

## CASE REPORT

# Thiamine deficiency in a dog associated with exclusive consumption of boiled sweet potato (*Ipomoea batatas*): Serial changes in clinical findings, magnetic resonance imaging findings and blood lactate and thiamine concentrations

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**Abstract**

Thiamine (vitamin B<sub>1</sub>) is an essential nutrient that significantly influences ATP production in the body. It needs to be supplemented consistently through an exogenous source to prevent deficiency; however, it is easily affected by a variety of mitigating factors. Additionally, thiamine requirements can be influenced by an individual's dietary composition. The nervous system is particularly vulnerable to thiamine deficiency due to its high metabolic demand. Thiamine deficiency is typically diagnosed based on clinical signs, dietary history and response to thiamine administration. A 5-year-old neutered male Maltese Terrier dog presented with an acute onset of seizures and generalized ataxia. The dog was exclusively fed boiled sweet potato (*Ipomoea batatas*) as a primary diet source for 4 weeks. MR findings and hyperlactatemic conditions were consistent with thiamine deficiency, and the diagnosis was confirmed by measuring thiamine concentrations in blood using high-performance liquid chromatography (HPLC). Appropriate thiamine supplementation and diet changes resulted in a rapid improvement in neurological signs. Repeated MR imaging 2 weeks after starting the treatment completely resolved the previously identified abnormalities, and repeated measurements of blood lactate and thiamine levels revealed complete recovery of the thiamine-deficient status.

**KEYWORDS**

dog, HPLC, lactate, MR imaging, thiamine deficiency, vitamin B<sub>1</sub>

## 1 | INTRODUCTION

Thiamine (vitamin B<sub>1</sub>) is an essential micronutrient that significantly influences carbohydrate metabolism and energy production. Thiamine pyrophosphate (TPP) is the phosphorylated form of thiamine and acts as a necessary cofactor in the enzymatic pathways of carbohydrate metabolism. TPP facilitates the conversion of pyruvate

into acetyl-CoA and alpha-ketoglutarate into succinyl-CoA in the tricarboxylic acid (TCA) cycle, and mediates transketolase activity for the synthesis of nucleotides from ribose 5-phosphate in the pentose phosphate pathway (Manzetti, Zhang, & van der Spoel, 2014). Therefore, thiamine is essential to maintain the energy metabolism and normal functioning of the body. Unfortunately, thiamine is one of the most poorly stored vitamins in most mammals, considering

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their inability to synthesize sufficient amounts of thiamine endogenously, due to which it needs to be consistently supplemented by ingesting an exogenous source to prevent deficiency (Council, 2006). While thiamine is found in a wide variety of foods, dietary thiamine is easily excreted from the body because of its water solubility, and stored in very small amounts in only a limited portion of major organs (Garosi et al., 2003). In addition, thiamine is easily destroyed by environmental stressors and food processing procedures, and is, therefore, vulnerable when maintaining its original structure and function (Combs and McClung, 2016; Tran, Hendriks, & van der Poel, 2008). Considering this, dogs and cats may fail to meet the thiamine requirements despite adequate dietary precautions in some circumstances.

Thiamine deficiency results in insufficient ATP production in the body with subsequent systemic dysfunction. Neurologic tissues have particularly high energy requirements, and the energy required to maintain normal brain function is chiefly covered by the degradation of glucose. Therefore, thiamine deficiency leads to brain damage and produces characteristic neurologic and pathological manifestations in dogs and cats (Houston & Hulland, 1988; Palus, Penderis, Jakovljevic, & Cherubini, 2010; Read & Harrington, 1986). Neurological dysfunctions in dogs and cats typically include ataxia, proprioceptive deficits, vestibular signs, mental alteration, spastic ventroflexion, blindness, pupillary dilation and seizure activity (Markovich, Heinze, & Freeman, 2013). The pathological features are characterized by bilaterally symmetric hemorrhagic and necrotic lesions in the grey matter of the brain, which affect the nuclei of the caudal colliculi (Houston & Hulland, 1988; Palus et al., 2010; Singh, Thompson, Sullivan, & Child, 2005; Steel, 1997). Considering these characteristics, brain MRI scans have been widely used to diagnose thiamine deficiency-induced encephalopathy in dogs and cats. Additional dysfunctions associated with thiamine deficiency in dogs and cats are variable and tend to be non-specific, but can commonly involve the gastrointestinal tract, cardiac and ocular systems (Markovich et al., 2013).

