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Brainstem Abscess Due to Plant Foreign Body in a Dog

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A 7-month-old male intact Dachshund was referred because of a 1-month history of ataxia, tetraparesis, and depressed mental status. Clinical signs were progressive until examination and remained static afterwards. The dog had been treated with prednisone[a] (2 mg/kg body weight PO q24h) and vitamin B complex.[b] Results of hematologic and serum biochemical analysis performed by the referring veterinarian showed mild thrombocytopenia (142×10^3 platelets/μL; reference range, 170–530 $\times$ 10^3 platelets/μL) and neutrophilia (82%; reference range, 40–75%). Serum antigen for *Dirofilaria immitis* and antibodies for *Ehrlichia canis* and *Borrelia burgdorferi* were negative. Hematologic and serum biochemical analysis performed 2 days before referral revealed increased protein concentration (8 g/dL; reference range, 5.7–7.7 g/dL) and total globulin (4.29 g/dL; reference range, 2–4 g/dL). Other variables were within reference range. Serum polymerase chain reaction for canine distemper virus was negative. The dog was current on routine vaccination and no history of systemic disease was noticed by the owner, except for sporadic neck pain.

No abnormalities were detected on physical examination. Neurologic examination revealed depressed mental status, spastic tetraparesis, and ataxia, with pelvic limbs more affected. Abnormalities on postural reactions included absent hopping reaction on the thoracic limbs and decreased extensor postural thrust reaction. Hopping reaction on pelvic limbs and all spinal reflexes were normal. Cranial nerve deficits included depressed menace reaction on both sides, although more severe on the right eye, and bilateral decrease on palpebral reflex. There was no visual impairment. Neuroanatomic lesion localization was considered focal in the pons and medulla. The depressed menace reaction could be attributed to the depressed mental status and/or facial paresis.

Magnetic resonance imaging (MRI) study of the brain and cervical spinal cord was performed using a superconductive magnet operating in 0.5 T.[c] Anesthesia was induced with propofol[d] (6 mg/kg body weight) and maintained with isofluorane[e] until the end of the procedure. MRI study included T1-weighted spin echo (SE) sequences (500/14; repetition time [TR]/echo time [TE]), T2-weighted fast spin echo sequences (4000/110/16; TR/TE/echo train), and gadolinium-dimeglumide enhanced T1-weighted SE sequences in the 3 orthogonal planes (gadolinium chelate[f] dose was 0.1 mMol/kg, IV).

The study revealed the presence of a focal swelling with prolongation of T1 and T2 relaxation rates in the medulla (Fig 1). An intra-axial round mass was localized in the center of the medulla. The lesion induced changes related to vasogenic edema, corresponding with hypointensity on T1-weighted images and hyperintensity on T2-weighted images. The mass enhanced heterogeneously in the periphery on T1-weighted postcontrast images. Mild dilation of the ventricular system, probably related to obstruction of the cerebrospinal fluid (CSF) flow caused by the lesion, was also present. On the basis of the MRI findings, the main differential diagnoses included neoplasm (diffuse glioma) and abscess. Because of the poor prognosis the animal was humanely euthanized.

On necropsy, no abnormalities were found apart from congestion on the entire brain, which was fixed in 10% buffered formalin and paraffin embedded. Sections 4-μm thick were obtained with a sliding microtome and stained with hematoxylin and eosin and Masson trichrome. Microscopic examination of the brainstem identified a focal pyogranulomatous reaction and associated areas of malacia with severe inflammatory reaction, localized ventrolaterally in the medulla oblongata (Fig 2A). Plant material, together with necrotic debris, was acting as a foreign body, surrounded by a thick wall formed by a proliferation of fusiform cells and abundant collagen fibers, together with inflammatory cells (neutrophils and macrophages) (Fig 2B). Masson trichromic stain revealed expanded connective tissue constituting a capsule around the plant material previously described. This inflammatory reaction was associated with numerous gitter cells, neutrophils, lymphocytes, and plasma cells. Chromatolytic and picnotic neurons, spheroids, diffuse spongiosis, and gliosis were observed in the adjacent parenchyma.

