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Case Report

Clinical signs, MRI features, and outcomes of two cats with thiamine deficiency secondary to diet change

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Two cats were presented with vestibular signs and seizures. Both cats were diagnosed with thiamine deficiency. The transverse and dorsal T2-weighted magnetic resonance (MR) images revealed the presence of bilateral hyperintense lesions at specific nuclei of the midbrain, cerebellum, and brainstem. After thiamine supplementation, the clinical signs gradually improved. Repeated MR images taken 3 weeks after thiamine supplementation had started showed that the lesions were nearly resolved. This case report describes the clinical and MR findings associated with thiamine deficiency in two cats.

Keywords: cat, MRI, thiamine deficiency

Thiamine is water-soluble vitamin B (B1) that acts as an essential cofactor for carbohydrate metabolism [2]. Thiamine-dependent biochemical processes includes the citric acid cycle and pentose phosphate pathway, which are major energy production process in the central nervous system (CNS) [2,9]. Since thiamine deficiency results in disturbances of energy metabolism, alternative anaerobic energy metabolism processes promote the accumulation of lactic acid in the CNS and lead to neuronal loss [2]. Carnivores must obtain thiamine from the environment due to impaired autosynthesis and minimal storage [4,6]. Causes of thiamine deficiency include consumption of raw fish that often contains thiaminase, cooked food in which the thiamine has been destroyed due to heating, or meats preserved with sulphite that inactivates thiamine [4,8-10].

Clinical signs of thiamine deficiency include various vestibular signs, vision loss, mydriasis without pupil light reflexes, incoordination, ataxia, and seizures [1,6]. Experimentally, thiamine deficiency produces typical histologic lesions such as bilaterally symmetric pannecrosis of brain tissue with peripheral hemorrhage and gliosis [2,5]. These pathological features are characterized as selective vulnerabilities of the brain at specific locations. The lesions predominantly form at specific nuclei of the midbrain, cerebellum, and brainstem [2,5,7,8]. Although diseases associated with thiamine deficiency have been reported in various species, diagnosis based on characteristic lesions viewed with magnetic resonance images (MRI) has only been conducted in one dog [3] and two cats [7,8].

Here, we report the clinical signs, MRI findings, and outcomes of two cats with a thiamine deficiency.

A 9-year-old, castrated male domestic short hair cat (Case 1) and a 6-year-old, spayed female Persian cat (Case 2) were presented with acute hindlimb paraparesis and seizures, respectively. Neither cats had a history of previous medical problems or vaccination while both had been housed indoors and fed commercial dry cat food. Their diets had been changed to boiled anchovies, shrimp, and cooked meats for Case 1, and stale food for Case 2 over the preceding 2 weeks or 3 month, respectively.

In Case number 1, clinical signs progressed rapidly and tetraparesis with rigidity developed. These signs were accompanied by intermittent ventroflexion of the head and vocalization that were precipitated by handling. Vestibular signs including head tilt and positional vertical nystagmus were also observed. The pupils were dilated without a pupillary light response (PLR) and menace response. In Case number 2, the owner stated that seizures were noted 3 days before referral to the clinic. On presentation, this cat was laterally recumbent. During the neurological examination, pupillary dilation with delayed PLR, paresis in the pelvic limbs, and cervical ventroflexion were noted. Marked incoordination, tremors, and decreased menace response were also observed.

Results of the complete blood count and routine radiographs for both cats were unremarkable. Serum

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biochemistry revealed markedly elevated muscle enzymes and mild azotemia in both animals. Based on the clinical signs as well as the physical and neurological examinations, the presence of intracranial lesions was suspected. Major diagnostic differentials for the presenting problems including vestibular signs, vision loss, mydriasis with a lack of (or delayed) PLR, incoordination, ataxia, and seizures in these cats were toxin ingestion, inflammatory or infectious CNS disorders, multifocal neoplasia of the brain, metabolic disease, and thiamine deficiency.