Well-known causes of thiamine deficiency in dogs and cats include ingestion of thiaminase-containing fish (Houston & Hulland, 1988; Smith and Proutt, 1944), sulphur-containing meat (Singh et al., 2005; Steel, 1997) or destruction of dietary thiamine by cooking or processing (e.g., heat, oxygen and neutral or alkaline pH) (Moon, Kang, & Park, 2013; Palus et al., 2010). Insufficient food consumption can also increase the risk of developing thiamine deficiency. Furthermore, dietary composition has been considered to influence thiamine requirements. One previous human nutrition research revealed that increasing the consumption of dietary carbohydrate can decrease the plasma and urine levels of thiamine (Elmadfa, Majchrzak, Rust, & Genser, 2001). This is likely due to the increased consumption of thiamine according to the activation of the TCA cycle for carbohydrate metabolism. However, the interactions between dietary carbohydrate and thiamine requirements have not yet been well studied in dogs and cats.

Here, we report the successful management of thiamine deficiency in a dog due to excessive intake of boiled sweet potato.

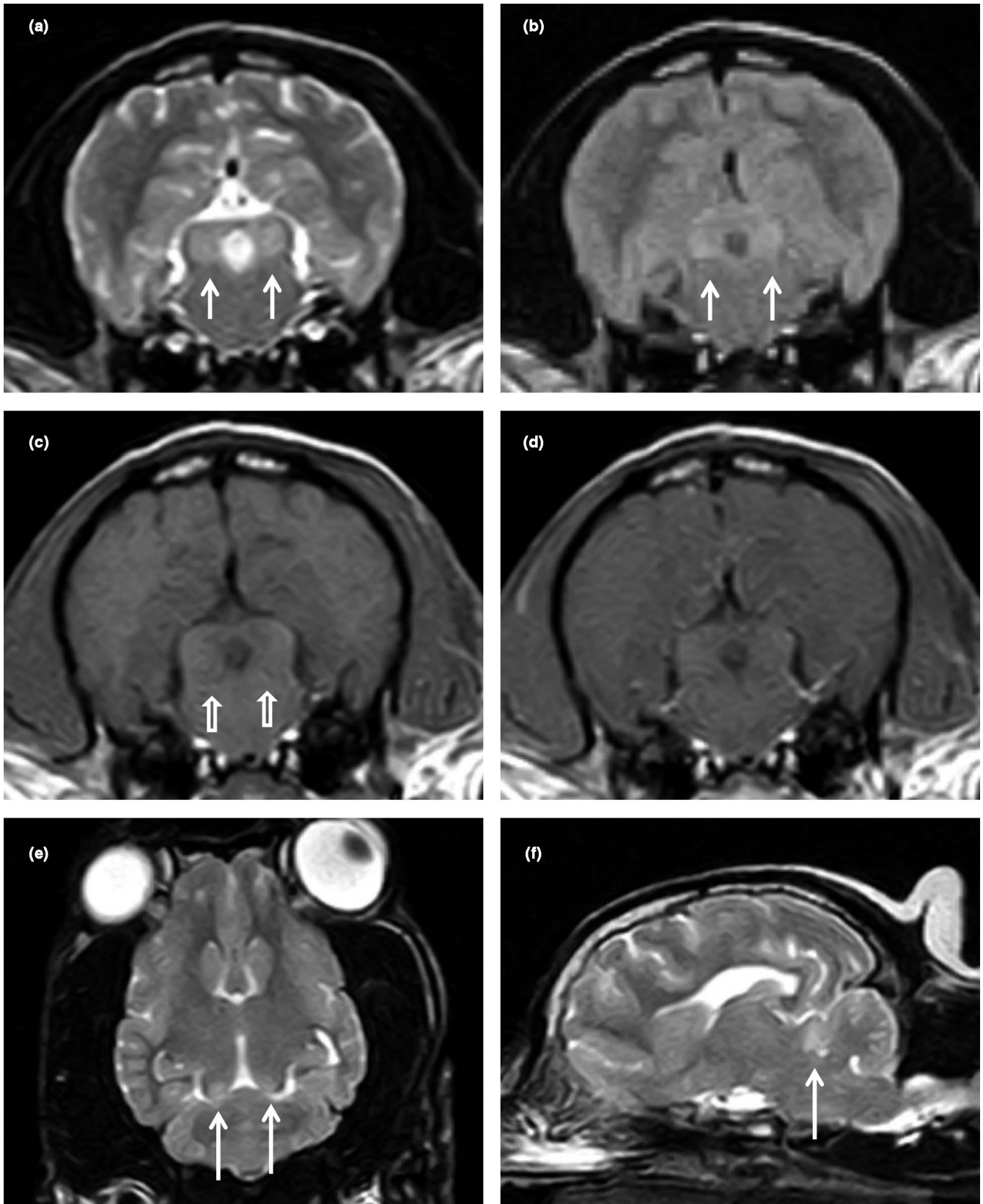
Although dogs with thiamine deficiency have been reported in a few previous studies, thiamine deficiency or exacerbation of thiamine-deficient status by the carbohydrate-rich diet coupled with low thiamine content has not yet been reported in the veterinary literature. Furthermore, to widen our knowledge of the diagnosis and treatment of thiamine deficiency, this study is intended to describe the detailed information of the serial changes in clinical and MRI findings. The diagnosis and monitoring based on blood TPP concentration by high-performance liquid chromatography have only been conducted in one feline case series (Marks et al., 2011), and not in dogs. We also have linked blood lactate levels with thiamine status to facilitate the diagnosis and screening of thiamine deficiency. Additionally, the diagnosis based on characteristic MRI findings has only been conducted in two dogs (Garosi et al., 2003; Singh et al., 2005), and only one of these dogs was subjected to repeated MRI scans following a period of thiamine supplementation.

