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NOTE

Surgery

Encapsulated gas accumulation in the spinal canal: Pneumorrhachis in two dogs

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ABSTRACT. A 17-year-old mongrel dog and 12-year-old Shiba Inu dog presented with ataxia and paresis of the pelvic limbs, respectively. Gas accumulation within the spinal canal adjacent to the herniated disc was suspected in both cases. Since the gas remained accumulated for a prolonged period, hemilaminectomy was performed to decompress the spinal cord. The bulged external lamina of the dura matter was removed and histopathologically examined. Granulomatous inflammation and hyperplasia of fibrous connective tissues was noted, suggesting that the gas was encapsulated and the fibrous nodules made reabsorption difficult. Clinical signs resolved post-surgery. This is the first report describing histopathological features of pneumorrhachis in dogs. The accumulated gas was successfully removed by surgery. Postoperative course remained uneventful in both cases.

KEY WORDS: dog, histopathological examination, intervertebral disc herniation, pneumorrhachis

Pneumorrhachis (PR) denotes the presence of gas in the spinal canal. Accumulation of gas may be caused by trauma, pyogenic infections, pneumothorax, the vacuum phenomenon as a physical factor, and iatrogenic etiologies [5]. The vacuum phenomenon, causing an accumulation of gas within a herniated intervertebral disc space or within an epidural space, is reported to occur frequently in middle-aged humans [7]. The pathomechanism of the vacuum phenomenon is thought to be related to gas formation from the surrounding extracellular fluid that diffuses to spaces with negative pressure caused by a herniated intervertebral disc [10]. The clinical features are often asymptomatic, but some cases have neurological signs and/or sciatica [12]. In humans, it is known that gas generated at the intervertebral disc moves to the epidural space and may cause neuralgia [12].

In the veterinary field, PR within the spinal canal has been reported in five dogs [1, 9, 13]. In these reports, PR was symptomatic and thought to be caused iatrogenically or by distraction of the end plates. In four out of the five dogs, vertebral disc degeneration caused PR secondary to the vacuum phenomenon. In some cases, the gas generated by the vacuum phenomenon can remain in the spinal canal for a prolonged period. However, it is not clear when and why the gas remains, and no histopathological features are known. Therefore, no clinical treatment guidelines exist [12], and PR has to be examined carefully to plan an appropriate treatment.

In the present report, we describe the characteristics and our treatment of PR in 2 canine cases with histopathological evidence.

Case 1: A 17-year-old male mongrel dog weighing 13.7 kg was presented to Watanabe Animal Hospital with a 1-week history of ataxia of the pelvic limbs (Day 1). A neurological examination revealed ataxia of the pelvic limbs and severe pain in the thoracolumbar area. The patient's mental status was alert. Postural responses and spinal reflexes in the four limbs were unremarkable. Based on these findings, the neurological localization was suspected to be the T3–L3 spinal segment. Complete blood cell counts and blood biochemical examination revealed normal findings.

A radiographic examination of the thoracolumbar area revealed narrowing of the T12–T13 intervertebral disc space. The patient was tentatively diagnosed with thoracolumbar intervertebral disc herniation (modified Tarlov functional grade II [14]) and treated with 1.5 mg/head meloxicam (Metacam, Boehlinger Ingelheim, Ingelheim am Rhein, Germany). Despite medical treatment, the patient's neurological signs deteriorated on day 7. A neurological examination revealed paresis of the pelvic limbs. Postural

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responses in the pelvic limbs were absent. Superficial pain perception was intact, and the spinal reflexes were normal. For further evaluation of the lesion, magnetic resonance imaging (MRI; 0.3 Teslas [T], AIRIS Elite, Hitachi Ltd., Tokyo, Japan) and computed tomography (CT; ECLOS, Hitachi Ltd.) were performed on day 9. On MRI, T2-weighted and T1-weighted images revealed a signal void area in the extradural space at the level of the T12–T13 intervertebral disc space, compressing the spinal cord from the ventral side. The epidural signal void area was also observed on the right dorsolateral side of T12–T13 spinal cord segment (Fig. 1A, 1B). No contrast enhancement of the lesion was observed. CT (helical mode, 1.25 mm slice thickness, 120 peak kilovoltage [kVp], 175 milliamps [mA]) showed a hyperattenuating lesion (mean Hounsfield Unit [HU] value: 800) compressing the T12–T13 spinal cord segment. The hypoattenuating area on the right dorsolateral side of the T12–T13 spinal cord segment on CT (mean HU value: −900) corresponded to the signal void area on MRI (Fig. 1C, 1D). The signal void area displaced the spinal cord to the left. Based on these MR and CT images, a diagnosis of T12–T13 intervertebral disc herniation and accumulation of gas in the vertebral canal, causing spinal cord compression, was made. The cerebrospinal fluid (CSF) was not evaluated.

