Dealing with hypernatraemia in cats and dogs: a case-based discussion

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Harriet Thomas takes a detailed look at the treatment of three of the canine and feline cases presented with high sodium levels at The Blue Cross

DURING the heavy snow fall of 2010/2011, The Blue Cross animal hospital in Victoria, London, admitted several cats whose symptoms were consistent with salt toxicity – possibly because of the use of salt to treat icy roads.

Sodium is primarily an extracellular ion and is the most important determinant of extracellular fluid (ECF) volume. The body is able to sense and respond to very small changes in sodium content to maintain effective circulating volume. Blood sodium levels reflect the ratio of sodium to water in the extracellular fluid.

Volume receptors (lowpressure mechanoreceptors) are located in the cardiac atria and pulmonary vessels, and pressure receptors (highpressure baroreceptors) are located in the aortic arch and carotid sinus. The kidney is the site of integration of the endocrine mechanisms for volume regulation. The juxtaglomerular apparatus responds to changes in perfusion pressure with changes in production and release of renin. Activation of the renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system (SNS) and atrial natiuretic peptide (ANP) are also important effector mechanisms. RAAS and the SNS result in increased sodium retention by the kidneys. ANP release results in decreased sodium retention. There may also be receptors located in the liver and central nervous system (CNS) that contribute to volume regulation via sodium homeostasis.

Regulation of water content (osmoregulation) will also affect serum sodium concentration. The principal mechanism of osmoregulation involves the hypothalamus. Release of antidiuretic hormone (ADH) by the hypothalamus stimulates thirst and increases water reabsorption from the collecting duct in the distal nephron[1].

Hypernatraemia as detected on serum sodium evaluation reflects an increase in sodium relative to water content. This may be a result of:

• sodium gain (salt toxicity, hypertonic fluid administration, hyperaldosteronism);

• loss of pure water (primary hypodipsia, diabetes insipidus, hyperthermia); and

• loss of hypotonic fluid (vomiting, diarrhoea, acute or chronic renal failure, burn injuries or iatrogenic – resulting from furosemide administration).

Loss of pure water from the ECF results in it being hypertonic relative to the intracellular volume. This results in a shift of fluid from the relatively large intracellular fluid (ICF) compartment to the ECF compartment. Thus, fluid losses are shared between the ICF and the ECF, with the ICF losing two-thirds of the volume and the ECF losing one-third due to their relative sizes. Since the circulating plasma volume is one-quarter of the ECF, plasma volume decreases by only one-and-a-half of the total volume of pure water loss. For this reason, the animal with pure water loss appears relatively normovolaemic.

In contrast, when hypotonic fluid is lost from the ECF, there is less stimulus for osmotic movement of water from the ICF to the ECF. Thus, hypotonic fluid losses cause a greater reduction in ECF volume than pure water losses and patients present with clinical signs of volume depletion. Hypotonic fluid losses are the most common type encountered in small animal medicine.

When evaluating hypernatraemia, it is vitally important to assess a patient’s volume status on physical examination (heart rate, pulse quality, mucous membrane colour, capillary refill time) to compose a differential diagnoses list and instigate effective treatment.

The following is an example of a differential diagnoses list:

• Sodium gain (salt toxicity, hypertonic saline, hyperaldosteronism) = hypervolaemia.

• Pure water loss (hypodipsia, diabetes insipidus, hyperthermia) = normovolaemia.

• Hypotonic fluid loss (renal failure, GI loss, burns, furosemide) = hypovolaemia.

Mild to moderate hypernatraemia usually causes minimal clinical signs. However, marked hypernatraemia may induce cerebral signs, such as depression, weakness, irritability, confusion,

circling, seizures, coma, and death in dogs and cats as a result of cellular dehydration of neurons[2].

Additionally, in humans and in experimental animals, brain tissue shrinkage may cause vessels to tear, leading to intracranial haemorrhage, infarction, venous thrombi, and cerebral oedema[3].

The following three cases represent the different causes of hypernatraemia.

Case one: solute gain

Messy, an 11-year-old male, neutered, domestic shorthaired cat, presented one morning with ataxia having failed to return home the previous night. He had no significant medical history.

On physical examination, his heart rate was 160bpm with moderately tall pulses, pink mucous membranes and a brisk capillary refill time (around one second). Respiratory rate and effort were normal and thoracic auscultation was unremarkable.