Final diagnosis was a caudal brainstem abscess because of migrating plant foreign body. Migration of plant material is a frequent problem in veterinary medicine, most often involving the external ear canal, oral and nasal cavities, interdigital web, and nictitating membrane.[1] Thus, a wide variety of symptoms could be seen, depending on where the inflammation is produced. In most cases, abscesses are located in the dermis, but they can be sporadically detected practically everywhere in the body, because of the capability of the grass awn to migrate. Signs can be attributable to vertebral osteomy-

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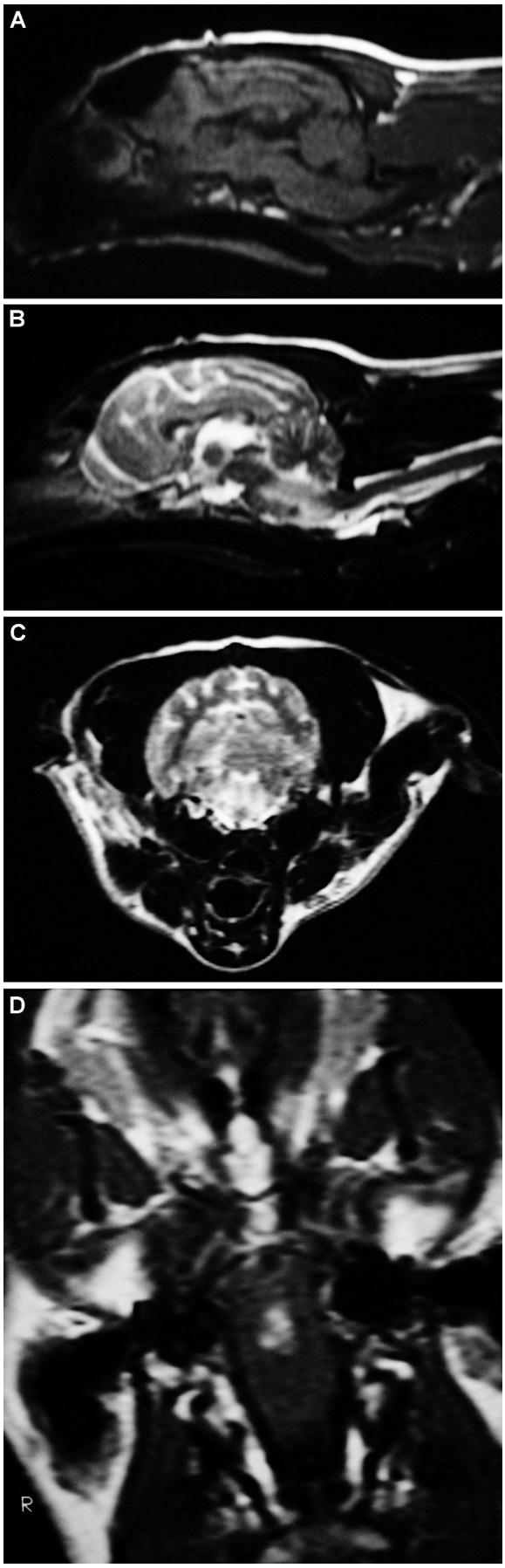

elitis and discospondylitis,[2] granulomatous pleuritis,[3] or peritonitis,[3] among others.

Foreign body granulomas in the brain of dogs and cats have been reported sporadically in the literature.[4–8] This dog had a plant foreign body granuloma in the medulla oblongata that was imaged on magnetic resonance. A review of the literature revealed that some other cases of intracranial foreign bodies, vegetable and nonvegetable, have been described, but in different locations. A porcupine quill in a Saint Bernard was localized in the occipital lobe of the left cerebral hemisphere. The quill probably penetrated into the cranial cavity through the foramen magnum.[4] A sewing needle penetrated the pharyngeal soft tissue and the base of the cranium and was localized in the left pyriform lobe[5] in a cross-breed adult dog.

