Pyogenic meningitis and trigeminal neuritis secondary to periodontogenic paranasal sinusitis in a red deer (Cervus elaphus)

Kenji KUTARA1)*, Sho KADEKARU1), Reiko SUGISAWA2) and Yumi UNE1)

1)Faculty of Veterinary Medicine, Okayama University of Science, 1-3 Ikoinooka, Imabari, Ehime 794-8555, Japan
2)Tohoku Safari Park, 1 Sawamatsukura, Nihonmatsu, Fukushima 964-0088, Japan

ABSTRACT. An adult female red deer died of a severe seizure and dysbasia. Postmortem computed tomography (CT) and magnetic resonance imaging (MRI) were performed. On CT, deciduous right maxillary second and third premolar teeth were observed, and the right infraorbital canal was disrupted. MRI showed that the right trigeminal nerve was enlarged and the right subarachnoid cavity was occupied by fluid and gas. On gross examination, the right paranasal sinus, swollen muscles of the orbit and tonsils, right trigeminal nerve, and right cerebrum surface contained a yellowish-white, cheese-like pus. Based on these findings, the deer was believed to have developed pyogenic meningitis caused by a neuropathic infection secondary to periodontogenic paranasal sinusitis.

KEY WORDS: infraorbital canal, paranasal sinusitis, pyogenic meningitis, red deer

In veterinary medicine, dental diseases occur in various animals [1, 11, 12, 15]. These periodontal diseases can lead to paranasal sinusitis or rhinitis via development of an oronasal fistula, which can result in fatal complications [2, 3]. Previous studies have presented cases of intracranial abscessation and bacterial meningitis in deer. Especially, the cause of these diseases was suspected to be via the hematogenous route owing to intestinal infection and trauma [4–6, 20].

Computed tomography (CT) and magnetic resonance imaging (MRI) can be performed to investigate the structure of and pathology in various animal heads. However, in large animals, these examinations have been limited by their inherent body size. Recently, this challenge has been overcome by the development of special machines for large animals (i.e., increase in the size of the gantry and setting of the bet tolerable to heavy weight) or by solely imaging the head after decapitation [8, 14, 18]. Postmortem images have provided the opportunity to investigate the three-dimensional anatomical and pathological features, without destruction of the specimen. Hence, recently, postmortem images have become important alternative or supportive tools during human autopsies [16, 19]. Here, we present a case in which postmortem CT and MRI were performed to determine the precise location and characteristics of pyogenic meningitis and trigeminal neuritis secondary to periodontogenic paranasal sinusitis in a red deer.

An adult female red deer (age, unknown; breeding for more 15 years), which weighed approximately 100 kg and was housed at the Tohoku Safari Park in Fukushima, Japan, died of a severe seizure and dysbasia. The red deer was bred in multi-headed breeding and received a diet of grass, hay, and feed mixtures. No detailed clinical examinations were performed. The red deer was subjected to necropsy approximately 3 hr after death. During necropsy, pneumonedema was observed in the lung; however, no other significant findings were found in other organs and lymph nodes. The head was removed from the body and transported to Okayama University of Science, Imabari, Japan, in a low-temperature condition, within 24 hr of decapitation.

Postmortem CT and MRI examinations were performed on the decapitated head. On CT, deciduous right maxillary second and third premolar teeth were observed (Fig. 1A). The maxillary alveolar bone at these levels was melted and formed a fistula (Fig. 1A, 1B). In addition, the right paranasal sinus was occupied by a gaseous lesion (Fig. 1B, 1C), and the right infraorbital canal was disrupted (Fig. 1C). Moreover, the right orbit and tonsil muscles were swollen, and the right orbital fissure was expanded. On MRI, the enlarged muscles of the right orbit and tonsil showed heterogeneous hyperintensity on T2-weighted images (Fig. 2A). The right trigeminal nerve was enlarged (Fig. 2B, 2C), and the right subarachnoid cavity was enlarged and occupied by fluid and gas (Fig. 2B, 2C). The right brain was enlarged, and the midline was displaced to the left (Fig. 2C). MRI showed that the optic nerve was not enlarged.

On postmortem gross examination, the right paranasal sinus and the enlarged muscles of the orbit and tonsil contained a yellowish-white, cheese-like pus (Fig. 3A). After performing craniotomy, the surface of the right cerebrum and the right
subarachnoid cavity were also observed to be covered by pus (Fig. 3B). After the brain was removed from the cucullus, the right trigeminal nerve at the base of the skull was found to be covered in pus (Fig. 3C). The right brain was enlarged, and the midline was displaced to the left (Fig. 3D). There was no evidence of foreign-body penetration into the paranasal sinuses. We did not obtain different findings after examining the postmortem gross examination findings and the postmortem imaging findings.

