

Subdural Hematoma of the Brainstem in a Dog: Magnetic Resonance Findings and Treatment

An 8-year-old, spayed female Dalmatian with a history of seizures was evaluated for cervical pain and bilateral scleral hemorrhages. Diagnostic evaluations revealed a mass displacing the ventral brainstem on magnetic resonance imaging (MRI). The mass was surgically removed and histologically confirmed to be a hematoma. The dog's neurological signs resolved completely after surgery. Although extradural, subdural, subarachnoid, and intraparenchymal hemorrhages have been reported in dogs and cats, this is the first known report of a subdural hematoma of the ventral brainstem in a dog. On the basis of the history and the appearance of the subdural hematoma on MRI, a traumatic event during the seizure episodes was considered the most likely cause of the subdural hematoma in this case. **J Am Anim Hosp Assoc 2005;41:400-405.**

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Introduction

A subdural hematoma is an accumulation of blood between the dura mater and the arachnoid mater and is thought to arise from the rupture of small veins traversing the subdural space.¹⁻⁴ Subdural hematomas in the dog may occur as focal intradural mass lesions or as diffuse lesions over the cerebral cortex, sometimes associated with massive accumulation of blood.^{5,6} Extradural, subdural, subarachnoid, and intraparenchymal hemorrhages have been reported in dogs and cats following head injuries; but to the authors' knowledge, a subdural hematoma of the ventral brainstem has not been reported in the dog.^{7,8} The purpose of this report is to describe the magnetic resonance imaging (MRI) findings and surgical removal of a ventral brainstem subdural hematoma in dog.

Case Report

A 34-kg, 8-year-old, spayed female Dalmatian was referred to the Veterinary Medical Teaching Hospital at the University of Wisconsin (VMTH-UW) for evaluation of cervical pain. The dog had a 2-year history of seizures, beginning at approximately 6 years of age. The seizures were generalized, with collapsing of the hind limbs and forelimb rigidity. The episodes were short in duration; there was no loss of consciousness; and the postictal phase lasted approximately 20 minutes. The owner reported three seizures during the last 2 years, with an interictal period of about 6 months.

Six days before presentation, the dog had a cluster of grand mal seizures and was brought to an emergency clinic where the seizures were controlled with diazepam intravenously (IV). At the emergency clinic, initial laboratory abnormalities included a packed cell volume (PCV) of 57% (reference range 35% to 55%) and total protein (TP) of 8.0 g/dL (reference range 5.2 to 7.8 g/dL), an elevated serum alkaline phosphatase (SAP, 1073 U/L; reference range 9 to 140 U/L), and mild hyperbilirubinemia (0.6 mg/dL; reference range 0.0 to 0.4 mg/dL). The dog had a previous history of elevated SAP. Hepatopathy as a cause of the seizures was ruled out on the basis of a normal albumin (3.2 g/dL; reference range 2.5 to 3.8 g/dL), normal alanine aminotransferase (ALT, 37 U/L; reference range

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15 to 84 U/L), and normal fasting ($12.0 \mu\text{M/L}$; reference range $<15 \mu\text{M/L}$) and postprandial ($12.9 \mu\text{M/L}$; reference range $<25 \mu\text{M/L}$) bile acid concentrations. Systolic blood pressure, using an ultrasound flow detector^a on the palmar artery, was measured several times; the average was 164 mm Hg (normal <180 mm Hg).⁹ Cerebrospinal fluid (CSF), harvested from the cerebello-pontinomedullary cisterna, was bloody; therefore, CSF analysis was not diagnostic.

