BRIEF COMMUNICATIONS and CASE REPORTS

Hypoglossal Neuritis with Associated Lingual Hemiplegia Secondary to Guttural Pouch Mycosis

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Guttural pouch mycosis in the horse is a pseudo-membranous inflammation of the diverticula of the Eustachian tubes usually caused by Aspergillus species. Inflammatory changes mostly occur in the dorsal part of the caudomedial compartment of the guttural pouch. They frequently erode the internal carotid artery, which passes with the superior cervical ganglion, vagal, and internal carotid nerves caudally along the medial compartment. Erosion of the condylar artery and cerebral vein, branches of occipital artery and vein, may occur when inflammation is localized dorsally in the caudomedial compartment. Inflammatory processes in the lateral compartment can lead to erosion of the maxillary artery, a branch of the external carotid artery. Thus, the most frequent clinical sign of guttural pouch mycosis is unilateral epistaxis. Other complications of this mycotic infection are inflammation of the atlanto-occipital joint, encephalitis, and osteitis of adjacent bones and pathologic fractures of the proximal end of the hyoid bone.

Neurologic complications of guttural pouch mycoses include laryngeal hemiplegia, caused by unilateral denervation atrophy of the laryngeal muscles due to degenerative lesions of the vagal nerve. Pharyngeal paralysis also can be caused by mycotic inflammation of glossopharyngeal and spinal accessory nerves because they pass along the lateral compartment of the guttural pouch. Clinical signs referable to the sympathetic nervous system lesions such as Horner’s syndrome, shivering, sweating, and colic are attributed to involvement of the superior cervical ganglion. Unilateral facial paralysis in connection with erosion of the facial nerve as it passes along the dorsal surface of the lateral compartment of the guttural pouch also has been documented.

Two cases of dysphagia in the horse due to unilateral lingual hemiplegia are described. This condition is due to hypoglossal neuritis secondary to the guttural pouch mycosis and represents a previously unreported complication of guttural pouch mycosis.

The first horse, a 12-year-old Standardbred gelding, had hypoglossal neuritis secondary to the guttural pouch mycosis.
Fig. 6. Hypoglossal nerve, cross section; horse No. 1. Immunocytochemical demonstration of S-100 protein in Schwann cells. Peroxidase-antiperoxidase technique, Papanicolaou’s solution 1b hematoxylin solution S counterstain. Bar = 50 μm.

Fig. 6a. Fascicle of hypoglossal nerve of nonaffected side showing Schwann cells associated with myelinated fibers. Fig. 6b. Persistence of dark staining Schwann cells in affected fascicle and extensive loss of myelinated fibers.
dysphagia for a period of 3 weeks accompanied by progressive weight loss and regurgitation. At necropsy, the animal was cachectic. The medial compartment of the left guttural pouch showed an ill-defined 5- x 2.5- x 1-cm area of pseudo-membranous inflammation situated in the caudal lateral area adjacent to hypoglossal nerve and internal carotid artery. The left half of the tongue was markedly indented (Fig. 1) because of severe atrophy of the lingual muscles (Fig. 2). The remaining organs were without significant macroscopic lesions.

The second horse, an 11-year-old Standardbred gelding, had a history of dysphagia, regurgitation, head shaking, and sneezing for a period of 5 weeks. Endoscopic examination revealed severe edema of the epiglottis and associated tissues and an accumulation of fluid within the right maxillary sinus. At necropsy, the right guttural pouch contained about 5 ml of sanguineous mucus. A 3- x 2- x 1-cm lesion, comparable to that in the first horse, was found in the caudal lateral area of the right medial compartment of the guttural pouch. The internal carotid artery was thrombosed for its full length and, with the hypoglossal nerve, was involved in the guttural pouch lesion. Purulent exudate was present in the right maxillary sinus. Tongue lesions of the right lingual half were identical with those in the first case. The remaining organs were without significant macroscopic lesions.

Aspergillus nidulans was isolated from material of the guttural pouch of horse No. 1.

For histologic examination, 5-10-µm sections of formalin-fixed, paraffin-embedded tissue specimens were stained with hematoxylin and eosin. In both cases, histologic examination revealed a deep chronic pseudo-membranous necrotizing inflammation of the guttural pouch with focal accumulation of fungal hyphae. Within this area, the internal carotid artery had a chronic obliterating thrombomarteritis with fungal hyphae in the media. A necrotizing and purulent neuritis of the hypoglossal nerve with fungal hyphae extending between myelinated nerve fibers was found adjacent to the site of the guttural pouch lesion (Figs. 3, 4). Parts of the nerve distal to this level, including its branches between the lingual muscles, showed degenerative changes consistent with Wallerian degeneration.

For immunocytochemical examination, 5-10-µm sections of paraffin-embedded tissue specimens were stained for neurofilament (monoclonal antibody, anti-human neurofilament protein, 70-kd and 200-kd component, clone 2F-11, Dako Diagnostica GmbH, Hamburg, Germany) and S-100 protein (rabbit anti-cow S-100 protein, Dako Diagnostica GmbH, Hamburg, Germany) by the peroxidase antiperoxidase technique and Papanicolaou’s solution 1h hematoxylin S solution counterstain. Intact hypoglossal nerve fascicles of the nonaffected side served as positive controls for both S-100 protein and neurofilament. Immunocytochemical demonstration of neurofilament indicated that only a few axons or parts of axons remained in the degenerate nerve (Fig. 5a, b). Only Schwann cells persisted within the nerve fibers when stained immunocytochemically for S-100 protein (Fig. 6a, b). Histologic examination of the lingual muscles of the affected side revealed a denervation atrophy of muscle fibers.

These case reports illustrate that guttural pouch mycosis can extend to and involve the hypoglossal nerve. Because the hypoglossal nerve innervates the lingual muscles, degenerative changes in this nerve may lead to irreversible unilateral lingual paralysis.

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References


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