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Prevalence of Effusion in the Tympanic Cavity in Dogs with Dysfunction of the Trigeminal Nerve: 18 Cases (2004–2013)

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Background: Animals with disorders involving the trigeminal nerve or its nuclei in the brainstem can have effusion in the tympanic cavity ipsilateral to the side of the neurological deficits. The tensor veli palatini muscle (TVP), innervated by the mandibular branch of the trigeminal nerve, opens the pharyngeal orifice of the auditory tube. With denervation of the TVP, dysfunction of the auditory tube may occur, which could lead to effusion.

Hypothesis/Objectives: To determine the prevalence of effusion in the tympanic cavity in dogs with disorders involving the trigeminal nerve.

Animals: Eighteen client-owned dogs were evaluated retrospectively.

Methods: Retrospective study.

Results: Diagnostic imaging databases were searched for dogs having undergone magnetic resonance imaging (MRI) evaluation for signs referable to dysfunction of the trigeminal nerve. Signalment and neurological examination findings were recorded. The MRI study was evaluated for the presence or absence of effusion. MRI characteristics of the affected trigeminal nerve and the muscles of mastication were recorded. Based on the location of the trigeminal nerve lesion, dogs were divided into three categories: brainstem, trigeminal canal, or extracranial. Eighteen dogs met the inclusion criteria. Six of 18 dogs (33%) had effusion in the tympanic cavity ipsilateral to the affected trigeminal nerve.

Conclusion and Clinical Importance: A substantial proportion of dogs with a lesion affecting the trigeminal nerve had effusion in the tympanic cavity. This finding likely represents denervation of the TVP muscle, which may have led to dysfunction of the auditory tube.

Key words: Tensor veli palatini denervation effusion.

Disorders involving the trigeminal nerve encountered in small animal practice include neoplasia such as nerve sheath tumors[1] and neoplastic lymphocyte infiltration of the trigeminal nerve,[2] trigeminal neuritis,[3] and trigeminal neuropathy.[4] Clinically, disorders of the trigeminal nerve can result in denervation of the muscles of mastication as a result of dysfunction of the mandibular branch of the trigeminal nerve. In addition, sensory deficits in the cutaneous areas innervated by the trigeminal nerve or of one or more of its individual branches also may be detected. An etiologic diagnosis is based on the history, unilateral or bilateral dysfunction, and clinicopathologic data often involving magnetic resonance imaging (MRI). The MRI appearance and contrast enhancement pattern of the normal trigeminal nerve have been reported.[5] Likewise, imaging findings in dogs with trigeminal nerve sheath tumors also have been published.[3] In addition to the enlargement and abnormal contrast enhancement of an affected nerve, atrophy and increased signal intensity of the muscles of mastication on T1-weighted images (T1WI) and T2-weighted images (T2WI) on the affected side are observed.[3] Abnormal contrast enhancement of the muscles of mastication also can be observed secondary to denervation.[6] We have frequently observed that effusion often is present in the tympanic cavity on the ipsilateral side of the affected trigeminal nerve. Interestingly, several veterinary publications have included figures in which effusion in the tympanic cavity is observed in animals with disorders of the trigeminal nerve without mention of a causal relationship.[3,7] In 1 report, a dog with effusion in the tympanic cavity ipsilateral to a Schwannoma of the trigeminal nerve was reported.[8] The authors suggested a causal relationship secondary to denervation of the tensor veli palatini (TVP) muscle leading to auditory tube dysfunction.[8] The aim of the present study was to report the prevalence of effusion in the tympanic cavity in dogs with disorders involving the trigeminal nerve. In addition, we provide support for the hypothesis that the effusion is secondary to denervation of the muscle.