After 2 days at the emergency clinic, serum bilirubin had mildly improved (0.5 mg/dL), but the SAP had increased (1176 U/L). The following day, the dog was lethargic and uncoordinated and had scleral hemorrhages and possible neck pain. Further tests were performed by the regular veterinarian. A coagulation profile was normal, with a platelet count of $420,000/\mu\text{L}$ (reference range $160,000$ to $525,000/\mu\text{L}$), a prothrombin time (PT) of 6.3 seconds (reference range 8.0 to 10.3 seconds), and an activated partial thromboplastin time (PTT) of 10.2 seconds (reference range 10.5 to 16.5 seconds). Enalapril^b (0.5 mg/kg per os [PO] *q* 24 hours) to decrease a presumptive hypertension (systemic blood pressure was not measured at this time) and cephalixin^c (15 mg/kg PO *q* 8 hours) to treat a suspected meningococcal meningitis secondary to the previous spinal tap were prescribed. The dog's neurological status continued to deteriorate; she became reluctant to stand and was extremely painful on cervical manipulation. Mannitol^d (1 gm/kg IV) and furosemide^e (0.7 mg/kg IV) were administered to decrease a suspected elevated intracranial pressure, and vitamin K^f (20 mg) was given subcutaneously to treat a possible occult coagulopathy. The dog showed mild improvement and was referred the following day for further assessment.

At the time of presentation to VMTH-UW, abnormalities on physical examination included mild dehydration; bilateral scleral hemorrhages; a grade I-II/VI, left-sided, focal systolic murmur; pain upon opening the mouth and extension of the neck; and panting. Neurological examination identified cervical pain, increased muscle tone in all four limbs, and crossed extensor reflex in the forelimbs. The rest of the neurological examination was unremarkable.

The history of seizures was typical of a forebrain (cerebrum and diencephalon) lesion, while the neck pain and the upper motor neuron signs in all four legs were suggestive of either a cervical myelopathy or intracranial disease. Differential diagnoses for seizures included idiopathic epilepsy, developmental abnormalities (e.g., hydrocephalus, quadrigeminal cyst), a previous vascular accident (e.g., brain infarction, hemorrhage), metabolic diseases, and neoplasia.¹⁰ Inflammatory diseases, infectious diseases, toxins, and trauma were considered less likely.¹⁰ Differential diagnoses for cervical myelopathy included infectious or inflammatory diseases (e.g., meningomyelitis, discospondylitis), intervertebral disk extrusion, neoplasia, and trauma.¹¹ Differential diagnoses for the bilateral scleral hemorrhages included coagulopathies, vasculitis, or trauma.¹²

Diagnostic evaluations were undertaken, and a complete blood count (CBC) showed mild lymphopenia ($860/\mu\text{L}$; reference range 1000 to $4800/\mu\text{L}$), monocytosis ($1630/\mu\text{L}$;