Few cases of brain neurologic lesions attributable to migrating plant material have been described in small animals; however, the migration route is usually not elucidated. In one dog plant material was localized in the right lateral and third ventricles, probably after a perioocular pathway.[6] In the other dog a grass awn was located in the ventrolateral aspect of the right occipital lobe, internal capsule, and rostral horn of the lateral ventricle, suggesting migration through the ventral aspect of the cranium.[6] In the latter dog, plant material was localized within the dura overlying the occipital lobe; the migration route probably involved the foramen magnum.[6] Pyogranulomatous otitis media/interna with osteomyelitis causing pyogranulomatous meningoencephalitis in 2 dogs and 1 cat was associated with intrathecal plant material, suggesting that this material (a foxtail) could have migrated through the ear canal into the tympanic cavity and finally into the brain, possibly through the internal acoustic meatus.[7] A cat with brain abscess in the right thalamic nuclei because of a migrating foxtail from the nasal cavities through the cribiform plate to the encephalon has also been described.[8] Presence of grass awn in nasopharyngeal localization has been previously reported in cats.[9] Foreign bodies from nasal cavities causing brain damage have also been reported.[10] Intracranial penetrating injuries via optic canal are relatively frequent in human medicine, and they are often caused by chopsticks.[11–13]

The migration route in the current dog remains obscure because no lesions were evident on physical examination or on postmortem studies. We suspect that the foreign body could have penetrated through pharyngeal tissues directly to the space between the

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Fig 1. Magnetic resonance images of the brain. **(A)** Sagittal spin echo (SE) T1-weighted image. Note the enlargement of the medulla and low signal intensity (T1 relaxation time prolongation). **(B)** Sagittal fast spin echo (FSE) T2-weighted image. Note the enlargement of the medulla and diffuse signal intensity (T2 relaxation time prolongation). **(C)** Transverse FSE T2-weighted image through the medulla shows asymmetric enlargement, high signal intensity, and edema. **(D)** Postcontrast dorsal SE T1-weighted through the medulla shows the lesions with central core nodular enhancement.

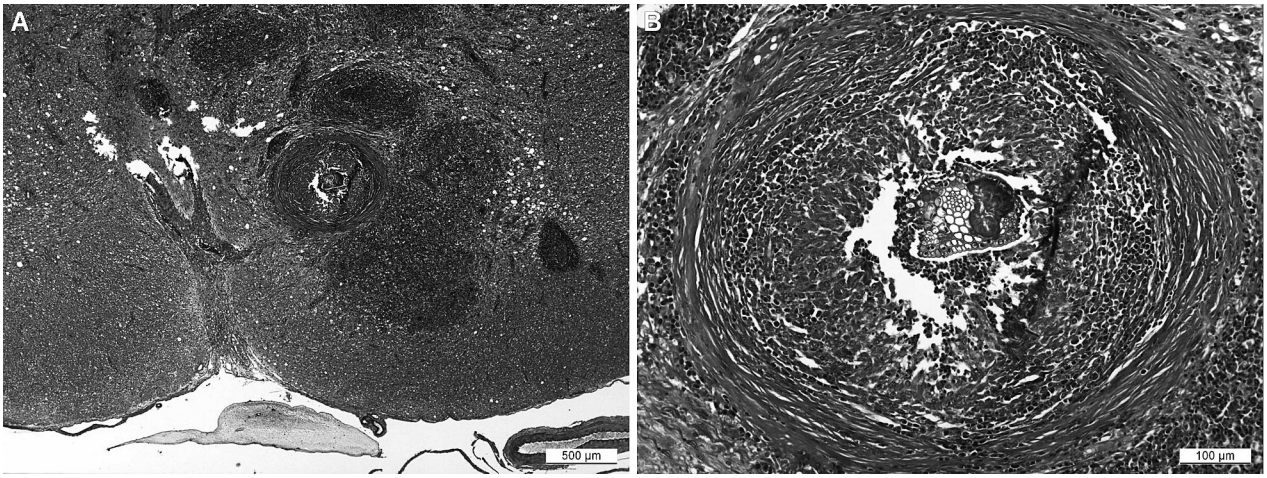

Fig 2. Histopathologic study. **(A)** Brain abscess at the level of the medulla oblongata showing the inflammatory reaction associated with the vegetal foreign body granuloma. Hematoxylin and eosin. Bar = 500 µm. **(B)** Detail of Figure 2A. Notice the connective tissue constituting a capsule around the vegetal tissue, which is surrounded by inflammatory cells (neutrophils and macrophages). Hematoxylin and eosin. Bar = 100 µm.

