Satellite Article

Diagnosis and management of guttural pouch mycosis

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History, structure and function of the guttural pouches

The equine guttural pouches were originally described in Lyon, France in 1764 by Claude Bourgelat, and since then their presence has been noted in a broad range of other mammals, from forest dwelling mice in South America to marine mammals, rhinoceros and bats (Turner 1850; Brandt 1863; Ellenberger and Baum 1943; Fraser and Purves 1960; Hinchcliffe and Pye 1960).

There have been recent advances in the understanding of the anatomy and physiological role of these structures (Figs 1–3) (Baptiste et al. 2000); however, the function of the guttural pouches remains a topic of hot debate. The current belief holds that the pouches act with the intracranial cavernous venous sinuses to cool the arterial blood en route to the brain, protecting this sensitive and vital organ from thermal shock.

The 2 guttural pouches are expansions of the eustachian tubes that connect the pharynx to the middle ear and are located side by side, on midline, dorsal to the pharynx, at the base of the skull. The rostral extent of the pouches is the basisphenoid bone, ventrally the pharynx, oesophagus and retropharyngeal lymph nodes, caudally the atlanto-occipital joint, laterally, the parotid and mandibular salivary glands and dorsally the petrous temporal bone, tympanic bulla and auditory meatus.

The pouches are separated by a thin membrane rostrally and by the longus capitis and rectus capitis ventralis muscles caudally.

The guttural pouches are accessed endoscopically through the pharynx via the guttural pouch ostia (also known as the plica salpingopharyngium). The stylohyoid bone separates each pouch into a large medial and smaller lateral compartment. A number of structures are found within the equine guttural pouches including cranial nerves IX, X, XI and XII (cranial nerves V, VII, VIII run in close proximity to the pouches and can be affected by guttural pouch disease) the cranial cervical ganglion, cervical sympathetic trunk, internal carotid, external carotid artery (and its branches, the caudal auricular artery, superficial temporal artery and maxillary artery) stylohyoid bone and temporohyoid joint.

Despite the risks involved in having guttural pouches, it is fair to say that their presence in the equine head offers the clinician a unique view of vital anatomical structures hidden from view in most mammals. The accessibility of the pouches within the respiratory tract probably leads to increased risk to these vital structures for the horse; nevertheless guttural pouch disease is comparatively rare and, as such, large evidence-based studies investigating disease and dysfunction are lacking.

Conditions affecting the equine guttural pouches include empyema, tympany and the topic of this article, mycosis.

Guttural pouch mycosis

Guttural pouch mycosis itself was first described by Rivolta (1868), who named the condition Gutturomyces equi and concluded that from the appearance of the fungus and the type of lesion that the organism responsible was an Aspergillus sp. fungus. In the years that followed, further cases of the disease were reported by a number of authors and studies of 22 and 32 horses affected by mycosis (Cook 1966, 1968; Cook et al. 1968) described clinical signs, treatment and outcome. Since then there has been a great deal of interest in this condition and many valuable contributions to the literature.

Clinical signs

Moderate to severe epistaxis (Fig 4), not associated with exercise, is by far the most common clinical sign and follows fungal erosion of the wall of the internal carotid (2/3 of cases) artery or the external carotid and maxillary artery (or its branches). A bloody mucoid discharge is common for several days following an acute haemorrhagic episode. Most affected horses have several episodes of moderate haemorrhage prior to a fatal bleed but this is not always the case.

The second most common presenting sign is dysphagia (Fig 5), following damage to the glossopteryngeal nerve and pharyngeal branch of the vagus nerve. Affected horses are dull, drool saliva and have food material present at the nares. In severe cases aspiration pneumonia develops. Damage to
other neurological structures within the pouches can lead to pharyngeal paralysis, laryngeal hemiplegia and Horner’s syndrome (Greet 1987). Affected horses may present with abnormal respiratory noise in the case of damage to the recurrent laryngeal nerve, and miosis, ptosis, enophthalmos and patchy sweating following damage to the cranial cervical ganglion.

In a number of horses, mucopurulent nasal discharge is the only obvious abnormality.