Histologically, a severe, purulent inflammation was observed in the right trigeminal nerve fiber bundle, ganglion, and its...
branches, and in the soft tissue surrounding the trigeminal nerve (Fig. 4A). The structure of the trigeminal ganglion could not be confirmed because of the high degree of colliquative necrosis, with only a few degenerated and necrotic nerve fiber bundles remaining. In the right cerebral hemisphere, a high degree of purulent inflammation was observed on the arachnoid mater surface (Fig. 4B). The subarachnoid space was dilated because of the accumulation of a large quantity of fibrin. In this space, the exudative fibrin was organized, and the meninges were fibrotic and thickened. Neuropil edema was observed in the cerebral molecular layer without inflammation.

The bacterial clusters were scattered throughout the trigeminal nerve, subarachnoid space, and meninges. The pus was aseptically collected from the cranial cavity and the right paranasal sinus and submitted for bacterial culture, with identification of *Escherichia coli* and *Pseudomonas aeruginosa* on aerobic and anaerobic cultures, respectively.
In this case, a right paranasal sinusitis caused by periodontal disease likely led to disruption of the infraorbital canal, which was along the path of the maxillary nerve (i.e., a branch of the trigeminal nerve). Moreover, the muscles along the maxillary nerve path and underlying the ophthalmic nerve (i.e., another branch of the trigeminal nerve) were swollen and suppurative. In the skull, the right trigeminal nerve was suppurative, and the right subarachnoid cavity was occupied by a pus. Hence, it was determined that this deer had developed pyogenic meningitis and trigeminal neuritis due to periodontogenic paranasal sinusitis.

In veterinary medicine, periodontal diseases can cause various complications. For example, in dogs, periodontal diseases can lead to oronasal fistula development, thus causing rhinitis [17]. In horses, the development of oronasal and sinus fistulas, a well-documented complication following loss or removal of maxillary molar teeth [9], can lead to sinusitis [17]. In deer, the relationship between dental diseases and sinusitis remains unclear; however, it has been reported that dental conditions can affect mortality in deer [10, 13]. In this case, the diet (consisting of grass, hay, and feed mixtures) was universal. The causation between the received diet and the disease was unclear. As this case was of an old deer, it was thought that she had a high risk of periodontal disease development. In previous reports of intracranial abscess and bacterial meningitis in deer, these diseases were caused through the hematogenous route owing to intestinal infection and trauma [4–6, 20]. In this case, intestinal infection and visible trauma were not present. Hence, the result of this case was considered that the bacterial meningitis may be occurred not only by hematogenous infection but also by neuropathic infection in deer.

Disruption of the infraorbital canal was demonstrated in this case. In horses, the infraorbital canal is located in the sinus; therefore, sinusitis can result from disruptions in this canal. Headshaking in horses is thought to involve the trigeminal nerve in the infraorbital canal, and it has been reported that horses with headshaking have increased mineralization and disruption of the infraorbital canal, with adjacent disease identified on CT imaging [7]. Furthermore, bacterial meningitis can be a fatal complication after sinus surgery in horses [2] and it can occur secondary to infections involving the paranasal sinuses and nasal cavity. In previous reports, bacterial meningitis in horses was fatal [2, 21]; however, these reports did not include CT or MRI findings. Therefore, the infections were only suspected to be caused by hematogenous spread. In this case, the infraorbital canal was located in the paranasal sinus, as in horses. Therefore, based on CT and MRI findings, it was believed that bacterial meningitis, which occurred secondary to sinusitis, was caused not only by hematogenous infection but also by neuropathic infection. Furthermore, it was believed that the periodontal disease may have caused an infection of the central nerve through the trigeminal nerve (infraorbital canal) in this deer.

Based on necropsy with postmortem CT and MRI, we can consider that this deer had developed pyogenic meningitis caused by a neuropathic infection secondary to periodontogenic paranasal sinusitis. Postmortem imaging may be provided with three-dimensional positional relation of the lesion without destruction of the specimen. From these images, pyogenic meningitis was deduced to be caused by an infection via the trigeminal nerve. A necropsy could confirm the postmortem CT and MRI findings. We believe that the performance of postmortem CT and MRI before autopsies may also provide helpful information for veterinary patients with neurological symptoms.

CONFLICT OF INTEREST. The authors have nothing to disclose.

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