Abbreviations:

LVP	levator veli palatini
MRI	magnetic resonance imaging
T1WI	T1-weighted images
T2WI	T2-weighted images
TVP	tensor veli palatini

Material and Methods

Case Selection

The medical record databases at the University of Georgia and Red Bank Veterinary Hospital were searched for dogs with

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the diagnosis of a trigeminal nerve disorder including neuropathy, neuritis, nerve sheath tumor, or disease process involving the trigeminal nerve. For inclusion, cases had to satisfy the following criteria: a complete medical record that documented physical and neurological examination findings from which a neuroanatomic diagnosis consistent with a lesion affecting the trigeminal nerve or ≥1 of its branches could be made and an archived MRI of the head that was available for review. To be consistent with the neuroanatomic diagnosis of a deficit involving the trigeminal nerve, all cases had to have visual evidence of unilateral or bilateral atrophy of the temporalis and masseter muscles alone or in conjunction with hypalgesia or analgesia of the skin in the cutaneous areas of ≥1 of the branches of the trigeminal nerve (mandibular, maxillary, ophthalmic nerves).[9] The MRI evaluations were performed using a 3.0 T unit (3.0 T Signa HDx)[a] at the University of Georgia or a 1.0 T unit (GE Genesis Signa)[a] or 1.5 T unit (GE signa Excite)[a] at Red Bank Veterinary Hospital. All dogs were imaged under general anesthesia in a prone position using an extremity coil (University of Georgia) or a head coil (Red Bank Veterinary Hospital). At a minimum, all MRI examinations had to consist of T1WI, T2WI, and postcontrast T1WI of the head obtained in the transverse plane. Although not necessary for inclusion, additional sequences were reviewed when present.

Medical Records Review

The following information was retrieved from the medical records for each dog: breed, age at the time of the MRI, sex, neuter status, and body weight. Information regarding the findings observed on the physical and neurological examinations and the neuroanatomic diagnosis were recorded. Documentation of the presence or absence of otitis externa upon visual inspection of the pinna and external ear canal and clinical signs related to otitis externa (erythema or abnormal discharge) or otitis media or interna (deficits involving cranial nerves VII and VIII and loss of sympathetic innervation to the eye) were recorded. The MRI studies were reviewed and the following information was recorded: presence or absence of effusion, the degree of the signal increase in comparison to the gray matter of the brain if effusion was present, the heterogeneity of the signal change, presence or absence of contrast enhancement, and pattern of contrast enhancement (no enhancement, homogenously increased, heterogeneously increased, or peripheral enhancement). The finding of effusion in the tympanic cavity was based on the observation of material with an increased signal compared to the signal void of the normal gas-filled tympanic cavity. The MRI characteristics of material in the tympanic cavity were not used exclusion criteria, because the MRI characteristics of material in the tympanic cavity vary widely.[8] The MRI characteristics of the affected trigeminal nerve were recorded. In comparison to the normal trigeminal nerve and its branches, the affected trigeminal nerve and its branches were classified based on size (normal or enlarged) and contrast enhancement pattern (normal, decreased, or increased), and location of the lesion. Dogs were divided into three groups based on the anatomic lesion involving the trigeminal nerve. The groups included the following: brainstem (lesion resulted in compression of the pons or medulla oblongata), trigeminal canal (lesion involved the trigeminal nerve within the trigeminal canal, oval or round foramina, or orbital fissure), and extracranial (lesion involved the extracranial branches of the trigeminal nerve) (Fig 1 and 2). If ≥1 segment was abnormal, classification was based on the most proximal extent of the lesion. Finally, the abnormal contrast enhancement of the denervated muscle in comparison to the unaffected side was recorded.