Mentation was severely obtunded. He was nonambulatory with horizontal nystagmus (fast phase to the right) that changed to vertical with changes in head position. Conscious proprioception was delayed on the left hind, although spinal reflexes were normal. He had intermittent generalised muscle tremors. The corneal reflex was reduced on the right, although menace and pupillary light reflexes were intact. The neurological examination was interpreted as multifocal CNS disease. Abdominal palpation was unremarkable. Rectal temperature was mildly increased at 39.1°C.

• Initial diagnostic database (reference range in brackets):

– Packed cell volume 45 per cent (24 per cent to 45 per cent).

– Refractometric total protein 76g/L (60g/L to 80g/L).

– Sodium 188mmol/L (145mmol/L to 157mmol/L).

– Potassium 3.4mmol/L (3.6mmol/L to 5.5mmol/L).

– pH 7.351 (7.25 to 7.40).

– Base excess -17 (-5 to +2).

– PCO_2 37.5mmHg (28mmHg to 34mmHg).

– Creatinine 166µmol/L (40µmol/L to 150µmol/L).

– Urea 13.6mmol/L (5mmol/L to 11mmol/L)

– Glucose 11mmol/L (3mmol/L to 3.6mmol/L).

Haematology showed a neutrophilia (21×10^9) with a lymphopaenia (0.9×10^9). This was interpreted as a stress leucogram.

The cat was diagnosed with solute gain hypernatraemia. There was no history of hypertonic saline administration and the blood results were not consistent with hyperaldosteronism (normokalaemia). On questioning the owner more closely this episode occurred during the snowy weather and the owners had sprinkled table salt on the path. It was deemed possible that salt ingestion with concurrent lack of water intake had resulted in hypernatraemia.

The mainstay of treatment is IV fluid therapy. When the serum sodium abnormality has been gradual in onset, it should be corrected slowly with a maximum change of 0.5mEq/L/hour. Because the serum sodium was 188mmol/L, the aim was to reduce it to 155mmol/L – a change of 33mmol/L over 66 hours. However, from the history, it was likely the hypernatraemia had developed much more acutely than at a rate of 0.5mEq/hour. For animals with acute onset symptomatic hypernatraemia, it is reported that a decline in serum sodium around 1.0mEq/L/hour can be safely performed[4].

The cat was treated with 0.9 per cent NaCl. This was chosen since it has a sodium content of 154mmol/L – more sodium than Hartmann's (130mmol/L) – so it would reduce the serum sodium more slowly.

A urinary catheter was placed to monitor hourly urine output so fluid administration could be titrated against urine output. The cat was hypervolaemic initially, so fluid overload was a concern.

After an hour the cat's mentation worsened and it started to seizure. Repeated biochemistry showed the serum sodium had increased to 194mmol/L. A more aggressive tactic was adopted to try to lower serum sodium – 0.45 per cent NaCl was administered, which has a sodium content of 77mmol/L. Furosemide administration was considered as this causes sodium diuresis by acting on the ascending limb of the loop of Henle. Seizures were controlled by administration of IV diazepam.

Gastric lavage was also considered, but abdominal radiography revealed no gastric contents and, due to time since ingestion, it was likely that gastric emptying was complete.

The patient continued to seizure despite therapy and was euthanised on welfare grounds.

Case two: pure water deficit

Loco, an 18kg, three-yearold male entire Staffordshire bull terrier presented with a three-day history of weakness and collapse.

On examination, his cardiovascular and respiratory systems were normal. Mentation was obtunded, but responsive. He was poorly ambulatory with a marked ataxia. Neurological examination revealed no spinal pain, but conscious proprioception was delayed on all four limbs. Spinal reflexes were normal. Cranial nerve reflexes were intact. Abdominal palpation was unremarkable. Rectal temperature was increased to 39.9°C.

• Initial diagnostic database:

– Packed cell volume 48 per cent (37 per cent to 55 per cent).

– Refractometric total protein 66g/L (55g/L to 80g/L).

– Sodium 215mmol/L (140mmol/L to 155mmol/L).

– Potassium 3.9mmol/L (3.6mmol/L to 5.8mmol/L).

– pH 7.39 (7.35 to 7.45).

– Base excess -15 (-5 to 0).

– PCO_2 17 (34 to 40).

– Lactate 3.17mmol/L (0.6mmol/L to 2.9mmol/L).

– Urea 17.1mmol/L (2.5mmol/L to 7mmol/L).

