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CASE REPORT

Serial Magnetic Resonance Imaging and Long-Term Medical Management of Intracranial Arachnoid Cyst in a Dog

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A B S T R A C T

A 2-year-old, intact female Pekingese was presented with a 2-month history of progressive tonic-clonic seizures and left side head tilt. The dog was diagnosed as intracranial arachnoid cyst (IAC) that was located in the quadrigeminal cisterna based on the physical, neurological examinations and magnetic resonance imaging (MRI) of the brain. Medical management using corticosteroids, diuretics, and anticonvulsants was initiated and long term follow-up (6 years) was made. Serial MRI demonstrated elevated occipital lobe compression rate (8%) during the follow-up periods. This case report first describes long term follow-up of the clinical, diagnostic and therapeutic outcome of IAC in a dog.

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INTRODUCTION

Intracranial arachnoid cysts (IAC) are rare congenital space-occupying lesions which constitute 1% in human (Martínez-Lage *et al*., 2011), 0.7% in dogs (Matiasek *et al*., 2007), and only 3 cases in cats (Reed *et al*., 2009). A cerebrospinal fluid (CSF) is accumulated within the splitting or duplicated arachnoid membrane, but the exact etiology is unknown (Wang *et al*., 2013). IAC occurs more commonly in male patients in both human and animals and small breeds, brachycephalic animals may be predisposed (Dewey *et al*., 2009). Diagnosis or recognition of IAC is incidental findings in most cases and clinical significance and therapeutic strategies for the IAC patients are still controversial (Duque *et al*., 2005; Matiasek *et al*., 2007). This report describes the clinical, diagnostic and therapeutic outcome of long-term medical management of IAC using serial magnetic resonance imaging (MRI) features in a dog.

History and clinical examination: A 2-year-old, 6.3-kg, intact female Pekingese was presented with a 2-month history of progressive tonic-clonic seizures. The dog had eye injury 1 year ago and phthisis bulbi on the left eye was observed. Physical examination revealed generalized erythema on the skin and erythema with swelling of both ear canals. Tympanic membrane of both ears was not detected due to the moderated ear discharges. A complete blood count, serum chemistry profile and thyroid functions were normal. The skull radiographs were unremarkable. Neurologic examination revealed left side head tilt. The menace and visual responses were not noted on the left eye, but appropriate on the other side of the eye. Other cranial nerves, spinal reflexes and mental status were normal. Based on the clinical signs, physical and neurological examination intracranial lesion and vestibular dysfunction was suspected. Differential diagnoses included hydrocephalus, caudal occipital malformation, meningoencephalitis, otitis media and interna, and idiopathic vestibular disease.

Diagnosis and treatments: A brain MRI scan using a 0.2 T (E-scan®; ESAOTE, Genova, Italy) was performed. Pre- and post contrast T1- and T2-weighted images (WI) were obtained (Fig. 1). Well-demarcated, fluid filled, cyst-like structures were detected. That was hypointense on T1-WI and hyperintense on T2-WI. The lesion was not enhanced on T1-WI after intravenous administration of gadolinium (Omniscan; Amersham Health, USA; 0.1 mmol/kg, IV). Fluid attenuation inversion recovery (FLAIR) images were not obtained at this time. IAC located in the quadrigeminal cisterna and associated with 3rd ventricle was revealed. The occipital lobe of the cerebrum was compressed rostrally. The degree of parenchymal compression of the occipital lobes was calculated from the MR images using the method previously described

(Matiasek *et al*., 2007) (Fig. 2A). The calculated compression percentage of the occipital lobes in this dog was 22%. Other than IAC, caudal occipital malformation and compression of the cerebellum was also noted. Tympanic bullae and ear canal were normal appearance at both ears. CSF analysis was normal and RT-PCR for canine distemper virus and *Toxoplasma gondii* were all negative. The dog was diagnosed as IAC and caudal occipital malformation.