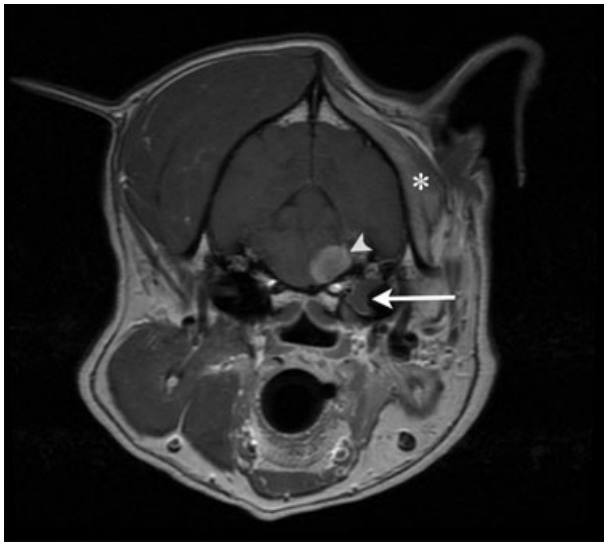

Fig 1. Example of a dog in the brainstem group. A transverse postcontrast T1WI of the head at the level of the medulla discloses an intra-axial lesion within the medulla (arrowhead). This lesion was contiguous rostrally with an enlarged trigeminal nerve. Note the effusion (long arrow) and peripheral enhancement of the tympanic cavity. Atrophy and abnormal enhancement of the temporalis muscle also are appreciated (asterisk).

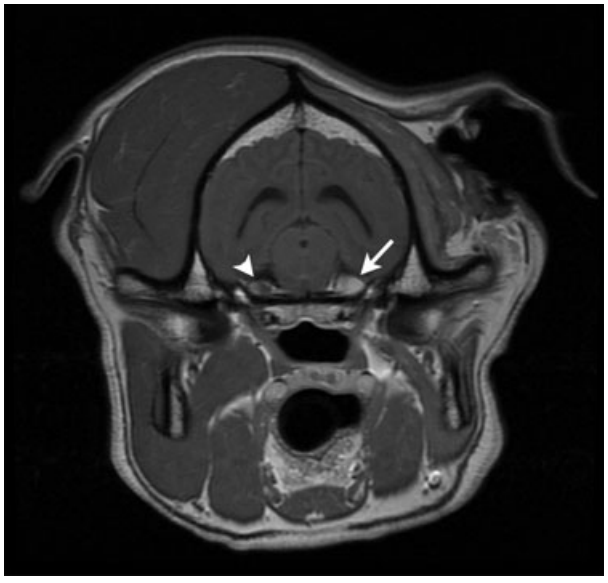

Fig 2. Example of a dog in the trigeminal canal group. A transverse postcontrast T1WI of the head at the level of the medulla discloses an enlarged trigeminal nerve within the trigeminal canal (arrow). In comparison to the right side (arrowhead), the left trigeminal nerve displays greater enhancement. Atrophy of the muscles of mastication also is present on the left. In an image caudal to this level, effusion was present in the left tympanic cavity.

Results

Eighteen dogs (10 from the University of Georgia and 8 from Red Bank Veterinary Hospital) met the inclusion criteria. Dogs ranged in age from 5 to

13 years with a median age of 10 years. There were 11 female and 7 male dogs. Body weight ranged from 8 to 43 kg with a median of 28.5 kg. There were 4 Labrador Retrievers, 4 Golden Retrievers, 2 Jack Russell Terriers, 4 mixed breed dogs, and 1 each of the following breeds: Standard Poodle, Miniature Poodle, Cocker Spaniel, and a Pitbull cross. On physical examination, none of the dogs had evidence of otitis externa or cranial nerve deficits (eg, cranial nerve VII, VIII), or loss of sympathetic innervation to the eye to implicate otitis media or interna.

A signal change consistent with effusion was observed in the tympanic cavity of 6 dogs (33%). In all cases in which effusion was observed, the effusion was noted only in the tympanic cavity ipsilateral to the affected trigeminal nerve. On T2WI, the effusion was homogenously hyperintense in 5 dogs. In 1 dog, the effusion was heterogeneously hyperintense. On T1WI, the effusion was homogenously hyperintense in 4 dogs, heterogeneously hyperintense in 1 dog, and homogenously isointense in 1 dog. In dogs with effusion, peripheral contrast enhancement of the tympanic cavity was observed in 3 dogs and no change was noted in 3 dogs on postcontrast T1WI.

