gastrointestinal tract was performed 4 weeks after initial presentation, pending the results of serum fTLI, folate and cobalamin because of the persistence of clinical signs and to evaluate for chronic intestinal disease. Blood potassium was 3.7 mmol/l at the time of endoscopy. Endoscopy did not reveal any macroscopic changes in the stomach, the duodenum and the colon. Histological examination of biopsy samples revealed mild-to-moderate chronic lymphoplasmacytic infiltration in duodenum and colon, and mild lymphoplasmacytic infiltration and *Helicobacter*-like organisms in the stomach. Serum fTLI was markedly decreased (<0.1 µg/l; RI: 12–82 µg/l). Serum folate was within normal limits (13.8 ng/ml; RI: 10–25 ng/ml) and serum cobalamin was severely decreased (<150 ng/l; RI: 300–1500 ng/l) (Table 1). A diagnosis of exocrine pancreatic insufficiency (EPI) was made. Supplementation with pancreas extract Eurobiol granules (containing 152.25 mg of porcine pancreas extract / dose), Mayoly-Spindler, Chatou, France, mixed with the same highly digestible canned food (prescription diet feline i/d; Hills) q8h long term] was initiated. Serum cobalamin (>1000 ng/ml) and serum folate concentrations were within the normal reference interval 6 weeks after initial discharge; the cat's body weight had increased to 2.5 kg and it was clinically normal. The cat did well during the first 4 months of treatment and was lost to follow-up thereafter. After the first 4 months the owner was unable to give the prescribed medications regularly. The cat died after several months of sporadic administration of the medications, and exhibited similar clinical signs as presented on initial evaluation, including muscle weakness and dyspnoea.

To our knowledge, this is the first report of the successful use of manual ventilation therapy in combination with aggressive potassium supplementation in the management of hypercapnoeic respiratory failure secondary to severe hypokalaemia in a cat with EPI.

Muscle weakness, PU/PD and impaired urinary concentration ability are the most common clinical signs in dogs and cats with symptomatic hypokalaemia.[1] Muscle weakness is a result of hypokalaemia-induced changes in neuromuscular excitability and typically does not become apparent until the plasma potassium

concentration is <2.5 mmol/l.[2] Respiratory muscle paralysis secondary to severe hypokalaemia has been reported in humans and cats.[3–8] Mechanical ventilation in combination with aggressive potassium supplementation is the treatment of choice for hypokalaemia-induced hypercapnoeic respiratory failure in cats.[6–8] In previous reports, initial serum potassium concentration ranged from 1.5 to 1.8 mmol/l, and the rate of potassium supplementation ranged from 0.22 mEq/kg/h to 1 mEq/kg/h.[5,6,8] Mechanical ventilation was discontinued after serum potassium concentration had increased to 2.2 mmol/l and 2.86 mmol/l, respectively.[6,8] Ventilation times ranged from 4 h to 2 days.[5,6,8] In the patient presented here, manual ventilation was discontinued 6.5 h after initiation of ventilation therapy, which is comparable to ventilation times reported previously.[6,8]

Hypokalaemia can result from decreased potassium intake, translocation from the extracellular fluid compartment to the intracellular fluid compartment, and excessive gastrointestinal or renal losses.[1] Ninety-five percent of total body potassium is located within the cells and 60–75% of intracellular potassium is within muscle cells.[1] Loss of muscle mass leads to a decrease in total body potassium.[9] The cat presented here had a markedly decreased muscle mass, which could have led to a significant decrease in total body potassium and predisposed it to the development of hypokalaemia due to additional potassium losses. Decreased dietary intake is rarely the main cause for hypokalaemia and is unlikely to be a significant factor in this case as the cat ate a normal commercial diet.[10] Translocation of potassium from the extracellular to the intracellular fluid compartment may occur secondary to alkalaemia, insulin release and the administration of beta-blockers or alpha-agonists.[1,10] None of these conditions were identified in the case reported here. Therefore, the hypokalaemia observed in the cat presented here was probably due to increased losses. The gastrointestinal tract seemed the most likely source of potassium losses. Renal potassium losses were considered unlikely based on a normal FE of potassium.[1,10]

There are no published data identifying the frequency of hypokalaemia in dogs and cats with EPI. In most cats with EPI results of routine blood tests are either within the normal range or may be related to concurrent conditions.[11,12] The proximal small intestine is the main site of dietary potassium absorption in humans.[9] The potassium concentration in gastric and intestinal secretions is higher than that of plasma.[13] In humans, normal stool water contains 83–95 mEq/l of potassium.[9] The amount of potassium lost in the stool is one factor that influences the development of hypokalaemia in humans.[9] In patients with diarrhoea, total body potassium may be decreased owing to a loss of muscle mass secondary to malnutrition and/or reduced net absorption of potassium.[9] A negative potassium balance is primarily caused by excessive potassium losses in the stool and influenced secondarily by the amount of potassium in the diet and the degree of renal potassium conservation.[9] Gastrointestinal potassium losses through vomiting and diarrhoea have been described as a cause for hypokalaemia in humans and small animals.[1,9,13–17] Specific causes for hypokalaemia secondary to chronic diarrhoea, such as inflammatory bowel disease, pancreatic insufficiency, carbohydrate malabsorption and sigmoid volvulus, have been identified in humans.[9,14] There is a lack of scientific reports on the significance of potassium loss due to diarrhoea in dogs and cats. In the case presented here, hypokalaemia developed most likely as a result of a combination of decreased total body potassium secondary to a loss of muscle mass from malassimilation and increased intestinal potassium losses, and possibly decreased potassium absorption due to chronic small intestinal diarrhoea/dysfunction.

The patient presented here had a mixed acid–base abnormality. Hypokalaemia-induced hypoventilation caused a severe respiratory acidosis. Small intestine diarrhoea may have resulted in intestinal loss of HCO_3^- and metabolic acidosis. Lactic acidosis could have contributed to the metabolic acidosis; however, serum lactate was not evaluated.

Conclusions

To our knowledge, this is the first report of the successful use of manual ventilation therapy in the management of acute hypercapnoeic respiratory failure due to severe hypokalaemia in a cat with EPI. Hypokalaemia should be considered as a cause for hypercapnoeic respiratory failure. Aggressive intravenous potassium supplementation at a rate of 1 mEq/kg/h was well tolerated and effective in rapidly increasing the blood potassium concentration. As shown in this report, the duration of ventilatory support is typically short in cats with acute respiratory failure due to severe hypokalaemia, and manual ventilation may be an option if mechanical ventilation is not available.

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