occiput and atlas, therefore migrating cranially to the brainstem. This same migration pattern has been suggested before in a case of a plant material granuloma involving a lesion in the junction between the medulla oblongata and first cervical segment.[14] Pharyngeal injuries are recognized sporadically, especially in medium and large breed dogs.[15,16] They may produce neurologic involvement affecting the spinal cord causing osteomyelitis[17] and discospondylitis with subsequent neck pain, gait abnormalities, and other neurologic deficits,[14,17–20]

Intracranial abscesses in humans generally have a hematogenous source and have been classified into 4 different stages as determined by MRI features and the time the abscess was formed. Early and late cerebritis and early and late abscess formation are the 4 progressive stages, although there is a gradual evolution in the phase of infection and host response. Early cerebritis, corresponding to stage 1, may show different T1 signal intensities, ill-defined high T2, and poorly defined enhancement because of the blood-brain barrier disruption. Later, in stage 2, central necrosis and formation of a capsule with neovascularization occur, resulting in a ring-enhancing lesion. MRI shows irregular periphery with iso- to high-signal on T1 and central high T2 core with peripheral enhancement on postcontrast sequences. Early abscess formation, or stage 3, which corresponds to early capsule formation, may show thin rim, high intensity on T1, very low intensity on T2, and intense enhancement. A capsule of decreased signal intensity in T2 sequence is not usually seen in tumors. The decreased signal intensity is caused by paramagnetic free radicals within phagocytic macrophages.[21] Late abscess, stage 4, which corresponds to late capsule formation, may show marked surrounding high T2-intensity edema, contraction of the core, irregular inner margin of rim, enhancement of rim, and parts of the core.[22] The length of time required to form a mature abscess varies from 2 weeks to several months.

The chronicity of clinical signs in the dog in this case report suggests stage 4 brain abscessation or late capsule stage. Nevertheless, not all the classic MRI features expected, such as capsular enhancement, were present, probably because of the unusual structure of the inflammatory process, namely foreign body core, which mimicked the features of a granuloma. Also, some abscesses may resemble primary or secondary neoplasm, and this differential diagnosis was considered. New MRI techniques, such as difussion-weighted imaging and proton magnetic resonance spectroscopy, are invaluable for accurate diagnosis in such cases.[22] Notwithstanding, conventional MRI has proved to be a highly sensitive and specific method to detect inflammatory lesions in the brain parenchyma.

CSF collection might have been useful to differentiate inflammatory disease from neoplasia. In this case, however, because of the encapsulated nature of the lesion, CSF would likely have been nondiagnostic. Besides, cellularity and culture in central nervous systems bacterial infections are often unrewarding in the chronic stage.[23]

Brain abscesses can be initiated from different routes and usually microorganisms reach the brain parenchyma as a result of contiguous infection (otitis media/interna, nasal sinuses infections), hematogenous dissemination or head injury.[24] The most frequent microorganisms isolated from lesions because of migrating plant material are *Streptococccus* sp., *Staphylococcus aureus, Pasteurella multocida, Actinomyces* sp., and *Nocardia* sp.[1]

Moreover, clinical symptoms may not present for several days, months, or even years after an injury.[12,13,23] This condition should be taken into account when signs of focal brain lesion are present together with compatible features in MRI, particularly for the medical and surgical implications.

Footnotes

[a] Dacortin comp. 5 mg, Merck Farma y Química, SA, 08100 Mollet del Vallés (Barcelona), Spain
[b] Nervobión caps., Merck Farma y Química, SA, 08100 Mollet del Vallés (Barcelona), Spain
[c] Gyroscan T5-NT, Philips. The Netherlands
[d] Propofol-Lipuro 1%, B. Braun Melsungen AG, D-34209 Melsungen, Germany
[e] Isoba Vet, Schering-Plough, SA, 28750 San Agustin de Guadalix (Madrid), Spain
[f] Magnograf 0.5 mmol/ml solución inyectable. Schering España, SA, 28045 Madrid, Spain

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