In all but 1 dog, the trigeminal nerve were enlarged and displayed homogenous contrast enhancement. In 12 dogs, there was contrast enhancement of the muscles of mastication on the side ipsilateral to the abnormal trigeminal nerve or ipsilateral to the side of the deficits observed clinically. Anatomically, the locations of the lesion involving the trigeminal nerve were classified as brainstem in 10 dogs, trigeminal canal in 6 dogs, and extracranial in 2 dogs. One of the 2 dogs classified as extracranial did not have an abnormality of the trigeminal nerve on MRI. Given the size and short course of the extracranial portion of the trigeminal nerve, it may be difficult to appreciate subtle changes in size and MRI characteristics. Such changes may have been overlooked. Consequently, this dog was classified as extracranial. Effusion was only observed in dogs with lesions classified as brainstem or trigeminal canal. In total, 5 of 10 dogs classified as brainstem had effusion. Only 1 dog classified as trigeminal canal had effusion.

In all but 1 dog, clinicopathologic data were consistent with a unilateral lesion involving either the right side (7 dogs) or the left side (10 dogs). Signs consisted of unilateral atrophy of the muscles of mastication (17 dogs) and abnormal facial sensation (14 dogs). In the remaining dog, clinical signs were consistent with bilateral involvement based on absent facial sensation bilaterally. However, in this dog, MRI disclosed left-sided atrophy and contrast enhancement of the muscles of mastication and abnormal contrast enhancement of the left trigeminal nerve. In this case, the size of the left trigeminal nerve was subjectively larger in comparison to the other side, and effusion in the tympanic cavity was not present. In all but 2 dogs, the presumptive diagnosis was a nerve sheath neoplasm consistent with described characteristics such as trigeminal nerve enlargement and homogenous contrast enhancement that is greater in intensity than in the unaffected nerve.[3] In 1 of the 2 dogs, although the trigeminal nerve was abnormal on MRI, bilateral deficits were present, making a presumptive diagnosis of nerve sheath neoplasm difficult. In the other dog, the trigeminal nerve was normal on MRI. Nerve sheath neoplasm was definitively diagnosed at necropsy in one dog. In the remaining 2 dogs, a presumptive diagnosis was difficult to make. Although 1 dog had an enlarged trigeminal nerve that had abnormal enhancement, sensory deficits were present bilaterally. In the other dog in which there was not enlargement of the trigeminal nerve, a presumptive etiology was not identified. Neuritis was suspected.

Discussion

The purpose of this study was to determine the prevalence of effusion in the tympanic cavity in dogs with disorders of the trigeminal nerve. We identified 18 dogs with disorders involving the trigeminal nerve. The neuroanatomic diagnosis of trigeminal nerve dysfunction was based on the neurological examination disclosing atrophy of the muscles of mastication alone or in conjunction with sensory loss involving the cutaneous areas of the trigeminal nerve. In addition, all dogs, except for one, had an abnormality on MRI examination of the head involving the trigeminal nerve along with changes in the musculature innervated by the trigeminal nerve such as atrophy and contrast enhancement. In the sole dog without an abnormality of the trigeminal nerve on MRI, neurological examination was consistent with dysfunction of the trigeminal nerve. Effusion was identified based on tympanic cavity signal abnormalities as described in previous reports.[8] We observed effusion in the tympanic cavity ipsilateral to the trigeminal nerve disorder in 6 (33%) dogs. None of the dogs had effusion in the tympanic cavity contralateral to the side of the trigeminal nerve dysfunction, which lends support to a relationship between trigeminal nerve dysfunction and effusion in the tympanic cavity. We propose that effusion in the tympanic cavity was a consequence of ipsilateral trigeminal nerve dysfunction, which resulted in a loss of the normal function of the auditory tube.

