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CASE REPORT
Thiamine deficiency in a cat: resolution of MRI abnormalities following thiamine supplementation

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Thiamine (vitamin B_1) is an essential component of a number of metabolic pathways and thiamine deficiency results in a progressive encephalopathy in both humans and animals. Confirming thiamine deficiency is problematic and relies on demonstrating reduced red blood cells transketolase activity, or indirect methods including urinary organic acid analysis and dietary analysis. The characteristic and selective vulnerability of different brain regions in carnivores has been demonstrated by magnetic resonance (MR) imaging in the dog and cat as an aid to diagnosis. A 2-year-old, female, domestic shorthair cat was presented with an acute onset of seizures and ataxia. MR imaging was consistent with thiamine deficiency and supplementation resulted in a progressive clinical improvement. Repeated MR imaging 4 days after starting thiamine supplementation revealed near complete resolution of the MR abnormalities. Repeated MR imaging following appropriate therapy may be useful to further confirm thiamine deficiency.

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A 2-year-old, neutered female, domestic shorthair cat was referred to Dick White Referrals (DWR) for evaluation of seizures, ataxia and altered behaviour. The cat was missing for 48 h preceding the onset of the neurological signs. At presentation to the referring veterinary surgeon the seizures were described as multiple episodes of partial complex seizures with pupillary dilation, cervical ventroflexion and disorientation and lasting between 10 and 30 s each. There was no history of any exposure to toxins or trauma and prior to going missing the cat had no medical problems and was fully vaccinated and dewormed. The cat was fed on a diet exclusively comprising cooked or smoked salmon, cooked chicken and milk. Complete physical examination at DWR demonstrated no clinical abnormalities besides the neurological abnormalities. Neurological examination revealed the cat to be obtunded, poorly ambulatory with tetraparesis and ataxia of all four limbs and blind. The segmental spinal reflexes were increased in all four limbs. Assessment of the eyes demonstrated bilateral mydriasis with bilaterally decreased pupillary light reflexes and menace responses. A Schirmer I tear test revealed tear production of 13 mm/min in the right eye and 9 mm/min in the left eye (normal = 20 ± 5 mm/min). No other deficits of cranial nerve function were present. The neuroanatomical localisation was multifocal intracranial.

Magnetic resonance (MR) imaging was performed under general anaesthesia using a 0.4 Tesla permanent magnet (Hitachi Aperto, Tokyo, Japan). The following pulse sequences were obtained: T1-weighted pre- and post-contrast (total dose of 0.5 mmol (1 ml) of gadoteric acid (Dotarem; Guerbet, Roissy, France)), T2-weighted and fluid-attenuated inversion recovery (FLAIR). Images were acquired in transverse and sagittal planes. Bilaterally symmetrical hyperintense foci were observed on both T2-weighted and FLAIR images affecting the lateral geniculate nuclei (Fig 1A and B), caudal colliculi (Fig 2A), facial nuclei (Fig 2A) and medial vestibular nuclei (Fig 2B). The hyperintense lesions on T2-weighted and FLAIR images appeared hypointense on T1-weighted images (Fig 1C). No contrast enhancement was evident on post-contrast T1-weighted images. Analysis of cerebrospinal fluid collected from the cisterna magna was consistent with blood contamination, but was otherwise unremarkable. Blood serological tests for toxoplasma IgG/IgM, coronavirus titres, feline immunodeficiency