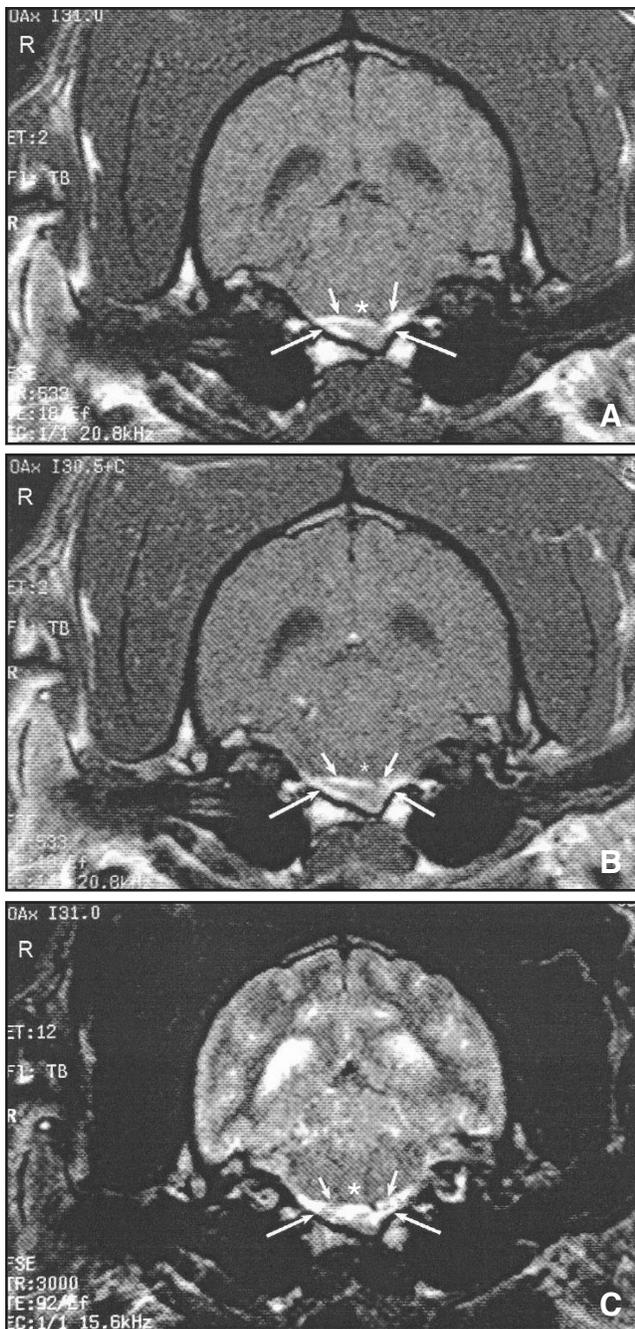
reference range 150 to $1350/\mu\text{L}$), and elevated SAP (779 U/L). Urinalysis collected by manual bladder expression showed proteinuria (3+), a moderate amount of triple phosphate crystals, and a few red blood cells [RBCs] and white blood cells (1 to 5 per high-power field; reference range 0 to 5), with no growth on bacterial culture. A coagulation profile was normal (PT of 6.2 seconds, control 7.5 seconds; PTT of 11.5 seconds, control 9.6 seconds). Thoracic and cervical radiographs were normal. Blood pressure using an oscillometer^g was measured five consecutive times. Systolic pressure was 189 mm Hg; diastolic pressure was 60 mm Hg; and mean arterial pressure was 118 mm Hg. A grade I/VI systolic murmur heard loudest at the left apex of the heart was consistent with mild mitral regurgitation. On ophthalmic examination, bilateral scleral hemorrhages were the only abnormalities noted. Because of the normal coagulation profile, the tentative diagnosis for the bilateral scleral hemorrhages was vasculitis, platelets dysfunction, systemic hypertension, or trauma.

Causes of vasculitis considered in this animal included toxic, immune-mediated, infectious, inflammatory, and neoplastic disorders.¹³ Possible causes of platelet dysfunction included inherited thrombopathies (i.e., von Willebrand's disease) and acquired thrombopathies (drug induced or secondary to diseases).¹⁴

Because of deteriorating neurological signs, further diagnostic evaluation of the possible hypertension and vasculitis was postponed. Platelet dysfunction tests (e.g., buccal mucosal bleeding time and von Willebrand's diseases) were not performed. Amlodipine^h (0.1 mg/kg PO *q* 24 hours) was administered to treat any hypertension. Butorphanolⁱ (0.1 mg/kg per hour IV as continuous-rate infusion) and oxymorphone^j (0.2 mg/kg intramuscularly [IM] *q* 4 hours) were given to control the severe neck pain.

Magnetic resonance imaging was performed with a 1.0 Tesla magnet^k using the following pulse sequences: **1.** transverse pre- and postcontrast T1-weighted (TR=533 msec, TE=18, 4.0-mm contiguous slices, 3 number of excitations [NEX], $256\text{H} \times 192\text{V}$ matrix); **2.** sagittal postcontrast T1-weighted (TR=350 msec, TE=20, 4.0-mm contiguous slices, 4 NEX, $256\text{H} \times 192\text{V}$ matrix); **3.** transverse T2-weighted (TR=92 msec, TE=3000, 4.0-mm contiguous slices, 3 NEX, $256\text{H} \times 192\text{V}$ matrix); **4.** sagittal T2-weighted (TR=3800 msec, TE=100, 4.0-mm contiguous slices, 4 NEX, $256\text{H} \times 192\text{V}$ matrix); and **5.** transverse proton-density (TR=2300 msec, TE=23, 4.0-mm contiguous slices, 3 NEX, $256\text{H} \times 192\text{V}$ matrix).

