7.0-Tesla Tesla Magnetic Resonance Imaging of Granulomatous Meningoencephalitis in a Maltese Dog: A Comparison with 0.2 and 1.5-Tesla

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(Received 14 April 2009/Accepted 22 July 2009)

ABSTRACT. A 6-year-old, intact female Maltese dog was presented with generalized seizures. Based on the neurological and physical examinations, intracranial lesion was suspected. Magnetic resonance imaging (MRI) of the brain was performed at three different magnetic field strengths (0.2, 1.5 and 7.0 T). Diffuse hypo- and hyperintense lesions involving the left caudate nucleus and internal capsule to the cranial diencephalon were identified on T2-weighted images. The detailed anatomical locations, the inflammatory and hemorrhagic field strengths (0.2, 1.5 and 7.0 T). Diffuse hypo- and hyperintense lesions involving the left caudate nucleus and internal capsule were more apparent at 7.0 T. Histopathologically, granulomatous meningoencephalitis (GME) was diagnosed. This is the first case describing histopathologically confirmed GME lesions using 0.2, 1.5 and 7.0 T clinical MR scanner.

KEY WORDS: 7.0 T, canine, GME, MRI.

Granulomatous meningoencephalitis (GME) is a non-suppurative, idiopathic inflammatory disease of the canine central nervous system (CNS) [2, 5, 7]. GME usually has an acute onset and progressive course, and if left untreated, it is usually fatal [10, 12, 13]. The clinical signs with neurological dysfunction are quite variable, which reflect the location and severity of the CNS lesions [2, 12]. The definitive diagnosis of GME is made only by biopsy or at postmortem evaluation. However, a presumptive diagnosis can be made on the basis of characteristic signs and suggestive clinical findings as well as comparable results on the cerebrospinal fluid (CSF) analysis [1, 14]. More recently, magnetic resonance imaging (MRI) and computed tomography (CT) have been shown to be useful diagnostic tools for the detection of GME [3, 4, 8, 9, 11, 14]. Here we report the first case of GME lesions with hemorrhage using 7.0 T clinical MR scanner.

A 6-year-old, 3.5-kg, intact female Maltese dog was presented with a 10-day history of progressive seizures. The dog also showed progression of seizure severity about 3 days prior to presentation. At presentation, the dog had cluster, tonic-clonic seizures and was laterally recumbent. Physical examination revealed a confused mental status, delayed pupillary light response, moderate dyspnea and hypothermia. The dog was treated with 4 mg/kg phenobarbital sodium (Luminal; Dai Han Pharm. Co., Ltd., Seoul, Korea) because of recurrent convulsions without complete recovery between seizures.

A complete blood count revealed a regenerative anemia (hematocrit 33%, reference range 37 to 55%; reticulocytes 278,000/μl, reference range > 60,000/μl). A serum chemistry profile showed an elevated lactate dehydrogenase (129 IU/l, reference range 20 to 109 IU/l), creatine kinase (335 IU/l, reference range 49 to 166 IU/l) and blood glucose (184 mg/dl, reference range 75 to 128 mg/dl). Thoracic radiographs were unremarkable.

Based on the clinical signs, physical and neurological examination, as well as the pathological findings, intracranial lesion was suspected. Thus, we performed a brain MRI scan using a 0.2 T (E-scan®; ESAOTE, Genova, Italy), 1.5 T (Magnetom Avanto; Siemens AG, Berlin, Germany) and 7.0 T (Magnetom 7.0 T; Siemens Healthcare, Berlin, Germany). Pre- and post contrast T1- and T2-weighted images (WI) were obtained at 0.2 and 1.5 T. Only T2-WI were obtained at 7.0 T (T1-WI were not available). Transverse T2-WI revealed a sharply delineated hypointense lesion under the left periventricular area and surrounding hyperintense lesions in the left caudate nucleus and internal capsule at 3 magnetic field strengths (Fig. 1A-C). Especially periventricular hypointense lesion was more apparent at 7.0 T compared to 0.2 and 1.5 T. These lesions were not enhanced on T1-WI after intravenous administration of gadolinium (Omniscan®; GE Healthcare, NJ, U.S.A.; 0.1 mmol/kg, IV). Blood vessels in the left caudate nucleus of the affected side were more evident than on the opposite side at 7.0 T (Fig. 1A). Asymmetrical ventriculomegaly and compressed brain parenchyma were also observed. On the left paramedian sagittal T2-WI, diffuse hyperintensities in the
area of the caudate nucleus and internal capsule to the cranial diencephalon were observed (Fig. 1D-F). The distribution and anatomical margins of the lesions were more apparent at 7.0 T. Ventriculomegaly suggested an acquired obstructive hydrocephalus secondary to the cerebral lesions. On CSF analysis, elevated protein concentration (60 mg/dl, reference range < 30 mg/dl) and mononuclear pleocytosis (8 cells/µl, reference range 0 to 5 cells/µl) were noted. RT-PCR for canine distemper virus and Toxoplasma gondii were all negative in serum and CSF. Thus nonsuppurative intracranial inflammation was strongly suspected.