Other less commonly associated clinical signs include facial nerve paralysis, paralysis of the tongue, abnormal head carriage, head-shaking, sweating, colic, exposure keratitis and corneal ulceration, blindness and parotid pain following palpation at the base of the ear. Infection of the middle ear, osteitis of the stylohyoid and petrous temporal bones and infection (Dixon and Rowlands 1981) or arthropathy of the atlanto-occipital joint (Walmsley 1988) have also been reported as possible sequelae to guttural pouch mycosis.

**Aetiology**

A number of bacterial and fungal organisms are normal inhabitants of the equine guttural pouches. Guttural pouch mycosis is caused by many species of fungi (Lepage 1997; Ludwig et al. 2005), although *Aspergillus* spp. are implicated in the vast majority of cases. The mechanism of why this fungus, a normal inhabitant of the equine upper airway and environment, should become a pathogen in animals that are not debilitated or immunosuppressed is not understood. Obtaining positive cultures of early mycotic lesions is difficult and this is complicated by the fact that most horses are not sampled until they have clinical signs, by which time the lesion is at an advanced stage. Unfortunately the presence of serum antibodies to *Aspergillus* spp. detected by ELISA cannot differentiate between healthy horses and those affected by the disease.

Considerable debate exists as to why the fungus seems to confine itself to specific anatomical areas. The presence of concomitant disease, such as guttural pouch empyema, low oxygen tension, low light levels, variations in temperature and humidity have all been suggested although they lack credible evidence. The presence of an aneurysm on the affected artery leads to elimination of the fungus through haemorrhage or irreversible neurological signs. Treatment of guttural pouch mycosis has historically been divided into medical and surgical options, or in some cases, a combination of both. The goal of therapy is aimed at occlusion of the damaged artery and/or elimination of the fungal plaque using topical antifungal agents. It is postulated that occlusion of the affected artery leads to elimination of the fungus through removal of nutrients in the blood substrate on which the fungus is maintained (Lane 1989). However, cases of spontaneous regression of fungal plaques and fungal plaques in atypical locations that do not involve blood vessels (Freeman 1999) may suggest that this hypothesis is too simplistic a view of what is probably a complex, dynamic process (Baptiste 2004).

Therefore, choice of treatment modality may be affected by the area occupied by the fungus, economic factors or personal preference of the clinician involved.

A variety of agents have been used in the medical treatment of mycotic lesions including systemic thiabendazole, topical nystatin (Caron et al. 1987), natamycin (Greet 1987), miconazole (Lane 1980) and enilconazole and topical, or systemic iodides (Owen and McKelvey 1979; Sherlock et al. 2007). Response to such treatments is slow and it is generally accepted that medical treatments give unsatisfactory or unpredictable results (Speirs et al. 1995) and are not without risk to the horse; however, if the fungal plaque does not involve a blood vessel and/or there are financial restraints, medical therapy can be an effective option. It should be noted, however, that if the mycotic lesion is in the proximity of a blood vessel or neurological structure, fatal haemorrhage or

Horses presenting with epistaxis should be treated with extreme care. Endoscopy of the pharynx will often demonstrate blood draining from one of the guttural pouch ostia (Fig 6), and if surgical facilities are not close at hand, no attempt should be made to enter the guttural pouch itself as the endoscope may dislodge a clot, potentially leading to a fatal haemorrhage.

In horses displaying clinical signs of dysphagia, the nasal passages and pharynx may contain food material (Fig 7) and the dorsal wall (roof) of the pharynx is often collapsed. Permanent displacement of the soft palate may also be a feature.

If endoscopy of the affected pouch is performed, the mycotic lesion usually appears as a yellow/brown/white and black diphtheritic plaque, although the colour and appearance can be variable, on the roof of the affected pouch (Figs 8a–c). If there has been haemorrhage it is often difficult to identify any structures within the pouch and identification of the damaged vessel can be challenging. In some cases identification of haemorrhage within either the medial or lateral compartment may aid in identification of the damaged vessel.

Other endoscopic findings include fistulas between the pharynx and the pouches or between the pouches themselves and in rare cases adhesions (Fig 9) within the pouches.