## 2 | CASE DETAILS

A 5-year-old neutered male Maltese Terrier dog was presented to a referring veterinarian with a 3-day history of depression and anorexia followed by vomiting, seizure clustering and generalized ataxia. The owner noticed a progressive deterioration of the neurologic signs over the preceding 3 days. Symptomatic treatment before the presentation with phenobarbital (2 mg/kg twice a day orally) failed to alleviate the seizures.

The dog had been generally healthy until the occurrence of acute seizure episodes. He had been fully vaccinated and dewormed, with no history of trauma or exposure to toxins. Further medical enquiry with the owner revealed that the dog had been fed an almost exclusive diet of boiled sweet potato (*Ipomoea batatas*) as a primary diet source for 4 weeks with an average consumption of approximately 100–150 g (about one to one and a half small whole sweet potatoes) per day due to his picky eating behaviours (chronic food refusal). Furthermore, this food refusal had gradually worsened over time.

At presentation, the seizures were classified as generalized tonic-clonic with vomiting aura. The initial onset of seizures was 3 days before the admission, and the cluster seizures activity was recorded over three sequential days (more than three seizures in a 24-hr period). The average duration of the 3 ranged from 20 to 30 s, and the time interval between consecutive seizures was approximately 10 min. The last seizure episode verified by the owner was at least 6 hr before admission. Except for neurological abnormalities, other abnormalities were not noted on complete physical examination. Mentation was normal. Body condition (body condition score of 4 on a scale from 1 to 9), weight (4.2 kg) and muscle mass were all normal at the time of admission, but the patient had a previous history of obesity. The results of complete blood cell count were unremarkable. Serum biochemical analysis (Catalyst One Chemistry Analyzer, IDEXX Laboratories, Inc., ME, USA) revealed alkaline phosphatase activity of 298 U/L (reference range, 23 to 212 U/L) and alanine aminotransferase activity of 430 U/L (reference range, 10 to 125 U/L).