Treatment was initiated with a combination of 1 mg/kg prednisolone (Solondo; Yuhan Medica, Seoul, Korea) twice daily and 5 mg/kg cyclosporine (Neoral; Norvartis Korea, Seoul, Korea) daily with 3 mg/kg phenobarbital (Phenobarbital; New Gen Pharm, Seoul, Korea) twice a day per oral. Despite the treatment, the symptoms gradually worsened. Three days after presentation, cytosine arabinoside (50 mg/m², Cytosar-u; Pfizer Pharmaceuticals Korea Ltd., Seoul, Korea) was intrathecally administered, followed by twice daily subcutaneous injections for two consecutive days. The clinical signs were gradually controlled without severe complications and seizure episode was decreased once daily. Six days after the initial treatment, prednisolone dose was reduced to 0.75 mg/kg. There was no more seizure episode and the respiratory distress syndrome was improved. The dog was bright, alert and responsive, but abnormal involuntary movements (nonambulatory tetraparesis) continued. Nine days after presentation, the dog was euthanized with the owner’s consent. A necropsy was performed.

Noninfectious inflammatory intracranial diseases in dogs are challenging to diagnose and treat [6]. Use of MRI is integral to diagnosing intracranial disease [3, 4, 9, 8]. The ability of MRI to differentiate similar soft tissues differs according to the field strength. In this case, we used 0.2, 1.5 T and 7.0 T MRI to compare the same lesions. The inflammatory lesions of this case were mainly distributed in the...

Fig. 1. MR images of brain of a dog with acute onset of seizure. (A-C) Transverse T2-weighted images at the level of the rostral caudate nucleus. Hypointense lesions (open arrow) are sharply delineated in the left periventricular area, representing an intracerebral hemorrhage. Hyperintense lesions (thin arrows) surrounding the areas of hemorrhage are noted in the left caudate nucleus and internal capsule. This finding is more apparent with the 7.0 T MR images than the 1.5 and 0.2 T MR images. Asymmetrical ventriculomegaly and compressed brain parenchyma are also observed. (D-F) Left paramedian sagittal T2-weighted MR images of the same dog. Diffuse hyperintensity involving from caudate nucleus and internal capsule extending to the cranial diencephalon is observed (arrows). Compared with the transverse images, more hypointensity is evident in the periventricular area with the 7.0 T MR images than with the 1.5 T and 0.2 T MR images (open arrow).
cerebrum, especially caudate nucleus and internal capsule regions extending to the cranial diencephalon. In addition, the intracerebral hemorrhage and blood vessels were more evident at 7.0 T compared to 0.2 and 1.5 T. Based on these findings, the detailed anatomical locations, the inflammatory and hemorrhagic changes of the lesions could be detected more apparently at 7.0 T. Thus, higher tesla MRI may have better resolution for small and subtle lesions.

The discolored area on necropsy was consistent with the sharply delineated hypointense MRI findings. On histological examination, the lesion consisted of hemosiderin-laden macrophage and lymphocyte infiltration. Thus, the hypointense lesions on MRI represented intracerebral hemorrhages. Around the hemorrhagic lesion, there was inflammation with severe infiltration of perivascular, mononuclear cells.

From this case report, it seemed that a higher tesla MRI can facilitate the diagnosis of small and subtle intracranial inflammatory lesions including blood vessel diseases of the brain in dogs and cats. For this, further case studies of using 7.0 T MRI examination in many GME patients and compare the images with those of 0.2 and 1.5 T MRI examination in same patients will be needed.

In conclusion, this case report demonstrates that a 7.0 T clinical MR scanner may be superior to 0.2 and 1.5 T that in imaging of intracranial inflammatory lesions.

ACKNOWLEDGMENT. This research was supported by the Korea Research Foundation Grant funded by the Korean Government (MOEHRD, Basic Research Promotion Fund) (KRF-2008–314-E00246).

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