MRI of the head using a 0.2 T machine (E-scan; ESAOTE, Italy) was performed on both cats. T1- and T2-weighted images (WI) along with a postcontrast T1-WI were obtained. MRI findings for the two cats were indicative of characteristic bilaterally symmetrical lesions. Lesions with hyperintense signals both on T1- and T2-WI were seen at the lateral geniculate nucleus, caudal colliculi, medial vestibular nuclei, and cerebellar nodulus. On the transverse images, bilateral lesions at specific neuroanatomical sites were more apparent, consistent with thiamine deficiency (Figs. 1A, C, E, and 2A～C). Areas of bilateral hyperintensity were also observed on the dorsal T2-WI at the lateral geniculate body and cerebral peduncle regions (Figs. 3A and C, and 2D and E). These lesions were not enhanced on T1-WI after intravenous administration of gadolinium (0.1 mmol/kg, IV, Omniscan; Amersham Health, USA). Results of the cerebrospinal fluid (CSF) analysis were normal. PCR findings for blood and CSF samples along with antibody titers of serum and CSF samples taken to identify virus and parasite infection were all negative. Electrolytes and serum thyroxin levels were normal, and neither cat had a history of toxicity. Based on

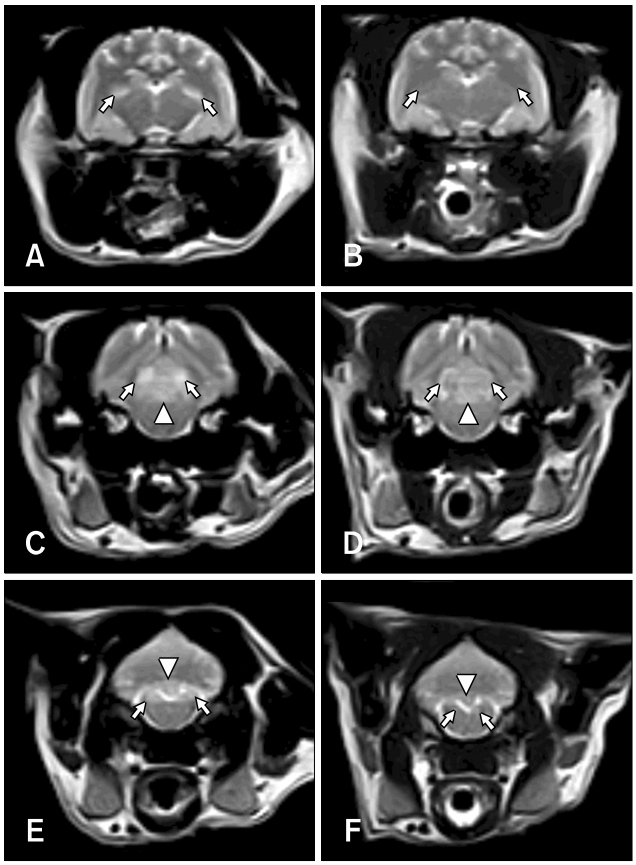

Fig. 1. Transverse T2-weighted MR images (WI) of (A, C, and E) a cat with thiamine deficiency (Case 1). (A) At the level of the thalamus, symmetrical hyperintense signals at the lateral geniculate nuclei (arrows) were noted. (C) Images of the caudal colliculi (arrows) and periaqueductal gray matter (arrowhead) revealed the presence of hyperintense lesions. (E) Bilateral symmetrical hyperintensities were also evident in the medial vestibular nuclei (arrows) and cerebellar nodulus (arrowhead). After 3 weeks of thiamine administration, transverse MR images showed that the lesions had resolved except for ones in the cerebellar nodulus (B, D, and F).