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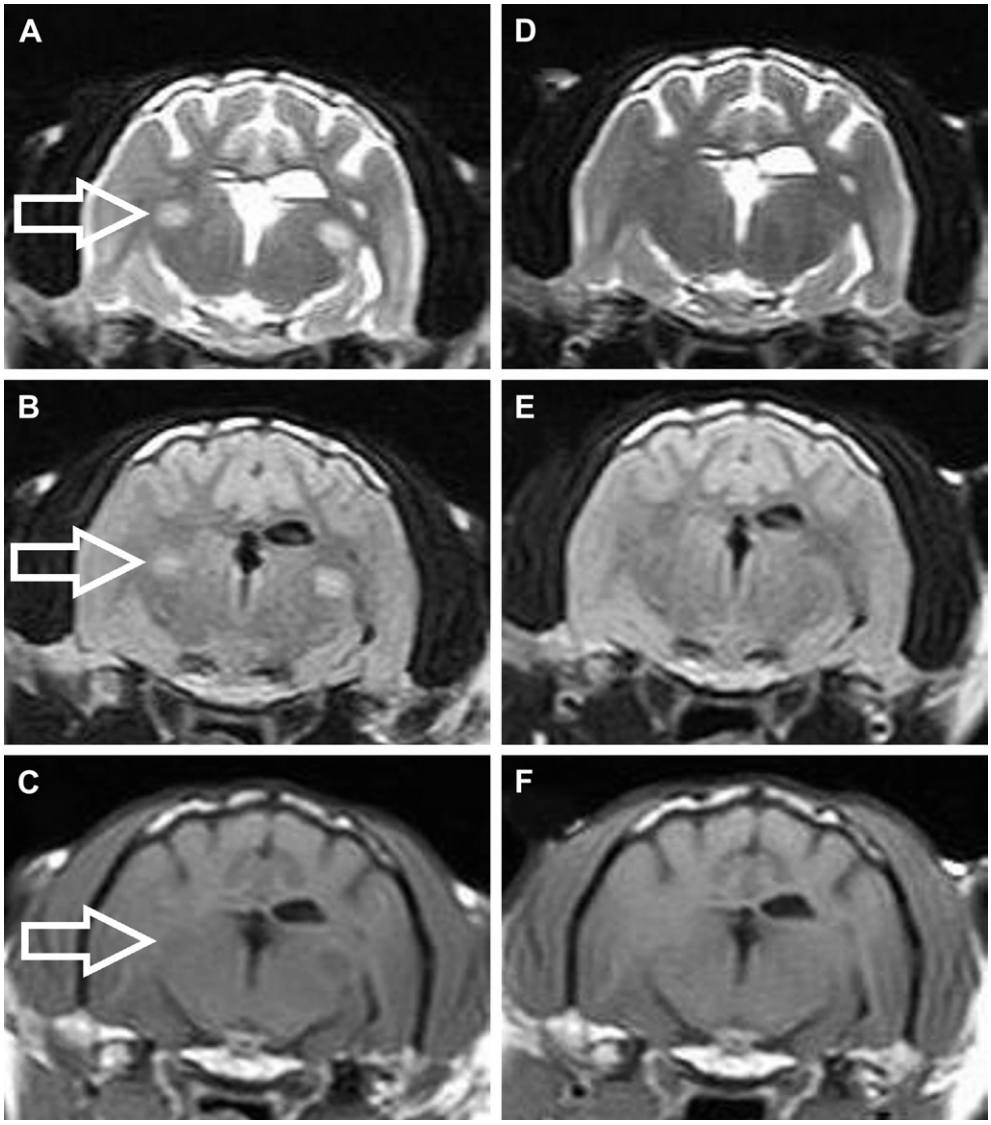

Fig 1. Transverse MR images at the level of the thalamus in a cat with thiamine deficiency: T2-weighted (A), FLAIR (B), T1-weighted (C) sequences. Bilaterally symmetrical hyperintense lesions are present on both T2-weighted (A) and FLAIR (B) images affecting the lateral geniculate nuclei (arrow). The hyperintense lesions on T2-weighted and FLAIR images are hypointense on a T1-weighted (C) image (arrow). Transverse images (D, E, F) are corresponding to the images (A, B, C), 4 days after initiation of thiamine supplementation. The symmetrical hyperintense lesions on T2-weighted (A), FLAIR (B) and hypointense lesions on T1-weighted images (C) have largely resolved.

virus, feline leukaemia virus, feline calicivirus, herpesvirus and parvovirus were negative.

A presumptive diagnosis of thiamine (vitamin B_1) deficiency was made in this case on the basis of the diet, the clinical and neurological presentation and the MR findings.[1,2] Confirmation of a presumptive diagnosis of thiamine deficiency is also possible through demonstrating a rapid clinical response to supplementation with thiamine. It has also been suggested that the earlier thiamine supplementation is instigated the better the clinical outcome.[3] In this case thiamine supplementation was started immediately following MR imaging and comprised dietary supplementation with oral thiamine at a total dose of 50 mg q 12 h for 1 month (Thiamine tablets; Natrahealth, UK), subcutaneous injections of a vitamin B-complex (Anivit 4BC; Norbrook Laboratories, Newry, Northern Ireland) at an initial dose of 4 ml (140 mg of thiamine), followed by twice daily injections of 1 ml (35 mg of thiamine) for 4 days and a change in the cat's diet to include a commercial cat food. The clinical signs resolved over the following 4 days, including a return of normal vision and restoration of normal tear production in both eyes. MR imaging of the brain was repeated at 4 days after starting thiamine supplementation and this demonstrated virtually complete resolution

