

Kathryn Jenkins of Gribbles Veterinary, Palmerston North, discusses an unusual case involving a cat, salt and a household lamp.

A RECENT SUBMISSION highlighted an unusual case of salt toxicity in a cat.

CLINICAL HISTORY: A one-year-old cat presented with recent neurologic signs (staring into space, head tilt, wide stance, not able to eat well and falling into the food bowl). In-house blood glucose was unremarkable.

LABORATORY RESULTS: Serum biochemistry and a complete blood count were performed. The most striking changes were seen in the electrolytes, with severe hypernatremia 207mmol/L (reference interval 147-160mmol/L) and hyperchloridemia 170mmol/L (reference interval 113-127mmol/L). Urea was mildly elevated and creatinine was within normal limits. PCV was 44% (reference interval 24–45%), providing support for mild dehydration, although urine-specific gravity was unavailable. In addition, given the cat's young age and a history of chronic ill-health, it was noted that creatinine may have been decreased due to low muscle mass.

DISCUSSION: Differential diagnoses for marked hypernatremia in cats include neurologic disease (eg, central diabetes insipidus – secondary to head trauma, congenital defects or mass lesions), restricted access to water (eg, cat inadvertently locked in a garage), free water loss in excess of sodium (eg, severe gastrointestinal disease, diabetic ketoacidosis, chronic kidney disease or non-oliguric acute kidney injury) and salt toxicosis.

On further discussion, the owners said that the cat had been seen licking a Himalayan salt lamp, which is a rare but reported cause of salt toxicity in cats. The young cat was rehydrated and neurologic signs abated, helping to support acute salt toxicity in this case.

Salt toxicity in companion animals is an uncommon but potentially lifethreatening condition. It is reported more often in dogs than in cats, most likely due to the latter's more fastidious tastes. In general, animals can tolerate a high level of salt/sodium in the diet provided they have continuous access to fresh water.

Reported causes of salt toxicity in dogs include ingestion of ocean water, paint balls, homemade playdough and homemade emetic solutions. In addition, rock salt is used in some countries (eg, North America) to help de-ice wintry roads, and pets may consume excessive salt via grooming in these conditions. Of interest, paint balls don't actually have any salt; rather the sorbitol/polyethylene glycol/glycerol they contain are osmotically active agents, resulting in marked fluid shifts across the intestinal mucosa.

Common clinical signs of salt toxicity in both dogs and cats occur within several hours of ingestion and include anorexia, lethargy, vomiting, muscle weakness, behavioural changes, disorientation, ataxia, seizures and, in some cases, coma and death.

DANGER VALUES: Clinical signs of hypernatremia are more related to the rapidity of onset rather than the magnitude of change. However, neurologic signs may occur with sodium concentrations greater than 170mmol/L in dogs and 175mmol/L in cats.

The neurologic signs in cases of hypernatremia may be primary or secondary (ie, intracranial disease can cause hypernatremia, and hypernatremia can cause neurologic disease). Of clinical importance, acute hypernatremia cases are at risk of CNS haemorrhage/thrombosis and chronic cases require slow fluid resuscitation to avoid cerebral oedema. (9)

REFERENCES:

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