– Creatinine 171µmol/L (40µmol/L to 150ìmol/L).

– Glucose 6.6mmol/L (3.3mmol/L to 6mmol/L).

The dog was diagnosed as having pure water deficit hypernatraemia. Differential diagnoses included diabetes insipidus, inadequate access to water, hyperthermia and primary hypodipsia.

Given the extreme hypernatraemia, but milder clinical signs than in case one, it was presumed the increase in serum sodium had been more gradual. On closer questioning, the owner revealed that, although offered, the dog had never drunk water. He ate as normal and had no neurological problems associated with prehension of food or swallowing. The owner also noticed the dog always became depressed and lethargic when fed dry food compared with normal mentation when fed tinned dog food.

Because of the chronic nature of the hypernatraemia, care was taken to reduce serum sodium levels slowly at no greater than 0.5mEq/L/hour. The aim was to reduce the serum sodium to a level

that resulted in no clinical signs associated with hypernatraemia, but not to reduce the sodium to the reference range as it is unlikely to have been, or be able to be, maintained at this level in this particular case.

• The free water deficit was estimated as:

– 0.6 × weight (kg) × (serum Na patient/normal serum Na) - 1.

– 0.6 × 18kg × (215mmol/L serum sodium patient/ 150mmol/L normal serum sodium) - 1 = 4.68L.

This is the volume of free water required to replace the deficit and correct the serum sodium level to normal. Giving this volume as five per cent dextrose in water would cause too rapid a decrease in serum sodium. This would result in brain oedema since brain cells subjected to chronic hypernatraemia generate intracellular idiogenic osmoles to protect the tissue from dehydration caused by osmotic movement of water out of the cells in response to the circulating hypernatraemia. If the hypernatraemia is reduced rapidly with fluid therapy the idiogenic osmoles will precipitate brain oedema by attracting water into cells down a concentration gradient[4].

An initial fluid of 0.9 per cent NaCl was selected. Given the dog was normovolaemic, a cautious fluid rate of 4ml/kg/ hour was used.

A urinary catheter was placed to monitor “ins and outs” and serum sodium was monitored every two to four hours to track sodium reduction and ensure a gradual decline. Systolic blood pressure, mentation and fundic examinations were recorded to monitor any evidence of increases in intracranial pressure as a result of brain oedema development.

Over the following 17 hours, serum sodium decreased to 194mmol/L – a reduction of 16mmol/L. The fluid rate was decreased to 2ml/kg/hr to slow the rate of decrease in sodium. The serum sodium continued to decrease gradually and mentation improved. Sodium decreased to 180mmol/L after a further 24 hours, which corresponded to resolution of neurological signs.

It is recommended that chronic hypernatraemia cases are managed by the oral administration of fluids to gradually correct the hypernatraemia. Due to primary hypodipsia, it was not possible in this case.

Recommendations made to the owner for chronic management included adding water to tinned food, avoiding dry food and the provision of extra water on food in hot weather and during exercise.

Case three: hypotonic fluid loss

James, an 18-year-old male neutered domestic shorthaired cat presented with a chronic history of polyuria and polydipsia over the previous few months and a three-day history of anorexia and

vomiting.

On physical examination he was tachycardic (200bpm) with mucous membrane pallor, prolonged capillary refill time of around three seconds and weak, thready pulses. He had a prolonged skin tent and tacky mucous membranes and was estimated to be seven per cent to 10 per cent dehydrated. Mentation was dull and depressed, but responsive. Abdominal palpation revealed small, firm non-painful kidneys. Rectal temperature was slightly reduced (36.9°C) and he was in poor body condition (score one out of five).

• Initial diagnostic database:

– Packed cell volume 28 per cent (24 per cent to 45 per cent).

– Refractometric total protein

– 86g/L (60g/L to 80g/L).

– Sodium 181mmol/L (145mmol/L to 157mmol/L).

– Potassium 5.1mmol/L (3.6mmol/L to 5.5mmol/L).

– Urea 68mmol/L (5mmol/L to 11mmol/L).

– Creatinine 332µmol/L (40µmol/L to 150µmol/L).

– Phosphate 4.42mmol/L (1.15mmol/L to 1.77mmol/L).

– Urine specific gravity 1.014, no active sediment on exam.