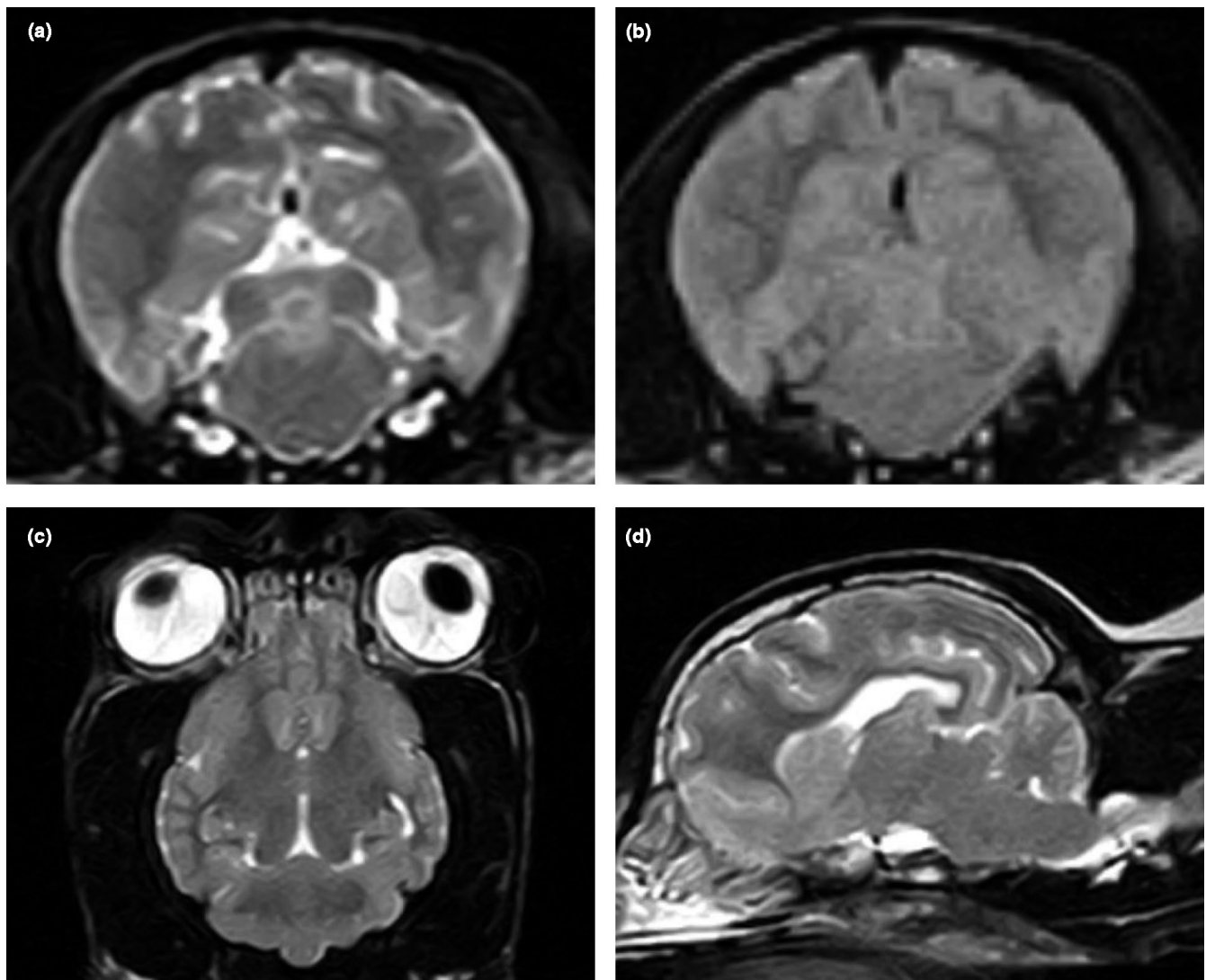


**FIGURE 1** Transverse (a–d), dorsal (e) and left parasagittal (f) plane MR images at the level of the caudal colliculi: T2W (a, e, and f), FLAIR (b), T1W (c) and CET1W (d) sequences. T2W and FLAIR images show bilaterally symmetrical focal hyperintensities in the region of the caudal colliculi (arrows). The identical lesions appear iso- to hypointense on the T1W image (empty arrow)

Blood lactate was measured by using the blood gas analyser (pHOx Ultra, Nova biomedical, MA, USA) and the concentration was significantly increased to 12 mmol/L (reference range, 0.5 to 2.5 mmol/L). The slightly elevated levels of hepatic enzyme activities seemed to be attributed to the phenobarbital administration. There was no clinical evidence to support the presence of hypoperfusion or tissue hypoxia. Survey radiographs of the head, thorax and abdomen were categorized as normal.