Treatment was initiated with a combination of prednisolone (Solondo®; Yuhan Medica, Seoul, Korea) (1mg/kg, twice daily, orally), phenobarbital (Phenobarbital®; Myung-In Pharm, Seoul, Korea) (3mg/kg, twice daily, orally), and furosemide (Lasix®; HanDok, Seoul, Korea) (1mg/kg, twice daily, orally). During two months after the initial treatment, prednisolone dose was gradually tapered off. The clinical sign was successfully controlled for the first year, however the seizure was recurred about one year later. Potassium bromide (KBr; Sigma-Aldrich Korea Ltd., Kyunggi-do, Korea) (35mg/kg, once daily, orally) was added to the treatment. The episode of seizure was disappeared at that time, but it was recurred 4 months later. Prednisolone was reapplied (1mg/kg, twice daily, orally) and zonisamide (Excetran®; Dong-A Pharm, Seoul, Korea) (10mg/kg, twice daily, orally) was added. Intermittent episode of seizure (less than once per month) was observed but it was well controlled with the current medication. Prednisolone dosage was increased when the seizure frequency was elevated and then tapered depend on dog's clinical signs.

The brain MRI was retaken 6 years after first presentation using a 3T (Philips, Achieva, Best, The Netherlands) (Fig. 3). The typical appearance of IAC on MRI was obtained on pre- and post contrast T1-WI and T2-WI. Complete fluid signal suppression on FLAIR images was also obtained (Fig. 3G). Using the higher magnetic field MR systems showed the better illustration of the lesions. The calculated compression of the occipital lobes was 30%, which was elevated results when compared to that 6 years ago (Fig. 2B).

The dog underwent 6-year follow-up examinations after first presentation. The compression percentage based on the MRI has increased for 6 years and the seizure frequency was also elevated. Several anticonvulsant drugs were added for controlling the dog's seizure during 6-year follow-up periods. Even though intermittent seizure was detected, the dog was well tolerated to the current medications without further complications.

DISCUSSION

IAC in animals are relatively rare and the occurrence is increasing due to the widely use of magnetic resonance imaging (MRI) and computed tomographic (CT) brain imaging (Duque *et al*., 2005). In humans, 50 to 60% of IACs have been in middle cranial fossa, 10% of IACs in suprasellar cistern and posterior fossa, and other various locations have been reported (Wang *et al*., 2013). However, all reported animal IACs were found in the caudal fossa, typically in quadrigeminal cistern (Lowrie *et al*., 2009). Seizure and headache are the common symptoms in humans (Martínez-Lage et *al*., 2011) and seizure and vestibular dysfunction are the common symptoms in dogs (Dewey *et al*., 2009). Clinical signs and prognosis is depending on the IAC location and size (Helland *et al*., 2010). IAC may be identified as incidental findings in human and animals (Wang *et al*., 2013) and

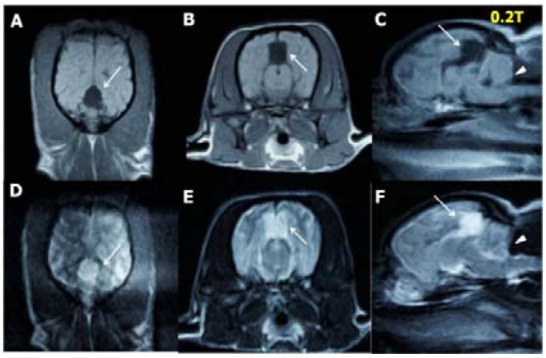

Fig. 1: MR images demonstrating an IAC in a dog. Dorsal (A, D), transaxial (B, E), and midsagittla (C, F) T1 (A-C) and T2 (D-F) weighted images demonstrated a fluid-filled, cystic structure (white arrow) and caudal occipital malformation (arrow head). The lesion was hypointense on T1-WI and hyperintense on T2-WI. IAC was located in the quadrigeminal cisterna and associated with 3rd ventricle was revealed. The occipital lobe of the cerebrum was compressed rostrally.