In veterinary medicine, little attention has been paid to anatomy and function of the auditory tube.[8,10] As a result, the pathological consequences of dysfunction of the auditory tube remain less well known. Anatomically, the auditory tube is a conduit that connects the tympanic cavity to the nasopharynx. Its structure can be divided into three components: an osseous portion that opens into the tympanic cavity, a cartilaginous portion that opens into the nasopharynx, and a junctional portion that connects the osseous and cartilaginous portions.[11] Functionally, the auditory tube equalizes the pressure within the tympanic cavity with atmospheric pressure. Similarly, the auditory tube allows for drainage of secretions created by the epithelial lining of the tympanic cavity and auditory tube itself.[11] These functions are accomplished when the

normally closed nasopharyngeal orifice of the cartilaginous portion of the auditory tube is opened.

Logically, disorders that alter the anatomy or ability to open the nasopharyngeal orifice can result in dysfunction of the auditory tube. Without the opening of the nasopharyngeal orifice of the auditory tube, negative pressure builds up within the tympanic cavity, which results in transudation of fluid and development of effusion.[12] In dogs, variations in pharyngeal conformation can lead to impairment or obstruction of the nasopharyngeal orifice, which results in effusion in the tympanic cavity.[7,13,14] Primary secretory otitis media in the Cavalier King Charles Spaniel may be the consequence of a brachycephalic conformation, which may impair drainage through the auditory tube.[13] To the authors' knowledge, neuromuscular disturbance of the nasopharyngeal orifice has not been studied in the veterinary medicine. Normally, the nasopharyngeal orifice of the auditory tube opens during swallowing. This is accomplished by the muscles of the pharynx, including the TVP and the LVP muscles. The TVP muscle originates from the muscular process of the temporal bone. From its origin, it courses rostro-medially to insert on the hamulus of the pterygoid bone. Along its course, muscle fibers radiate medially into the soft palate. Innervation of the TVP muscle is provided by the nerve of the TVP, which is a branch of the mandibular branch of the trigeminal nerve.[15–17] The LVP muscle also originates from the muscular process, but it courses ventrocaudally to insert on the caudal half of the soft palate. The LVP muscle is innervated by the pharyngeal branch of the glossopharyngeal and vagus nerves with its motor neurons located in the nucleus ambiguous.[15,18]

The described function of the TVP muscle is to stretch the soft palate between the pterygoid bones.[19] In addition, contraction of the TVP muscle opens the nasopharyngeal orifice of the auditory tube.[20–25] In fact, it may be the only muscle to open the pharyngeal orifice of the auditory tube.[20–25] The described function of the LVP muscle is to elevate the soft palate.[19] It also dilates the lumen of the cartilaginous portion of the auditory tube.[26] Although not known with certainty, the LVP muscle also may work in concert with the TVP to open the nasopharyngeal orifice of the auditory tube.[20]

With experimental transection or transposition of the TVP muscle, effusion develops in the tympanic cavity.[22] Likewise, selective paralysis of the TVP muscle also results in effusion in the tympanic cavity.[23] However, selective loss of function of LVP muscle does not result in effusion.[27] In all of the dogs reported here, atrophy was observed in all muscles identified on MRI that were innervated by the mandibular branch of the trigeminal nerve. Moreover, in each dog a lesion involving a proximal segment of the trigeminal nerve before giving rise to the mandibular branch or involving the mandibular branch alone was observed. Based on these findings, dysfunction of the TVP likely also was present in all dogs in this study. However, given its size, the TVP muscle could not be identified on MRI. Effusion more often was observed in dogs with involvement at the level of the pons and medulla, which suggests that the axons that ultimately innervate the TVP may leave the trigeminal nerve proximally. Although speculative, based on experimental data selectively affecting the function of the TVP muscle,[22,23] we hypothesize that effusion in the tympanic cavity in the dogs in this report was a consequence of denervation of the TVP muscle. In the future, gross and microscopic evaluation of a dog with trigeminal nerve dysfunction and effusion in the tympanic cavity should be done to determine whether or not neurogenic atrophy of the TVP muscle exists.