Neurological examination identified generalized ataxia with dysmetric movement and an inability to bear full weight on the hind limbs. Conscious proprioception was reduced in all four limbs. An assessment of the cranial nerves showed spontaneous vertical nystagmus in both eyes with an upward fast phase. Segmental spinal cord reflexes and cutaneous trunci reflexes were normal. No other neurological abnormalities were identified. The localization of neuroanatomical lesions was multifocal intracranial, including both the forebrain and brainstem.

The intracranial lesions were identified via brain magnetic resonance (MR) imaging using a 0.4 Tesla scanner (Aperto; Hitachi Medical Corporation, Tokyo, Japan) and cerebrospinal fluid (CSF) analysis (collected from the atlanto-occipital cistern tap using a 22-gauge needle). T1-weighted (T1W) images, T2-weighted (T2W) images, fluid-attenuated inversion recovery (FLAIR) images and contrast-enhanced T1-weighted (CET1W) images were obtained from the MRI scan. The CET1W images were obtained after intravenously injecting Omniscan (Gadolinium EDTA; GE-Healthcare, Little Chalfont, United Kingdom) at a dosage of 0.20 mmol/kg body weight. After acquisition of the MRI data and the CSF samples, mannitol (15% D-Mannitol inj., Daihan Pharm., Seoul, Korea) was administered as a bolus of 1 g/kg over 15 min for preventing a rebound of intracranial pressure after the general anaesthesia. MR images (Figure 1) revealed bilaterally symmetrical hyperintense lesions in the caudal colliculi, specifically in the T2W and FLAIR images. The lesions appeared iso- to hypointense on T1W images, with no



**FIGURE 2** Transverse (a and b), dorsal (c) and left parasagittal (d) plane MR images at the same anatomic level as in Figure 1 after 2 weeks of diagnosis and thiamine treatment: T2W (a, c and d) and FLAIR (b) sequences. The abnormal bilaterally symmetrical hyperintensity lesions detected on the initial MR images completely disappeared



evident contrast enhancement on CET1W images. These abnormal findings on MR images were considered to be a pathologic change in brain metabolic derangement. No abnormalities were found on conventional CSF analysis (total nucleated cell count of 3 cells/ $\mu$ L; reference range from 0 to 5 cells/ $\mu$ L, protein concentration of 15 mg/dl; reference range from 0 to 25 mg/dl). There was no cytological CSF evidence of relatively well-recognized CNS disorders (e.g. inflammatory, neoplastic and infectious causes). Therefore, based on the inadequate diet history, the hyperlactatemic condition (Type B hyperlactatemia) and the abnormalities seen on MRI, a presumptive diagnosis of thiamine deficiency was established in this patient.

To confirm our presumptive diagnosis of thiamine deficiency, the whole blood sample was submitted to evaluate the blood thiamine concentration. Thiamine content was measured by the high-pressure liquid chromatography (HPLC) method (Agilent 1,260 Infinity II LC system, Agilent Technologies, CA, USA) with the assistance of a commercial laboratory (Neodin Vetlab, Seoul, Korea). The HPLC is a preferable and reliable method of thiamine analysis in humans and animal studies, which helps by directly measuring the phosphorylated form of thiamine (thiamine pyrophosphate, TPP) within RBCs (Talwar, Davidson, Cooney, & Jo'reilly, 2000). TPP is the most biologically active form within the body of humans and animals and has a main role in carbohydrate metabolism (National Research Council, 2006). Following the MRI scan and the blood sampling, a therapeutic trial using thiamine supplementation with dietary intervention was performed on the same day to evaluate the associated response and to rapidly recover the clinical signs. Sweet potato supplementation was immediately stopped prior to starting the treatment. Thiamine supplementation was started with oral vitamin B complex (Beecom-C, Yuhan Corp., Seoul, Korea) at a total thiamine dose of 45 mg once a day for a month. Furthermore, the dog's diet was temporarily changed to a good-quality prescribed dog food containing high levels of protein and fat (Hill's Prescription Diet a/d, Hill's Pet Nutrition Inc., KS, USA).