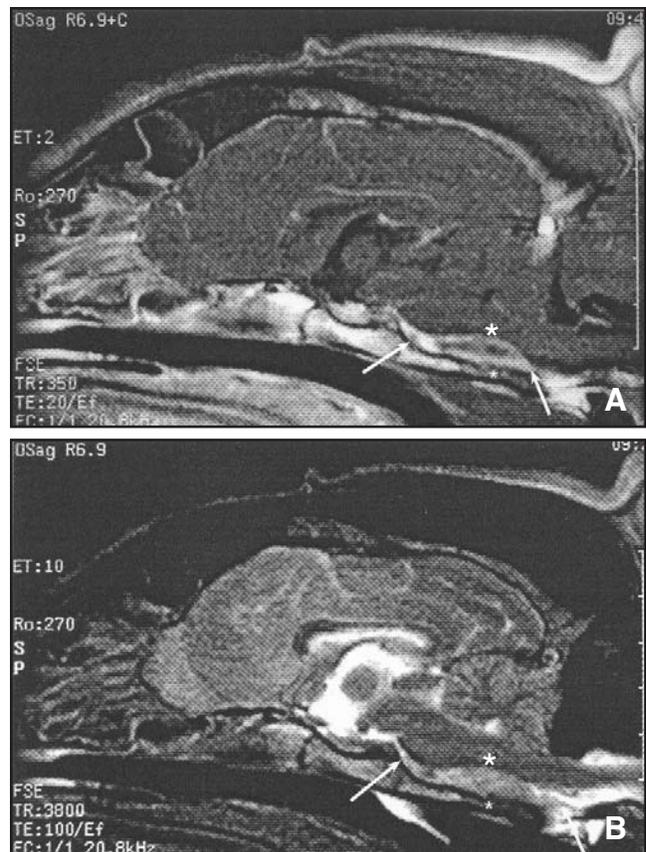
Abnormalities on MRI consisted of a crescent-shaped caudoventral brainstem lesion causing dorsal displacement of the brainstem primarily toward the right side. The lesion had a rim of hyperintensity on T1-weighted images [Figures 1A-1C, 2A, 2B], with an isointense central region. The lesion did not enhance following IV administration of 0.1 mmol/kg of gadopentetate dimeglumine.^l On T2-weighted images, the lesion was hyperintense compared to brain parenchyma, and it was heterogeneous in appearance. Despite the appearance of a caudal brainstem mass effect,



Figures 1A-1C—Transverse (A) precontrast T1-weighted, (B) postcontrast T1-weighted, and (C) T2-weighted images of a subdural hematoma (arrows) on the ventral aspect of the brainstem (asterisk). Right (R) is to the left in all images.

there was no evidence of obstruction to the ventricular system. The MRI abnormalities were suggestive of a subdural hematoma on the ventral aspect of the brainstem.¹⁵⁻¹⁷

Cerebrospinal fluid collected from the cerebello-pontomedullary cisterna was blood-tinged and had a hazy appearance. Fluid analysis revealed a marked neutrophilic pleocytosis with a mild elevation in TP (28.7 mg/dL; reference range <25 mg/dL). The total nucleated cell count was 885 cells/ μ L (reference range <5/ μ L), with 85% neutrophils,



Figures 2A, 2B—Sagittal (A) postcontrast T1-weighted and (B) T2-weighted images of a subdural hematoma (arrows) of the ventral medulla oblongata (large asterisk), at the level of the basioccipital bone (small asterisk).

3% lymphocytes, and 12% macrophages. There were 710/ μ L RBCs (reference range 0/ μ L). The neutrophils were nondegenerate and occasionally hypersegmented. Macrophages were minimally vacuolated and exhibited rare leuko- and erythrophagocytosis. No organisms were seen. These CSF abnormalities were consistent with an infectious disease (e.g., bacterial, fungal, protozoal, and rickettsial), neoplasia (e.g., meningioma), postseizure abnormalities, or any insult causing brain ischemia or necrosis.¹⁸ Results of CSF bacterial culture were negative.

