

Thiamine deficiency in dogs and cats

Clinical thiamine deficiency occurs sporadically in dogs and cats in New Zealand and usually manifests as an acute neurological syndrome, leading to death if untreated. The deficiency usually results from the feeding of commercial or homemade foods with an inadequate thiamine content.

The incidence of the disease in the dog and cat populations in New Zealand is unknown, but is not common. The MAF Quality Management (MQM) Batchelar Animal Health Laboratory, which receives submissions from veterinary practitioners throughout the lower half of the North Island, diagnoses a few cases each year, mainly in dogs.

Animals with thiamine deficiency disease usually present with acute neurological signs, which are preceded by anorexia. These signs typically become progressively more severe over several days and often, particularly in dogs, end in death. In dogs the signs commonly develop in the following chronological order: anorexia, emesis, lethargy, CNS depression, paraparesis, ataxia, spasticity, tonic-clonic convulsions and death⁽¹⁾. Occasionally dogs die suddenly without neurological signs and this is believed to be caused by acute cardiac failure⁽¹⁾. In cats the convulsions are characterised by kyphosis (ventroflexion of the head), hyperaesthesia and dilated unresponsive pupils⁽²⁾. Parenteral thiamine injection early in the course of the disease usually results in rapid complete recovery.

Diagnosis is often based on the response

to thiamine injection, but can be confirmed by blood testing. The MQM Animal Health Laboratory network offers a thiamine assay (requiring 2 ml EDTA blood) and a blood thiamine concentration of 50-80 nmol/l is regarded as marginal. The histopathological brain lesions are characteristic. These consist of bilaterally symmetrical foci of haemorrhagic necrosis in periventricular grey matter involving consistently, and most severely, the caudal colliculi, but often also other brain stem nuclei⁽³⁾. A few dogs have focal myocardial degeneration⁽³⁾.

Carnivores do not synthesise thiamine or store significant amounts in the body and therefore depend upon a regular dietary intake to maintain good health⁽⁴⁾. The AAFCO (Association of American Feed Control Officials) minimum required concentrations of thiamine in dog and cat foods are 1.0 and 5.0 mg/kg DM, respectively⁽⁵⁾. Meat, fish, cereal grains, dairy products and yeast extracts are significant sources of thiamine⁽⁴⁾. However, thiamine is heat labile and cooking or processing can result in losses⁽⁶⁾. Some fish species contain a thiaminase, which may significantly reduce the thiamine content of the food⁽⁷⁾. Cats in particular should not be fed tinned fish exclusively unless it is labelled as being AAFCO tested. Sulphites, which are commonly added as preservatives to fresh pet minced meat, also destroy thiamine⁽⁸⁾.

Manufactured pet foods cannot be assumed to contain an adequate thiamine concentration unless the label states that

the food is 'complete and balanced'. Such a claim is ideally substantiated by AAFCO feeding trials or by meeting AAFCO established nutrient levels for cat or dog foods. However, under New Zealand pet food regulations, the basis for the claim does not have to be specified on the label.

Most cases of thiamine deficiency diagnosed at MQM Animal Health Laboratories have involved dogs fed exclusively on a single brand of untested commercial dog roll. A few cases have been seen in pets fed exclusively on fresh minced meat, which may have contained sulphite preservatives. The disease has also occasionally affected farm dogs on unsupplemented diets of cooked mutton or beef.

Pet owners can follow an easy rule-of-thumb to ensure that their animal's intake of thiamine and other nutrients is sufficient. At least 70% of the dog's or cat's food should be comprised of a commercial complete and balanced food, and up to 30% of the diet may consist of untested commercial foods or homemade foods. However, if owners give their pets mainly untested commercial food, they are less likely to encounter nutritional disorders if they feed a mixture of brands and types of food (dry, rolls, canned).

References

- (1) Read DH, Harrington DD. Experimentally induced thiamine deficiency in Beagle dogs:

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- Clinical observations. *American Journal of Veterinary Research* 42, 984-91, 1981.
- (2) Oliver JE, Greene CE. Diseases of the brain. In: Ettinger SJ (ed). *Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat* (2nd edition). Pp 460-532. WB Saunders, Philadelphia, 1983.
 - (3) Jubb KVF, Huxtable CR. The nervous system. In: Jubb KVF, Kennedy PC, Palmer N (eds). *Pathology of Domestic Animals* (4th edition). Pp 267-439. Academic Press, San Diego, 1993.
 - (4) McDowell LR. *Vitamins in animal nutrition*. Academic Press, San Diego, 1989.
 - (5) Association of American Feed Control Officials: *Official Publication AAFCO Inc*, Atlanta, 1993.
 - (6) Read DH, Jolly RD, Alley MR. Polioencephalomalacia of dogs with thiamine deficiency. *Veterinary Pathology* 14, 103, 1977.
 - (7) Jubb KV, Saunders LZ, Coates HV. Thiamine deficiency encephalopathy in cats. *Journal of Comparative Pathology* 66, 217-27, 1956.
 - (8) Studdert VP, Labuc RH. Thiamine deficiency in cats and dogs associated with feeding meat preserved with sulphur dioxide. *Australian Veterinary Journal* 68, 54-7, 1991.

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