A rapid improvement in neurological signs was seen with the initiation of thiamine supplementation and diet change. There were no additional seizures after being discharged from the hospital. Furthermore, the symptoms of anorexia improved along with the normalization of previous metabolic derangement with the treatment, which consequently accelerated the recovery rate of the remaining neurological abnormalities. Nystagmus resolved within the first 24 hr, and ataxia resolved over the following 2 days. The general condition also recovered gradually along with the recovery of neurological signs. Finally, no residual neurological deficits were observed at 3 days after continuous management. In addition, we identified that the blood level of TPP (40.2 ng/ml; reference range, 46.0 to 112.0 ng/ml, provided by the laboratory), which was measured before starting the treatment, was particularly low. Given these findings, the presumptive diagnosis of thiamine deficiency secondary to inadequate dietary intake was confirmed.

Two weeks after starting thiamine supplementation and dietary intervention, there were no abnormalities on physical and neurological examination. Repeated blood examination showed that the

previously observed biochemical abnormalities had entirely normalized: alkaline phosphatase activity of 98 U/L (reference range, 23 to 212 U/L), alanine aminotransferase activity of 82 U/L (reference range, 10 to 125 U/L) and blood lactate level of 0.9 mmol/L (reference range, 0.5 to 2.5 mmol/L). MR imaging was repeated at the same level as in Figure 1. The previously observed caudal colliculi lesions on the initial MR images had completely resolved (Figure 2). Additionally, re-evaluating the blood TPP levels indicated that its level was significantly above the normal limits (138.5 ng/ml; reference range: 46.0 to 112.0 ng/ml).

One month after the initial presentation, thiamine supplementation was discontinued, and the dog's diet was changed to quality well-balanced commercial maintenance dog food. The improvements were well maintained at a 1-year follow-up.

### 3 | DISCUSSION

Dogs and cats largely depend on the dietary intake of thiamine to achieve thiamine requirements that the body needs to function normally. Dietary thiamine requirements in adult dogs and cats are documented by the National Research Council (NRC) and the Association of American Feed Control Officials (AAFCO) (National Research Council, 2006). According to this, the minimum thiamine requirement for an adult dog is 0.56 mg per 1,000 kcal metabolizable energy, whereas the minimum thiamine requirement for an adult cat is 1.4 mg per 1,000 kcal metabolizable energy. Many commercial pet diet manufacturers follow these nutritional recommendations when formulating pet diets. However, unconventional diets such as unbalanced foods, raw foods and homemade diets are not nutritionally adequate to meet the dietary thiamine recommendations, which in turn increases the risk of developing thiamine malnutrition. The present patient was exclusively fed a diet of boiled sweet potato for 4 weeks and this nutritionally incomplete diet eventually led to the suboptimal thiamine status of the body. The sweet potatoes fed to the patient were well-boiled before feeding, and this heating procedure for cooking would have led to a sufficient amount of thiamine loss in sweet potatoes. Because of the low thiamine content in boiled sweet potato (0.09 mg per 100 g of boiled sweet potato) (Woolfe, 1989), a large quantity of sweet potatoes is needed to be significant to supply adequate amounts of thiamine. Furthermore, intake of dietary carbohydrates has been known to increase the dietary requirements for thiamine based on data from previous human studies (Elmadfa et al., 2001; Fattal-Valevski, 2011). Boiled sweet potatoes are not only low in thiamine, but rich in carbohydrates (approximately 80% of the dry matter in sweet potatoes) (Lou et al., 2006). Given this background, the consumption of boiled sweet potatoes may further increase the metabolic demand for thiamine and may precipitate thiamine deficiency in a short period. Previous canine studies were unable to provide sufficient information regarding the duration required to develop clinical signs attributable to thiamine deficiency. Despite the presence of several mitigating factors (e.g., the amount of thiamine in the food, the amount of food consumed, the uptake,