Because of the severity of brainstem displacement and the possibility of the suspected subdural hematoma to expand and compress further the critical brainstem structures, surgical therapy was considered the most effective intervention in this case. Decompressive surgery of the caudal brainstem using a standard ventral approach was performed the next day.¹⁹ Anesthesia was induced with propofol^m (3 mg/kg IV) and midazolamⁿ (0.15 mg/kg IV) and was maintained with isoflurane^o in oxygen. Cephazolin^p (20 mg/kg IV) was given at the time of the induction and every 2 hours during the course of surgery. Carprofen^q was also given preoperatively at 4.4 mg/kg IV. Mannitol (1 gm/kg IV) was given over 20 minutes during the craniotomy.

A high-speed pneumatic drill was used to make a mid-line oval defect in the basioccipital bone overlying the mass. The craniectomy extended caudally to the intercondyloid notch, rostrally to the synchondrosis sphenoccipitalis, and abaxially to a few millimeters before the medial wall of the tympanic bullae. After removing the last layer of basioccipital bone with, most likely, the attached cranial dura mater, a red-brown mass was visible. The mass appeared gelatinous, and it extended along the brainstem. Pieces of the lesion were removed with a lens loop and fine ophthalmic forceps, and they were submitted for cytology and aerobic and anaerobic bacterial culture. After the mass removal, the ventral aspect of the brainstem and the basilar artery were easily visible. The authors assumed that the cranial dura mater was removed during the craniectomy, because it serves as periosteum intracranially, and no structure resembling dura mater was seen after the mass removal. A fat graft was placed over the craniotomy site. The muscle layers, the subcutaneous tissues, and the skin were closed routinely. Fentanyl^r (10 μ /kg per hour) was initiated IV and maintained at a rate of 2 to 5 μ /kg per hour during the following 12 hours. During recovery, the dog had one grand mal seizure that was controlled with a bolus of midazolam (0.5 mg/kg IV).

The morning following surgery, the dog was resting comfortably. Upon neurological examination, the dog had a mild head tilt to the right and a mild sensory ataxia that were attributed to the surgical manipulation.

Cytology of the biopsied lesion showed a single cluster of poorly preserved neutrophils embedded within pale, basophilic, fibrinous material. A small number of lymphocytes and monocytes were scattered in a hemorrhagic background, and heme breakdown products were also visible. The neutrophilic cluster was interpreted as an area of mild inflammation or cells trapped within a fibrinous clot. Histopathological examination of the lesion revealed small fragments of fibrin with admixed hemorrhage and several foci of hemorrhage. These findings were consistent with an organized hematoma. Culture results were negative.

Because of the history, type of seizures, and the negative forebrain findings on MRI, idiopathic epilepsy was considered to be the most likely etiology for the seizures, and phenobarbital^s therapy (loading dose of 15 mg/kg IV) was started and continued at 3 mg/kg PO *q* 12 hours. Carprofen was given for 5 days (2.2 mg/kg PO *q* 12 hours) to control any postoperative inflammation, and amlodipine was continued (0.1 mg/kg PO *q* 24 hours) for suspected hypertension.

Three weeks later, the referring veterinarian reported that systolic blood pressure was 160 mm Hg, serum phenobarbital was 24 μ g/mL (reference range 15 to 40 μ g/mL), and an adrenocorticotropic hormone (ACTH) stimulation test was normal. Mild proteinuria persisted (30 mg/dL), but the urine protein/creatinine ratio (0.449, reference range <1) was within normal limits. On the basis of these results, hyperadrenocorticism and protein-losing renal diseases were considered less likely. An abdominal ultrasound to rule out pheochromocytoma was not performed. Phenobarbital therapy was maintained at the same dosage as before.

