Surgery

Traumatic Intracerebral Hematoma in a Dog: MR Images and Clinical Findings

Masato KITAGAWA1), Midori OKADA3), Kiichi KANAYAMA2) and Takeo SAKAI1)

1)Departments of Preventive Veterinary Medicine and Animal Health and 2)Veterinary Physiology, Nihon University School of Veterinary Medicine, 1866 Kameino, Fujisawa, Kanagawa 252–8510 and 3)Pet Clinic ANIHOS, 1–14–11 Minami-Tokiwadai, Itabashi, Tokyo 174–0072, Japan

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ABSTRACT. A nine-year-old, male, mongrel dog was unsteady after falling down a set of stairs. The dog exhibited a mildly abnormal gait 2 days after injury, and was not able to stand, with spasticity of the right limbs, 4 days after injury. MR imaging revealed a clearly demarcated mass on the top of the left lateral ventricle that showed mild hyperintensity on T1-weighted images and hyperintensity on T2-weighted images. The authors diagnosed the dog as having a traumatic intracerebral hematoma. This type of case, in which the clinical signs deteriorated due to edema associated with hematoma, is extremely rare.

KEY WORDS: canine, cerebral hematoma, MRI.

The causes of intracerebral hemorrhage include infectious diseases, toxins, neoplasia, disseminated intravascular coagulopathy, idiopathic thrombocytopenia purpura, vascular anomalies, and peripartum asphyxia. The most common cause of brain hemorrhage is trauma [9]. Subdural, epidural, subarachnoid, and/or intracerebral hematoma occur due to head injuries, and it is known that a characteristic clinical course occurs for each type of hematoma in humans [5]. It is also known that changes in magnetic resonance (MR) images occur with elapsed time [20]. In dogs, subdural hematoma [7, 10, 19], hemorrhage in the arachnoid cyst [17], intracerebral hemorrhage with vascular anomaly [15], and MR imaging of intracerebral hematoma in an experimental model [18] have been reported, but traumatic intracerebral hematoma has not. We encountered a dog diagnosed as having traumatic intracerebral hematoma, and report here in the clinical course and MR images.

The case was a nine-year-old, male, mongrel dog that was unsteady after falling down a set of stairs after being surprised by thunder. The dog exhibited a mildly abnormal gait 2 days after injury, and was not able to stand, with spasticity of the right limbs, 4 days after injury. The dog was referred to Nihon University Animal Hospital 5 days after injury for detailed examination. Initial examination revealed that its body weight was 15.9 kg, temperature was 38.7°C, heart rate was 64 beats/min, and respiration rate was 28/min. Neurological examination showed a deficit of the postural reaction of the right limbs, depression of the postural reaction of the left limbs, bilateral miosis and clouding of consciousness. The dog’s blood pressure values were 149 mmHg for the systolic blood pressure (SBP), 118 mmHg for the mean arterial pressure (MAP), and 98 mmHg for the diastolic blood pressure (DBP) (reference values: SBP 147 ± 28 mmHg, MAP 83 ± 15 mmHg, DBP 104 ± 17 mmHg) [3]. In the dog’s blood coagulation test, the prothrombin time (PT) was 8.4 sec, the activation partial thromboplastin time (APTT) was 15 sec, and the fibrinogen (Fib) was 520 mg/dl (reference values: PT 6–8.5 sec, APT 12–18 sec, Fib 200–400 mg/dl), suggesting that the patient had no coagulopathy.

The authors performed X-RAY examination, MR imaging (FlexArt, Toshiba, Tokyo, 0.5T), and cerebrospinal fluid examination. No abnormalities, such as fractures, were revealed on radiographical examination of the head (Fig. 1). MR imaging revealed a clearly demarcated mass on the top of the left lateral ventricle that showed mild hyperintensity on T1-weighted images and hyperintensity on T2-weighted images. The authors diagnosed the dog as having a traumatic intracerebral hematoma. This type of case, in which the clinical signs deteriorated due to edema associated with hematoma, is extremely rare.
Contrast medium (gadoteridol, 0.1 mmol/kg; ProHance®, BRACCO International) (Fig. 2c). A region on the surface of the left frontal lobe showed mild hyperintensity on T1-weighted images and hyperintensity on T2-weighted images. A peripheral region of the mass showed hypointensity on T1-weighted images, and hyperintensity on T2-weighted images (Figs. 2a, b, c). CSF analysis revealed a bloody xanthochromic color with a cell count of 4/mm³, a protein concentration of 22 mg/dl, and a negative Pandy’s test.