metabolism and excretion abilities of thiamine once it enters the body, etc.), in adult dogs with thiamine deficiency whose dietary history was relatively well recorded, the time lags between the supply of the defective food and the onset of the clinical signs were reported from approximately 6 weeks to 6 months (Houston & Hulland, 1988; Singh et al., 2005; Studdert & Labuc, 1991). Compared with previously reported cases, this case showed a relatively early disease onset and rapid aggravation of clinical manifestations. It is also likely that the atypical clinical course of this patient was caused by a carbohydrate-rich diet coupled with low thiamine content. Therefore, in suspected cases of thiamine deficiency or in all sick dogs, it is imperative to thoroughly study the dietary history to predict the clinical course of the condition. Furthermore, we suggest that dietary carbohydrate composition should also be considered as a risk factor for thiamine deficiency.

Chronic food refusal, which was initially thought to be picky eating behaviour in this case, was reversed to a certain degree after thiamine administration. Therefore, we suspected that the chronic food refusal of the patient was one of the clinical signs of thiamine deficiency. Currently, chronic poor food intake has been noted as a symptom of thiamine deficiency in only a few canine cases (Garosi et al., 2003; Houston & Hulland, 1988). However, considering the clinical course of the present case along with previous reports, inadequate thiamine status in dogs over a period of time would chronically influence appetite and food intake. Therefore, the assessment of thiamine status and empirical support with thiamine in dogs with decreased appetite might be helpful for the early diagnosis of thiamine deficiency and may prevent further progression of the disease.

Thiamine deficiency can be treated with thiamine supplementation and supportive management of symptoms, but the treatment response and prognosis may vary according to the associated aetiology, severity and duration of thiamine deficiency (Kritikos, Parr, & Verbrugghe, 2017). Previous case reports of dogs with thiamine deficiency documented that the neurological signs typically began to improve within a week after thiamine supplementation (if they had a response to thiamine supplementation) and required variable amounts of time for a full recovery depending on the severity of the case, ranging from 2 months to 2 years (Garosi et al., 2003; Houston & Hulland, 1988; Singh et al., 2005). Although thiamine requirements in cats are different from those in dogs (National Research Council, 2006), some cats in previous studies with a long history of thiamine deficiency had a significantly slower recovery rate and a poor prognosis (Chang, Chiu, Lin, Liu, & Liu, 2017; Moon et al., 2013; Palus et al., 2010; Penderis, McConnell, & Calvin, 2007). In some of these cats that had responded to thiamine therapy, a permanent neurological deficit (ataxia, tremor, weakness, etc.) occasionally remained after treatment discontinuation. One experimental study also demonstrated an irreversible neurological damage due to chronic thiamine deficiency based on the therapeutic response according to different disease stages of cats with thiamine deficiency (Everett, 1944). Given these points, it is likely that the neurological manifestations observed in the present case were caused by acute

reversible damage to the central nervous system; therefore, the patient responded favourably and rapidly to appropriate thiamine supplementation and achieved complete recovery within 3 days following treatment. Accordingly, permanent neurological damage secondary to thiamine deficiency can be prevented by an earlier diagnosis and intervention, which would subsequently affect the outcome of therapy.

Thiamine supplementation can be achieved with short-term parenteral administration of thiamine hydrochloride, followed by repeated oral administration of vitamin supplements. In addition to thiamine supplementation, dietary intervention with a change to a high-quality well-balanced diet should be considered as most patients with thiamine deficiency have a history of inadequate dietary thiamine intake. In this study, we first attempted to nutritional approach in managing thiamine deficiency in dogs. The dog in our study was immediately administered oral thiamine hydrochloride on the basis of his dietary history and MR observations. The diet was simultaneously changed to a low-carbohydrate, high-fat and high-protein diet to reduce the thiamine consumption by carbohydrates and induce "the thiamine-sparing effect" of fat and protein (Arnold & Elvehjem, 1939; Scott & Griffith, 1957). The level of thiamine dosage administered in this dog was lower than that conventionally used in previous reports (Garosi et al., 2003; Singh et al., 2005), and antiepileptic therapy or other treatments were not performed. Nonetheless, the patient showed dramatic, rapid and sustained responses after the treatment initiation, confirming that the dietary management was significantly effective. Therefore, dietary management based on an understanding of the interaction of thiamine with other nutrients may effectively help to correct thiamine deficiency in dogs. We also suggest that nutritional intervention with a low-carbohydrate, high-fat and high-protein diet can be effectively applied on all thiamine deficiency regardless of the cause. However, further studies are needed to clarify the relationship between diet composition and thiamine requirements in dogs.