Our study had several limitations. A definitive etiology for the trigeminal nerve disorders was not established in all cases. Many had a presumptive diagnosis of nerve sheath tumor and the signal change observed in the tympanic cavity may have represented neoplastic invasion. In 1 dog that underwent necropsy, neoplastic invasion of the tympanic cavity was not observed. Moreover, the MRI characteristics of the material in the tympanic cavities were very different than that of the abnormal trigeminal nerves. Typically, effusion in the tympanic cavity has MRI characteristics similar to that of water. With chronicity, there is reabsorption of water and an increase in the production of mucus which has high protein content. Given the loss of water and increased mucinous material, T1 relaxation times are shortened and therefore the material is hyperintense on T1WI. The material in the tympanic cavity was hyperintense in 5 dogs and isointense in 1 dog, which is most compatible with mucinous material. Likewise, abnormal contrast enhancement was not observed in 3 dogs and 3 dogs had peripheral contrast enhancement of the tympanic cavity, which was different than the abnormal contrast enhancement pattern of the affected nerves. Combined, these findings suggest that the material in the tympanic cavities was different than abnormal trigeminal nerves. A thorough otoscopic examination was not performed in all the animals. Consequently, otitis externa with extension into the tympanic cavity or otitis media were not definitively excluded underlying reasons for effusion in the tympanic cavity. Despite this, the medical records did not disclose information about the presence of otitis externa in any of the cases. As part of the physical examination, the ears of all dogs were evaluated but only the concave surface of the pinna and grossly visible portion of the external ear canal were evaluated and served as a surrogate for the determination of otitis externa. Moreover, the effusion when present was not evaluated cytologically for inflammation. Effusion itself could lead to inflammation and therefore identification of inflammation does not eliminate the possibility of its presence being related to auditory tube dysfunction secondary to denervation of the TVP muscle. In the present study, effusion could have been an incidental finding. Effusion in the tympanic cavity has been observed in 7% of dogs undergoing MRI examination of the head with approximately 2% of dogs being classified as unlikely to have otitis media based

on lack of evidence of otitis externa and incompatible clinical signs.[8] Likewise, the prevalence of effusion in the tympanic cavity of cats without clinical signs of otitis ranges between 1.7 and 11%.[10,28,29] Although our cohort was a small population, the greater number of animals having effusion in the tympanic cavity seems disparate from these reports of incidental finding of effusion. In further support of a causal relationship, effusion was observed in the tympanic cavity ipsilateral to the side with the trigeminal nerve dysfunction in all animals. Finally, evaluation of the ability of the affected animals to open the nasopharyngeal orifice of the auditory tube was not explored. Consequently, another pathophysiological explanation may underlie the presence of effusion. Despite these limitations, an alternative explanation for the number of animals with trigeminal nerve dysfunction and effusion in the ipsilateral tympanic cavity remains undefined.

In conclusion, none of the animals in the present study displayed a clinically evident adverse effect of effusion in the tympanic cavity. It is possible that, in time, dogs with effusion may develop secondary infection. However, without a clinically evident pathologic effect, there is little impetus to further examine the existence of a causal relationship between denervation of the TVP muscle and effusion in the tympanic cavity. Therefore any relationship will likely remain speculative based on available clinical data. It is important for clinicians, however, to be aware of the occurrence of effusion in the tympanic cavity in dogs with trigeminal nerve disorders. Lack of recognition may lead to an incorrect diagnosis or distract from the pursuit of treatment directed at the disorder involving the trigeminal nerve. In the future, it may be advisable to evaluate the effusion cytologically and by bacterial culture to determine if there also is a need to direct treatment at the effusion. In animals with septic effusion, appropriate antimicrobial therapy is warranted.

Footnote

[a]GE Healthcare, Waukesha, WI

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Conflict of Interest Declaration: Authors disclose no conflict of interest.

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