Discussion

In the present case, because of the limited surgical exposure and the intimate relationship of dura mater with the basioccipital bone, a clear distinction of hemorrhagic material under the dura and its relationship to the subarachnoid space could not be defined. The term subdural hematoma is used in this report because after removal of the hemorrhagic material, the ventral aspect of the brainstem and the basilar artery were easily visible, and many MRI similarities are described in human subdural hematomas.¹⁵⁻¹⁷

Subdural space is the virtual space located between the inner dural layer and the arachnoid.²⁰ Traumatic laceration of veins crossing this potential space is the usual cause of acute subdural hematoma formation in people.²¹ The time between a traumatic event and the development of clinical signs of intracranial dysfunction is inversely related to the severity of cerebral and vascular damage sustained at the time of injury.² Acute subdural hematomas are often accompanied by cerebral contusion and laceration, and they can be rapidly life threatening.^{2,22} Chronic subdural hematomas are often associated with a minor traumatic episode that may have been forgotten by the time the patient develops clinical signs.² Subacute subdural hematomas have an intermediate pattern of development of clinical signs.² Small subdural hematomas in people usually resolve completely, leaving only a residual membrane that represents the residual capsule.¹⁵ Larger hematomas tend to undergo clot lysis, become encysted, and may subsequently show a tendency to expand.^{2,23}

Several observations have suggested that the genesis of a local hyperfibrinolytic state leads to the development of an expanding hematoma.^{2,21,23} Elevated fluid levels of fibrinogen degradation products have been demonstrated.^{2,21,24} Immature blood vessels in the capsule of the hematoma are poorly formed; these may leak erythrocytes into the hematoma or be more likely to bleed secondary to minimal trauma.^{15,25} This low-grade persistent hemorrhage is perpetuated by the anticoagulant, fibrinolytic environment and is the primary mechanism for hematoma expansion.^{2,21} Daily hemorrhage of up to 10% of existing hematoma volume has been demonstrated.^{2,21} The potential for expansion of the hematoma along with the already large mass effect on the brainstem in this case led to the decision to surgically remove the subdural hematoma.

In people, subdural hematomas are classified into four categories according to the time elapsed between the traumatic incident and the onset of clinical signs: hyperacute, acute, subacute, and chronic.¹⁵ The MRI findings correlate well with the age of a hematoma and the onset of related symptoms.²⁶ Characteristic MRI intensities allow for determination of the age of hemorrhage based on biochemical transformation of oxyhemoglobin (hyperacute stage) to deoxyhemoglobin (acute stage) to methemoglobin (subacute stage) and hemosiderin (chronic stage).^{15,16,27,28} The MRI appearance of intraparenchymal brain hematomas in dogs has been previously reported.^{27,28} To the authors' knowledge, however, the MRI characteristics of an intracranial subdural hematoma in dogs have not been reported.

The MRI characteristics of the mass in this case correlated well with what is described for subdural hematomas in people.^{15,25} In the case reported here, subdural hematoma was considered the primary differential diagnosis because of the characteristic MRI signal intensities, the lack of contrast enhancement, and the convex extramedullary appearance of the mass conforming to the shape of the brainstem.^{15,17} Based on the MRI features in this case, the subdural hematoma was staged between the acute and subacute phases, suggesting that the hematoma occurred approximately 1 week previously. The isointense region within the center of the hematoma on T1-weighted images likely represented intracellular deoxyhemoglobin that had yet to be oxidized to methemoglobin.^{15,16,27,28} The hyperintense rim present on T1-weighted images may have represented the initial conversion of deoxyhemoglobin to methemoglobin, which proceeds centripetally from the periphery and occurs in the subacute phase of subdural hematoma formation.^{15,16} The hyperintense but heterogeneous appearance on T2-weighted images was thought to be caused by a mixture of intracellular deoxyhemoglobin or methemoglobin with extracellular methemoglobin, which occurs in the subacute phase as RBCs lyse.^{15,16,27,28} The lenticular shape of the hematoma in this case was typical of subacute subdural hematomas in people. Osmotic pressure within the hematoma increases as cellular lysis occurs, resulting in an influx of water.¹⁵ A gradient echo pulse sequence, also known as T2 weighting, was not performed in this case, although it would likely have aided in the diagnosis. Local magnetic field inhomogeneities, caused by the paramagnetic effects of deoxyhemoglobin and methemoglobin, result in a hypointense signal from enhanced susceptibility effects in gradient echo imaging.²⁹⁻³¹ As a result, gradient echo pulse sequences have been used in dogs and people as a sensitive method of detecting hematomas associated with the brain and spinal cord.²⁹⁻³¹