Selective vulnerability of particular brainstem nuclei is a key feature of the neuropathology of thiamine deficiency (Houston & Hulland, 1988; Singh et al., 2005; Steel, 1997). MR imaging is an effective method identify these characteristic lesions and facilitate the diagnosis of thiamine deficiency in dogs and cats (Garosi et al., 2003; Penderis et al., 2007). MRI scanning in the present case could be used to detect the characteristic lesions on the caudal colliculi, and the abnormalities were entirely resolved on repeated scanning following 2 weeks of treatment. Therefore, this highlights the value of MRI in the diagnosis of thiamine deficiency and the importance of repeated MRI. However, the MR imaging-based approach has some disadvantages, including its relatively high cost and prolonged anaesthesia time. Moreover, normal MRI findings cannot fully exclude the possibility of a thiamine deficiency (Chang et al., 2017). A definitive diagnosis of thiamine deficiency can be obtained by determining blood thiamine levels. A simplified and accurate HPLC method to directly measure the blood TPP level is currently available in several commercial laboratories and the test's price is relatively

low compared with those of general diagnostic imaging services in Korea. This test takes a couple of days and requires EDTA anti-coagulated whole blood sample. TPP in the blood sample can be stable for a week under the refrigerator temperature (4 °C) or deep-freezer temperature (−80 °C). The HPLC method has already been validated in humans and is preferable to assess the thiamine status in clinical practice because of its sensitivity, specificity and precision (Talwar et al., 2000). However, the HPLC method has not been validated in dogs and cats and has rarely been used to confirm thiamine deficiency because of its poor accessibility in veterinary clinical settings (Marks et al., 2011). We used HPLC to reveal low blood thiamine levels and could identify the relevant changes in the levels in response to appropriate thiamine supplementation. Therefore, measuring blood thiamine levels through HPLC appeared to be an essential diagnostic tool, and this test has some advantages in terms of intuitiveness, objectivity, cost and invasiveness. However, only a small number of cases have been subject to this test, which includes the present case, additional large-population studies are required to validate this approach for the diagnosis of thiamine deficiency in dogs and cats.

TPP is an essential cofactor for the enzyme pyruvate dehydrogenase, which facilitates the conversion of pyruvate to acetyl CoA. This reaction does not occur in the absence of TPP, and pyruvate is converted into lactate (Markovich et al., 2013). Consequently, the blood lactate levels can be used as an indirect method to assess thiamine status. Contrary to human medicine (Amrein, Ribitsch, Otto, Worm, & Stauber, 2011), blood lactate levels in dogs and cats have not been linked with blood thiamine status. Here, we identified that blood lactate levels are well correlated with blood thiamine levels measured by HPLC. Although lactate levels can be impacted by various physiologic factors and diseases, it must be a useful aid in the diagnosis when combined with other clinical clues due to its greater access convenience for clinicians.

In summary, this report describes the diagnosis and management of thiamine deficiency in a dog. Thiamine deficiency causes metabolic encephalopathy that can be reversed with early diagnosis and appropriate treatment. Obtaining a comprehensive dietary history is extremely important in suspected cases of thiamine deficiency or in sick animals and urges prompt intervention for nutrition. Measuring blood TPP concentration by HPLC is the most reliable and intuitive method to confirm the diagnosis of thiamine deficiency. MR imaging and blood lactate levels can also provide clinically meaningful insights into the thiamine status. Clinicians should be aware that diet composition can markedly influence the thiamine requirements of dogs. Dietary changes based on dietary composition as well as a complete and well-balanced diet might be helpful in the rapid recovery of thiamine deficiency.

#### AUTHOR CONTRIBUTION

**Joong-Hyun Song:** Conceptualization; Data curation; Methodology; Resources; Software; Writing-original draft. **Dong In Jung:** Formal analysis; Funding acquisition; Project administration; Supervision; Writing-review & editing.

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