Methylprednisolone sodium succinate (5 mg/kg body weight, IV) (Solu-Medrol®, Pharmacia & Upjohn) was administered for 3 days and furosemide (0.7 mg/kg body weight, IV) (Lasix®, Hoechst Marion Roussel) for 7 days to prevent edema and to raise intracranial pressure. To prevent infection, piperacillin sodium (PENTOCILLIN®, Piperacillin Sodium, Sankyo, Tokyo, Japan) was administered for 14 days. Compulsive feeding was performed because the dog could not eat.

On the 7th day after injury, the dog was able to stand, but showed disorientation. Eleven days after injury, the disorientation improved. However, the outline of the mass became unclear and the degree of cerebral compression was unchanged on the MRI (Fig. 3a). The dog still exhibited mild right hemiparesis 14 days after injury, but as its general condition was improving, the dog was discharged from our hospital.

On the 30th day after injury, a region, which located on the surface of the left frontal lobe, showed isointensity on T1-weighted images and was enhanced on post-contrast T1-weighted images (Figs. 3b and 4a, b). No mass was observed, and no significant neurological signs were noted.

MRI signals for intracranial hematoma are affected by the amount of time from ictus. For example, a hematoma shows...
mild hyperintensity on T1-weighted images and hyperintensity on T2-weighted images between 12 to 24 hr after injury. The same MR images are also seen 5 or 6 days after the injury [20]. Ring-like enhancement of the periphery of the hematoma with contrast medium was seen from 5–6 days after injury [11]. The mass of this dog was suggestive of hematoma between 5–6 days after injury based on the MR images, so it was suspected that the hematoma occurred at time of injury. The MR images of the periphery of the mass of this dog showed hypointensity on T1-weighted images and hyperintensity on T2-weighted images, suggestive of edema formation. A hemorrhage was suggested because a region showing mild hyperintensity on T1-weighted images
in the left frontal lobe was spread across the surface. The periphery of the region showed hypointensity on T1-weighted images and hyperintensity on T2-weighted images, so edema was also diagnosed. Brain contusions may affect the crest of the gyri superficially or involve the entire depth. There is usually some bloody extravasation, which may spread to the surface, resulting in a subpial or subarachnoid hemorrhage [14]. Therefore, we diagnosed the lesion in the left frontal lobe as a contusion. On the 30th day after injury, the region, which located on the surface of the left frontal lobe, was enhanced on the post-contrast T1 weighted images. Chemical arachnoiditis occurs because of blood flowing into the subarachnoid during brain surgery or trauma, and subsequently the meninges become thick and vascularity increases. Consequently, the meninges that are associated with arachnoiditis are enhanced on post-contrast T1-weighted images [2]. It was suspected that the meninges of this dog were enhanced on the post-contrast T1-weighted image because chemical arachnoiditis occurred with traumatic brain hemorrhage. It has been reported that vascular anomalies appear honeycomb-like due to a flow void effect, which occurs by high-velocity signal loss on MRI [10]. We ruled out vascular anomalies because these findings were not detected.

This dog showed hemiparesis, inability to stand, and disorder of consciousness from 2–3 days after injury. In human medicine, it has been reported that delayed deterioration of clinical signs after head injury is caused by enlargement of hematoma, delayed intracerebral hematoma, or deterioration of edema [4, 6, 8, 12, 13, 16]. Delayed intracerebral hematoma following head injury is characterized by the appearance of focal neurological signs or depression of the level of consciousness developing the day after trauma [6, 16]. Because it was suspected that the hematoma of this dog occurred at the time of injury, we excluded delayed intracerebral hematoma. Enlargement of a hematoma after head injury usually occurs in patients with a predisposition for coagulopathy or chronic hypertension [4, 13]. We excluded enlargement of the hematoma because this dog had no predisposition for coagulopathy or chronic hypertension. It has been reported that activation of the coagulation cascade and thrombin release predominantly contributes to the perilesional edema in hematoma [12]. Furthermore, it has been described that edema after head injuries begin early, at 1–6 hr, are maximal at 48 hr, and are resorbed within 5–7 days [1]. Also, edema-related neurological deterioration is often observed 1 to 4 days after hemorrhage [12]. As the developmental time of edema after injury, which has been reported in the past [1, 12], was coincident with the time of deterioration of the clinical signs for this dog, we suspected that development of edema deteriorated the clinical signs for this dog.

This type of case, in which the clinical signs deteriorated due to edema associated with hematoma, is extremely rare.

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REFERENCES