In this case, the clinical signs of cervical pain and scleral hemorrhage occurred between 24 and 36 hours after a cluster of grand mal seizures and a CSF tap. The MRI was performed 1 week later. It is still unclear if the brainstem subdural hematoma in this case was secondary to a traumatic event during the seizures, a complication of the CSF tap, a consequence of suspected hypertension, or from other unknown causes. The most likely theory in this case is that head trauma during seizure activity was the cause of the subdural hemorrhage, and, as in people, the hematoma occurred at the countercoup site.¹⁵ A complication of an improper CSF tap was unlikely, as the spinal needle would have had to pass through the entire brainstem and hit a vessel in the subdural space without causing any additional neurological signs. Hypertension in this case was suspected based on repeated abnormally elevated measurements separate from the immediate postictal period; however, an underlying cause of the hypertension was not identified. The bilateral scleral hemorrhages resolved spontaneously and were not investigated further.

Subdural hematomas in people are common neurosurgical lesions with an incidence rate of one to two occurrences per

100,000 cases per year, and they are often associated with traumatic head injury.^{32,33} However, subdural hematomas of the posterior fossa are very rare in adults.^{32,34} The rarity of posterior fossa subdural hematomas in people is probably related to the small number of bridging veins in the posterior fossa in comparison with the supratentorial subdural space.³² Subdural hematomas in dogs are rarely reported, and to the authors' knowledge, this is the first documented subdural hematoma of the ventral brainstem that was revealed on MRI and successfully treated with surgery.^{2,5}

Conclusion

A subdural hematoma of the brainstem was suspected in an 8-year-old Dalmatian on the basis of the MRI findings. The hematoma was surgically removed and confirmed histopathologically. From its MRI features, the hematoma was staged as between the acute and the early subacute phase and was thought to be approximately 1 week old. Because of the history of grand mal cluster seizure occurring 1 week before the MRI, a traumatic event during the seizure episode was considered the most likely cause of the hematoma in this case.

^a Parks Electronics, Aloha, OR 97007

^b Enacard; Merial, Iselin, NJ 08830

^c Cephalexin generic; Ivax Pharmaceutical, Inc., Miami, FL 33137

^d Mannitol generic; Abbott Laboratories, North Chicago, IL 60064

^e Salix; Intervet, Inc., Millsboro, DE 19966

^f Generic Vit K1; Phoenix Pharmaceutical, Inc., St. Joseph, MO 64503

^g Dinamap; Critikon, Tampa, FL 33634

^h Norvasc; Pfizer, Inc., New York, NY 10017

ⁱ Torbugesic; Fort Dodge Animal Health, Fort Dodge, IA 50501

^j Numorphan; Endo Pharmaceutical, Inc., Chadds Ford, PA 19317

^k General Electric Medical Systems, 1.0 T Signa, Milwaukee, WI 53201

^l Magnevist; Berlex Laboratories, Wayne, NJ 07470

^m Propofol; Abbott Laboratories, North Chicago, IL 60064

ⁿ Midazolam generic; American Pharmaceutical Partners, Inc., Schamburg, IL 60173

^o Isoflo; Abbott Laboratories, North Chicago, IL 60064

^p Cephazolin generic; American Pharmaceutical Partners, Inc., Schamburg, IL 60173

^q Rymadil; Pfizer Animal Health, Exton, PA 19341

^r Fentanyl generic; Abbott Laboratories, North Chicago, IL 60064

^s Phenobarbital injection-generic; Elkins-Sinn, Inc., Cherry Hill, NJ

08003; Phenobarbital tablet-generic; West-ward Pharmaceutical Corp., Eatontown, NJ 07724

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