He was diagnosed as having hypernatraemia caused by hypotonic fluid loss. Common causes of this are:

• renal failure (both acute and chronic);

• vomiting;

• diarrhoea (although gastrointestinal losses can result in hyponatraemia, depending on whether the fluid lost contains more sodium than water or vice versa);

• burn injuries; or

• iatrogenic causes, such as frusemide or mannitol administration.

The elevation in serum sodium was likely to be chronic, due to lack of specific clinical signs relating to hypernatraemia. Suspected chronic renal failure was the presumptive diagnosis.

As described earlier, losses of hypotonic fluid resulting in hypernatraemia cause more severe extracellular volume contraction than losses of pure water, since the loss is borne mostly by the extracellular space with little movement of water from the intracellular space as the fluid loss becomes closer to isotonic. Thus, clinical signs of hypovolaemia are present.

The initial replacement fluid should be isotonic so extracellular volume replacement proceeds rapidly. A volume of up to four times the suspected intravascular deficit may be required because the isotonic crystalloid solution distributes rapidly throughout the extracellular fluid compartment and extracellular fluid volume is three times the intravascular volume.

The cat was given a fluid bolus of Hartmann’s fluid at 15ml/kg over 20 minutes. This resulted in an improvement in pulse quality and his heart rate decreased to 160bpm. The cat was also offered water per os to allow gradual replacement of the fluid deficit. Once the volume deficit was corrected, gradual replacement of the dehydration deficit in addition to a maintenance fluid requirement to cover ongoing losses was administered. This results in a fluid rate of 8ml/kg/hour for the subsequent 24 hours.

No specific fluid plan was instigated to reduce the hyponatraemia since it was not causing clinical signs. In most cases of hypernatraemia the serum sodium level does not need specific treatment, but will correct as the underlying disease is treated.

With fluid therapy, the azotaemia improved to a creatinine of 210ìmol/L and a urea of 19.1mmol/L. A nonregenerative anaemia of 21 per cent emerged once the fluid deficit was replaced, which was presumed due to the chronic renal disease. The cat was hospitalised for two days and discharged with a guarded longterm prognosis.

Discussion

• Case one.

The clinical signs on presentation were attributable to hypernatraemia as a result of osmotic movement of water out of the brain cells. A rapid decrease in brain volume can cause rupture of the cerebral vessels and focal haemorrhage.

The severity of clinical signs is related to the speed of hypernatraemia onset rather than the specific level of serum sodium. However, clinical signs are usually only apparent above a serum sodium level of 170mEq/L in cats and dogs.

If hypernatraemia develops slowly the brain has time to adapt by producing idiogenic osmoles,

which are intracellular solutes that act to retain water inside cells.

• Case two.

Hypodipsia has been identified as abnormal osmoregulation of ADH release, usually caused by underlying hypothalamic lesions[4]. A case report exists of a hypodipsic dog with abnormal osmoreceptor function[5]. It has also been described in a young Dalmatian with dysplasia of the rostral diencephalon[2].

Dogs with congenital adipsic hypernatraemia may have a spectrum of neuroanatomic abnormalities. An example of spontaneous remission of primary adipsia after two years was recorded in a Norwegian elkhound[6]. Several other case reports of primary adipsia exist[7] [8] [9].

• Case three.

The cat was diagnosed as having hypernatraemia caused by hypotonic fluid loss. Common causes of this are renal failure (both acute and chronic), vomiting, diarrhoea (although gastrointestinal losses can result in hyponatraemia, depending on whether the fluid lost contains more sodium than water or vice versa), burn injuries or iatrogenic causes, such as frusemide or mannitol administration.

Summary

The three cases highlight the importance of sodium measurement in practice as well as careful physical examination to allow categorisation of hypernatraemia and, thus, construction of a differential diagnoses list.

Emphasis must be placed on serial sodium monitoring when attempting to correct serum sodium levels as rapid resolution can result in more severe and irreversible clinical signs than the initial hypernatraemia.

The Blue Cross

The Blue Cross is one of the UK’s leading animal charities, providing practical support, information and advice for pet and horse owners. Its hospitals provide veterinary care for the pets of people who cannot afford private vets’ fees. Through its network of small animal and equine adoption centres, it rehomes thousands of animals each year.

The Blue Cross also actively campaigns to improve all aspects of pet and equine welfare. The charity works with the Government, schools and other organisations to create a greater understanding of responsible animal ownership and improve the lives of animals and their owners. For more information, visit www. bluecross.org.uk.

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