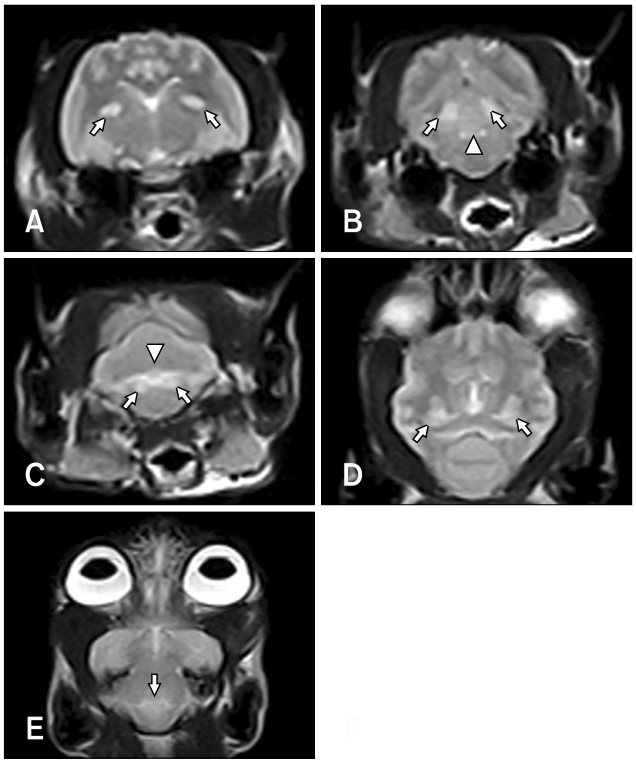

Fig. 2. Transverse (A～C) and dorsal (D and E) T2-WI of a cat with thiamine deficiency (Case 2). (A) Symmetrical hyperintense signals in the lateral geniculate nuclei (arrows) were noted at the level of the thalamus. (B) Images of the caudal colliculi (arrows), periaqueductal gray matter (lesions between the arrows), and facial nuclei (arrowhead) revealed the presence of hyperintense lesions. (C) Bilateral symmetrical hyperintensities were also evident in the medial vestibular nuclei (arrows) and cerebellar nodulus (arrowhead). Bilateral symmetrical hyperintensities were observed in the lateral geniculate body (D; arrows) and cerebral peduncle (E; arrow). After 3 weeks of thiamine administration, clinical signs were nearly resolved. However, additional MRI was not attempted for this cat.

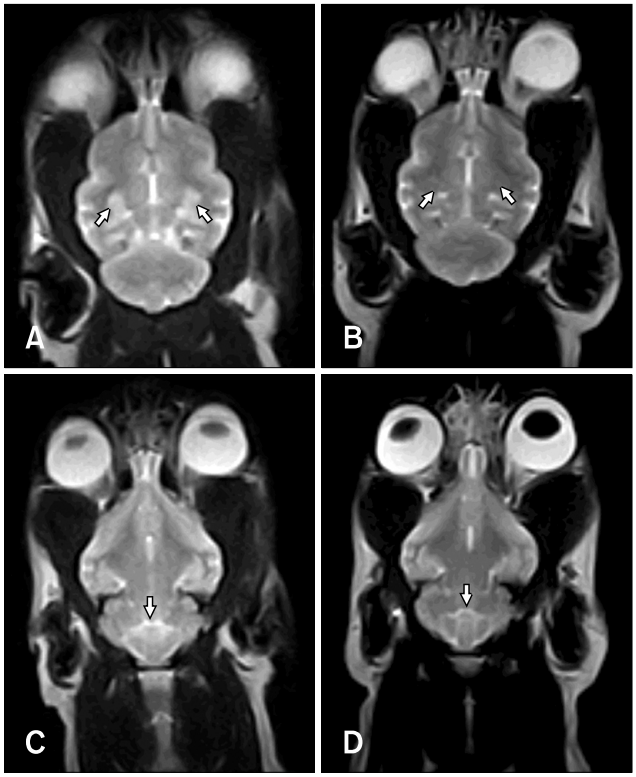

Fig. 3. Dorsal T2-WI of a cat with thiamine deficiency (Case 1). Bilateral, symmetrical hyperintense lesions at the lateral geniculate body (A; arrows) and cerebral peduncle (C; arrow) were observed. Panels B and D correspond to the images shown in panels A and C after 3 weeks of thiamine supplementation. Completely resolved lesions were noted.

the animals' history, clinical signs, and MRI findings, the cats were diagnosed with thiamine deficiency.