On day 26, a right hemilaminectomy for removal of the compressive lesion was performed from the caudal end of T12 to the cranial end of T13. The intervertebral disc materials compressing the spinal cord were removed. The extradural cystic structure on the right dorsolateral side of the spinal cord was also removed and submitted for histopathological examination (Fig. 3A), which revealed that the lesion was composed of degenerative fibrous nodules including a calcified area at the center. The calcified area was hollowed and covered with fibrous connective tissue, and some areas showed osteoproliferative and fibrocartilage changes. Rehabilitation was started 6 days after surgery. The dog regained ambulation 49 days after surgery.

Case 2: A 12-year-old male Shiba Inu dog weighing 10.9 kg was presented to the Animal Medical Center of Gifu University with a 1-month history of paresis of the pelvic limbs and lumbar pain (Day 1). The dog had a history of allergic dermatitis and hypothyreosis. A neurological examination revealed wobbly gait and paresis of the pelvic limbs, which was especially severe on the right. Superficial pain perception was intact. Postural responses in the pelvic limbs were absent. The patient's mental status was alert and the spinal reflexes were normal. Based on these findings, the neurological localization was suspected to be the T3–L3 spinal segment.

Radiographic examination revealed narrowing of the T12–T13 intervertebral disc space. The vertebral rim around the T12–T13 endplates showed an irregular shape and increased radiopacity. On MRI (0.3 T, APERTO Lucent, Hitachi, Ltd.), T2-weighted, T1-weighted, and FLAIR images revealed two signal void areas compressing the spinal cord on the right dorsolateral and left lateral sides of the T12-T13 spinal cord segments (Fig. 2A, 2B). These areas showed no contrast enhancement. CT (Alexion^{TM}/Advance Edition, Canon Medical Systems, Tochigi, Japan; helical mode, 0.5-mm slice thickness, 120 kVp, 50 mA) showed protrusion of intervertebral disc materials compressing the spinal cord at the level of the T12–T13 intervertebral disc space. In the same area, the intervertebral disc space was collapsed and osteophytes were observed, suggesting spondylosis deformans (Fig. 2C, 2D). There were two lesions on the CT in the right dorsolateral and left lateral areas of the T12–T13 spinal cord segment (mean HU value:

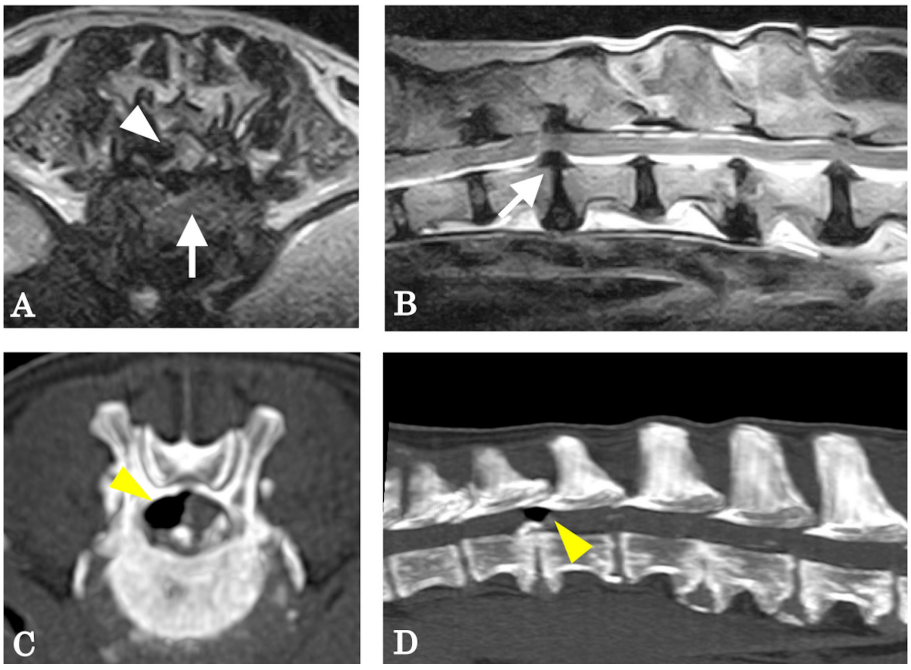

Fig. 1. Case 1: T2-weighted transverse (A) and sagittal (B) magnetic resonance images at the T12–T13 level. Signal void area compressing the spinal cord (white arrow) and extradural signal void area representing gas accumulation on the right dorsolateral side of the spinal canal (white arrowhead). Transverse (C) and sagittal (D) computed tomography (CT) images. Hypoattenuating area (yellow arrowhead) corresponded to the signal void area on MRI. These images show the combination of intervertebral disc herniation and gas bubbles.