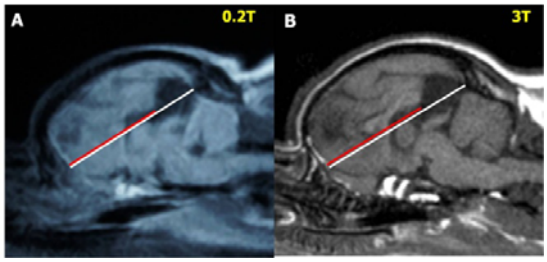

Fig. 2: Midsagittal T1-weighted MRI scan by using 0.2T (A) and 3T (B) in a dog. A parenchymal compression of the forebrain was calculated on midsagittla T1-weighted images. The expected longitudinal dimension of the forebrain (white line) and the actual longitudinal dimension of the forebrain (red line) were calculated according to the Matiasek's methods. The calculated compression of the occipital lobe was 22% at first (A) and it was progressed to 30% later (B). (A) Image was taken when the dog was first presented by using 0.2T and (B) image was taken 6 years after the first presentation by using 3T MIR.

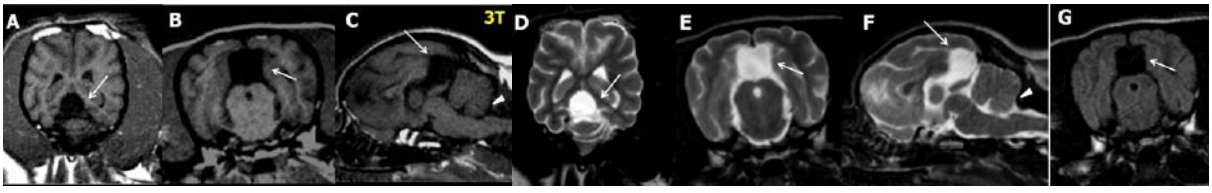

Fig. 3: MR images demonstrating an IAC in a dog 6 years after the first presentation. Dorsal (A, D), transaxial (B, E), and midsagittla (C, F) T1 (A-C) and T2 (D-F) weighted images demonstrated a fluid-filled, cystic structure (white arrow) and caudal occipital malformation (arrow head). The lesion was hypointense on T1-WI and hyperintense on T2-WI. Complete fluid signal suppression on FLAIR image (G) was presented. The location oand size of the IAC was similar during 6 years follow-up periods and using the higher magnetic field MR systems showed the better illustration of the lesions.

careful evaluation of the correlation of clinical symptoms with IAC is needed. Matiasek *et al.* (2007) classified the IAC patients according to the compression of the occipital lobe and/ or cerebellum. Clinical IAC dogs had 22% cerebellar compression (range 6 to 46%), 17% occipital lobe compression ranged from 12 to 23% and dogs with more than 14% occipital lobe compression had high prevalence of clinical signs including seizure.

In our case, relatively young, brachycephalic dog had progressive seizure and vestibular dysfunction. MRI features of the dog revealed comparatively large IAC at quadrigeminal cistern and caudal occipital malformation. The compression of the brain parenchyma was calculated from the MRI scan according to a previous method and occipital lobe was compressed approximately 22% when first diagnosed and which was increased to 30% during 6-year medication periods. Other differential diagnosis could be ruled out based on the serial MRI features and CSF analysis.

Treatment of the incidentally founded IAC is controversial if it is not the primary cause of the disease. If the IAC is main cause of the clinical signs and the compression rate of the occipital lobe is more than 14% then medical and surgical therapy is considered (Martínez-Lage *et al*., 2011). Although some successful surgical management outcomes were reported in animals (Dewey *et al*., 2007), long-term prognosis based on the clinical signs and neuroimaging findings are limited in veterinary fields. Medical treatments using corticosteroids, diuretics, and anticonvulsants were started in this dog for reducing the brain edema and controlling the seizure. During the 6 years of the follow-up periods, the IAC is enlarged, occipital lobe compression rate is elevated to 8% and seizure activity is increased. To control the symptoms of this dog, adjustment of corticosteroids dosage and triple-anticonvulsants were used and the dog is still healthy. Usually, medical management of the IAC was considered temporally, however long-term management information was unknown. Long-term follow-up of this disease would be useful to establish better management strategy because it could be helpful to understand the disease process.

Conclusion: This case report demonstrates the long-term medical management of the IAC in a dog based on the clinical signs and the neuroimaging findings.

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