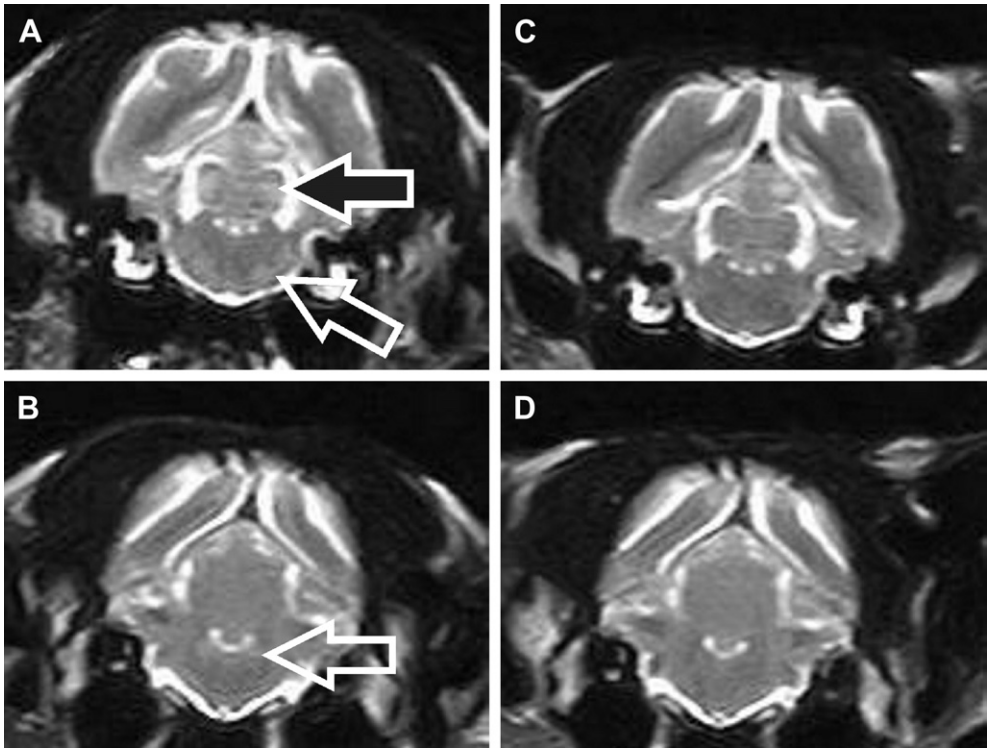

Fig 2. Transverse T2-weighted MR images in a cat with thiamine deficiency at presentation (A and B) and 4 days after starting thiamine supplementation (C and D). Bilaterally symmetrical hyperintense lesions are evident at the level of the caudal colliculi (A, filled arrow), facial nuclei (A, open arrow) and medial vestibular nuclei (B, arrow). Following 4 days of thiamine supplementation there is near complete resolution of the MR imaging lesions (C and D).

of the MR imaging changes (Fig 1D–F and Fig 2C and D). One year after initial presentation, a follow-up clinical examination demonstrated no clinical or neurological deficits.

Thiamine is essential in carbohydrate, amino acid and fatty acid synthesis and metabolism, comprising an essential component of transketolase and 2-oxoacid dehydrogenases.[4] Thiamine is destroyed by heat, sulphur preservatives and in diets with high thiaminase activity, such as raw fish. Thiamine deficiency results in a progressive encephalopathy in both humans and animals, with the particular vulnerability of the brain due to its reliance on metabolic pathways requiring thiamine for energy metabolism and neurotransmitter synthesis.[5] Thiamine deficiency is a well-recognised entity in carnivores. Cases of naturally occurring or experimentally produced thiamine deficiency have been described in cats,[2,6,7] dogs,[1,8–10] mink,[11] and foxes.[12] The neurological signs of thiamine deficiency in cats vary, and may include impaired vision, mydriasis, ataxia, vestibular signs and seizures. These signs are often preceded by anorexia and vomiting.[2,7] Thiamine deficiency has been described in cats in connection with a variety of diets: meat preserved with sulphur dioxide,[13] commercial food,[2] or fish containing thiaminase.[6] In this report, the cat had been fed exclusively on a diet of cooked chicken, milk and salmon that was either cooked or smoked. It is likely that the thiamine deficiency in this case was caused by the cooking process degrading the thiamine, rather than due to high thiaminase levels within the diet as thiaminase is destroyed by cooking and it has been reported that smoked salmon does not contain thiaminase.[14] The diagnosis of thiamine deficiency in carnivores may be suspected on the basis of the history (in particular diet) and clinical presentation, but absolute confirmation is technically difficult and relies on the demonstration of reduced transketolase activity in red blood cells, or other indirect methods including urinary organic acid analysis and dietary analysis.[1,2,15] Several mechanisms have been implicated in pathogenesis of selective neuronal loss in thiamine deficiency, including impaired cerebral energy metabolism, lactic acidosis or the release of glutamate.[16] The pathological features of thiamine deficiency in cats have been described and are characterised by symmetrical petechial haemorrhages located in the caudal colliculi, lateral geniculate bodies, medial vestibular, oculomotor, red and habenular nuclei and periaqueductal grey matter. Histopathological features are characterised by haemorrhages, neutrophil oedema, neuronal degeneration, gliosis and prominent capillaries with oedematous endothelial cells.[3,6,7,13] This selective vulnerability of consistent grey matter regions of the brain evident on neuropathology has previously also been demonstrated by

MR imaging in both the dog and cat and this can be a useful aid in the diagnosis.[1,2]

This report highlights the value of MR imaging in the diagnosis of thiamine deficiency, with additional confirmation of the diagnosis through resolution of the MR abnormalities occurring rapidly in response to appropriate therapy. If MR imaging is planned in a case of suspected thiamine deficiency it is important to recognise that the MR imaging features of thiamine deficiency will resolve relatively quickly following supplementation with thiamine and MR imaging, therefore, cannot be relied upon to make a retrospective diagnosis of thiamine deficiency once treatment has been started and clinical improvement has occurred.

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