Both cats were given vitamin B complexes (Vitamedin; CJ Pharma, Korea) at a total dose of 50 mg thiamine hydrochloride twice daily. For Case 2, oxygen supplementation and vitamin B complexes [an initial dose of 70 mg/kg body weight (BW), intravenous injection, followed by 30 mg/kg BW, subcutaneous injection, for 3 more days; Yuhan, Korea] were initially administered. Three days after thiamine supplementation, the neurological abnormalities resolved significantly including recovery of vision and disappearance of vestibular signs. One week later, no other clinical signs were observed in Case 1. Mild tremors and incoordination were still noted in Case 2. However, these clinical signs resolved 3 weeks after presentation. After 3 weeks of thiamine supplementation, MRI was repeated on Case 1 to reassess the lesions. Except for changes in the cerebellar nodulus, the lesions had resolved (Figs. 1 and 3). Long-term supplementation with thiamine along with consumption of a nutritionally balanced diet was recommended.

Thiamine deficiency results in energy metabolism disturbance in the CNS and several types of neurological dysfunction [2]. Carnivores must obtain thiamine from the environment because this compound cannot be autosynthesized [4,6]. Thiamine deficiencies associated with feeding on raw fish, home cooked food, and meats preserved with sulphite have been previously been reported in dogs and cats [1,8-10]. Various balanced diets have recently been commercialized in veterinary medicine, and diseases associated with nutritional deficits are now less common. Nevertheless, commercial diets can occasionally induce thiamine deficiency in cats [7]. In the present study, one cat (Case 1) was fed boiled anchovies, shrimp, and cooked meats. Because high temperatures can destroy thiamine, dietary change was likely the chief cause of thiamine deficiency in this animal. The other cat (Case 2) was fed commercial cat food; however, it was stored for several months under poor conditions.

In this case report, the clinical and MRI findings for two cats with thiamine deficiency were presented. Numerous neurological signs including multiple vestibular signs and cranial deficits were observed. Upon MRI examination, symmetrical hyperintense lesions in the lateral geniculate body, caudal colliculi, cerebellar nodulus, and medial vestibular nuclei consistent with thiamine deficiency were detected. After thiamine supplementation, clinical signs were resolved and lesions at specific sites improved. These findings were confirmed by MRI re-examination in one cat. There are few case reports of MRI characteristics of thiamine deficiency in dogs and cats. In this study, the two animals showed characteristics similar to those described in previous case reports [3,7,8].

Palus *et al.* described the resolution of MRI abnormalities following thiamine supplementation in a cat [8]. In that case, lesions in the animal had completely resolved upon repeated MRI examination performed 4 days after treatment. Although the cats in the present study received long-term thiamine treatment (3 weeks), minimal lesions such as ones in the cerebellar nodulus persisted. Similarly, MRI findings in a dog with thiamine deficiency revealed the presence of remnant lesions 8 weeks after thiamine supplementation despite dramatically improved clinical signs [3]. Considering the different progression of these cases, longer follow-up periods, patient monitoring, and vitamin supplementation appear to be required for complete recovery.

We described distinctive features of MRI lesions based on transverse and dorsal images. Generally, bilateral lesions extending from the midbrain to brainstem can be more easily detected on transverse images than sagittal or dorsal images. In previous reports describing MR images, abnormalities were detected on the transverse images [3,4,7]. In the present cases, lesions affecting the lateral geniculate body were more evident on the dorsal images than the transverse images. Minimally increased signals

can go unnoticed because the lesions predominantly affect specific nuclei in cases of thiamine deficiency and the nuclei appear slightly hyperintense compared to other regions. Therefore, repeated multiple views of MR images are required to accurately evaluate brain lesions in animals with thiamine deficiencies. In addition to multiple planes, sequences with suppression of CSF (fluid-attenuated inversion recovery) may be used to detect subtle lesions.

In conclusion, thiamine deficiency was identified in two cats based on patient history, specific clinical signs, and MR images of brain lesions. In this case report, we described distinct features of thiamine deficiency in the cats due to consumption of cooked foods or a commercial diet that had been improperly stored. It is important to recognize thiamine deficiency based on a differential diagnosis for cats with neurologic signs and a history of diet change. Moreover, repeated multiple views of characteristic MR images could be valuable for accurately diagnosing and confirming the response to treatment.

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