−830 HU and −960 HU, respectively). These MR and CT images suggested bilateral gas accumulation in the T12–T13 spinal cord segment. The number of cells in the CSF collected from the cisterna magna was within normal limits and aerobic bacterial culture examination of the CSF was negative.

On day 7, although gas on the right dorsolateral side of the spinal cord was observed on CT performed before the operation, there was no gas accumulation at the left side. The gas at the right side displaced the spinal cord to the left side, causing spinal cord compression. Therefore, a right hemilaminectomy at the T12–T13 intervertebral disc space and vertebral fixation were performed to decompress the spinal cord and stabilize the vertebral bodies. During surgery, the bulging external lamina of the dura mater was observed. The bulged dura matter was removed and submitted for histopathological examination and a bacterial culture. After decompressing the spinal cord, the T12 and T13 vertebral bodies were stabilized with 2.7 mm screws (Cortex screw, Synthes®, Solothurn, Switzerland) and bone cement (Simple™ P Radiopaque Bone Cement, Stryker, Tokyo, Japan).

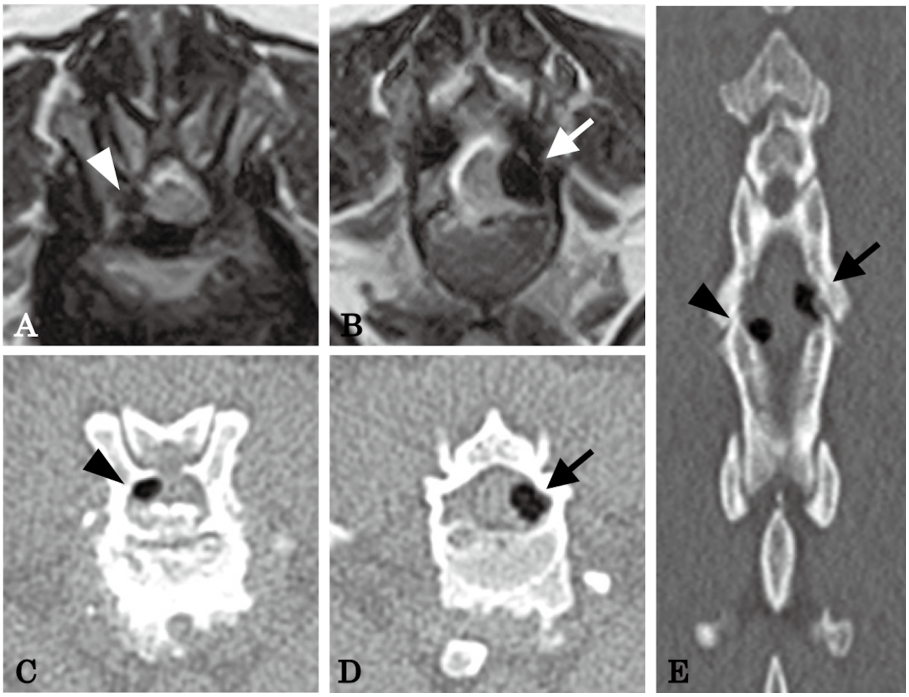

Fig. 2. Case 2: (A, B) T2-weighted transverse magnetic resonance images at the level of the T12–T13 disc space show bilateral signal void areas (right side: white arrowhead and left side: white arrow). (C, D, E) Computed tomography images at the level of the T12–T13 disc space show the spondylosis deformans. There are bilateral hypoattenuating areas in the same area on MRI (arrow and arrowhead). The gas at the left side (arrow) was not present on the preoperative CT image.

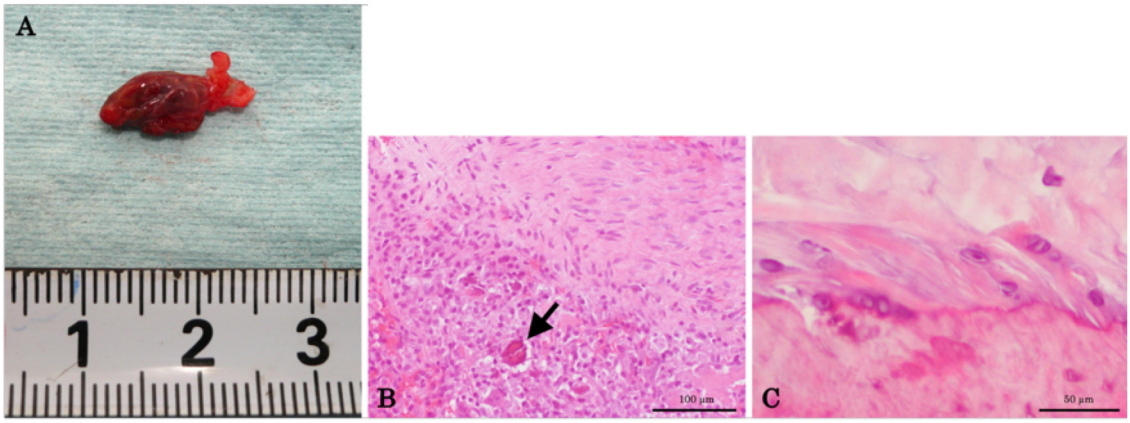

Fig. 3. (A) The removed cystic lesion of Case 1 and (B, C) histopathological images of the removed tissues of Case 2. (A) The bulged dura matter (0.8 × 0.3 × 0.3 cm) was removed. (B) The dura matter shows a calcified area in the center of the granuloma (arrow) and hyperplasia of fibrous connective tissues. (C) The substance compressing the spinal cord shows cartilage tissues with calcification. (Hematoxylin and eosin staining).

Histopathological examination revealed that the substance compressing the spinal cord was cartilage tissue with calcification. The bulged periosteal dura matter was composed of an accumulation of macrophages and hyperplasia of fibrous connective tissues including a calcified area at the center (Fig. 3B, 3C). Aerobic and anaerobic bacterial culture examinations were negative. Although the dog remained ataxic, he could ambulate and postural responses were recovered by 28 days after surgery.

The etiology of PR is divided into iatrogenic, traumatic, and non-traumatic causes [12]. In the veterinary field, the common causes of PR are associated with trauma and intervertebral disc herniation [11]. In the present report, CT and MR imaging indicated gas accumulation in the spinal canal. Neither dog had a history of surgery before the first presentation. Infection with aerogenes was considered negative based on the bacterial culture results. The spinal cord was compressed by intervertebral disc materials, and gas accumulation was observed within the epidural space in the spinal canal where the intervertebral disc herniation had occurred. We consider that the gas, generated in the intervertebral disc space by the vacuum phenomenon, moved to and accumulated in the spinal canal [4].

Generally, gas generated in the body can be reabsorbed into neighboring tissues and is less likely to cause spinal cord compression [2]. However, in humans, it was reported that an encapsulated gas pseudocyst might remain in the spinal canal for a long time [12, 15]. In Case 1, the removed lesion was encapsulated by connective tissues (Fig. 3A). Furthermore, on CT and MR images in both cases, the gas within the spinal canal formed the round shape, suggestive of encapsulated gas accumulation. In Case 2, although the gas on the left side of the spinal cord had disappeared at the time of surgery, that on the other side remained. Histopathological examination revealed granulomatous inflammation and hyperplasia of fibrous connective tissues, suggesting that the fibrous nodules made reabsorption of the gas difficult. Similarly, histopathological examination in Case 1 revealed fibrous nodules. In both cases, a calcified area was observed at the center of the bulged external lamina of dura matter. It has been suggested that such encapsulation is caused by an inflammatory reaction to the calcified intervertebral disc substance [8]. It is thought that gas accumulates in the spinal canal via several processes as follows: (1) When the degenerated intervertebral substance extrudes, the intervertebral disc space experiences negative pressure and gas is generated by the vacuum phenomenon [4]; (2) the gas moves into the spinal canal and remains in the area [2, 6]; (3) an inflammatory reaction is induced by the calcified intervertebral substance [8]; and (4) the encapsulation makes reabsorption of the gas difficult [15].

PR is often asymptomatic or treatable by conservative treatment [16]. However, it is recommended that cases showing progressive neurological deficits receive surgical treatment [3, 9]. In the present report, gas beside the intervertebral disc herniation remained for a prolonged period and might have caused the neurological signs. Thus, in addition to progressive neurological deficits despite conservative management, when gas associated with intervertebral disc herniation remains for a prolonged period, surgical removal is of considerable importance.

This case report describes PR with histopathological analysis in dogs. Gas that remains in the spinal canal for a prolonged period may cause neurological signs because encapsulation by fibrous tissues makes spontaneous reabsorption of the gas difficult. In this report, surgical removal of epidural gas resulted in good outcomes.

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