**Treatment**

If left untreated guttural pouch mycosis may result in a fatal haemorrhage or irreversible neurological signs. Treatment of guttural pouch mycosis has historically been divided into medical and surgical options, or in some cases, a combination of both. The goal of therapy is aimed at occlusion of the damaged artery and/or elimination of the fungal plaque using topical antifungal agents. It is postulated that occlusion of the affected artery leads to elimination of the fungus through removal of nutrients in the blood substrate on which the fungus is maintained (Lane 1989). However, cases of spontaneous regression of fungal plaques and fungal plaques in atypical locations that do not involve blood vessels (Freeman 1999) may suggest that this hypothesis is too simplistic a view of what is probably a complex, dynamic process (Baptiste 2004).

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neurological damage are possible complications of these treatments, and horse owners should be warned of these possible sequelae prior to instigation of medical therapy alone.

A number of surgical techniques for the treatment of guttural pouch mycosis have been described, particularly for horses presenting with epistaxis or those with fungal lesions overlying blood vessels. Surgical removal of the fungal plaque has been described, although this treatment has led to fatal
haemorrhage and neurological signs (Johnson et al. 1973) and is therefore not recommended.

Ligation of the internal carotid artery at its origin, at the level of the carotid trifurcation in the parotid region (Johnson et al. 1973; Owen 1974; Greet 1987), although effective at reducing the risk of fatal epistaxis, is not an effective treatment option alone, because blood flow in the circle of Willis maintains arterial blood pressure in the affected segment of the artery, which can lead to retrograde fatal haemorrhage (Freeman and Donawick 1980a,b). For this reason a number of techniques were developed to occlude blood flow on both sides of the affected portion of artery. Open surgical access and ligation of the affected vessels is difficult or impossible, depending on the vessel affected, and can lead to complications through damage of other vital structures. As a result several closed surgical techniques have been developed including balloon tipped catheter placement, in which a catheter is passed into the affected blood vessel at a distant site, peripherally, and passed beyond the lesion, where the balloon is inflated, thereby occluding blood flow. A ligature is then placed on the cardiac side of the vessel where it is accessible at the level of the carotid trifurcation. If the maxillary artery in the lateral compartment of the guttural pouch is affected by a mycotic lesion, a balloon must be passed into the transverse facial artery on the lateral aspect of the face and orally into the palatine artery in order to occlude all possible sources of retrograde haemorrhage. The balloon catheter is then removed approximately 14 days after the initial surgical procedure (Freeman and Donawick 1980a,b; Freeman et al. 1989). Problems encountered with this technique include the presence of a catheter within the oral cavity, failure of the balloon section, catheter breakage, incisional complications, wound infections, prolonged anaesthetic periods, the requirement for a second surgery and the potential for a cosmetic blemish caused by the presence of the catheter subcutaneously (Leveille et al. 2000). A similar technique using catheters with detachable balloons does not require a second surgery (Cheramie et al. 1999).

More recently, occlusion of both sides of the affected vessels using the closed technique of transarterial coil embolisation of the internal and external carotid and maxillary arteries has been described (Matsuda et al. 1999; Leveille et al. 2000; Lepage and Piccot-Crezollet 2005). Intravascular platinum microcoils or Dacron, fibre-covered, stainless steel spring embolisation coils are passed into the affected vessel under fluoroscopic guidance, using contrast angiography (Fig 10), through an arterial catheter. All the surgical procedures described are technically demanding, and require specialised equipment and an excellent

**Fig 7:** Food material, rostral to the epiglottis, and haemorrhage emanating from the left guttural pouch ostium is noted in this horse which presented with a history of dysphagia and epistaxis.

**Fig 8:** The mycotic lesion usually appears as a yellow/brown/white and black diphtheritic plaque, although the colour and appearance can be variable, on the roof of the affected pouch.

**Fig 9:** Severe adhesions were noted in this guttural pouch, 6 months following successful treatment and resolution of guttural pouch mycosis.
knowledge of vascular anatomy. These techniques are further complicated by the fact that a percentage of horses exhibit variations in the vascular anatomy of the affected vessels, particularly the carotid trifurcation (Freeman et al. 1993). For this reason, the use of most of the surgical procedures described is confined to specialist practices.

Complications associated with surgical treatment include failure to prevent epistaxis and the development of neurological disorders and blindness following surgery. The mechanism for the development of these complications is not well understood and the incidence varies with the technique used, such that ligation techniques carry the highest risk (Freeman et al. 1990). Nevertheless owners should be warned of these potential complications prior to surgery.

**Outcome and prognosis**

Successful treatment of guttural pouch mycosis is dependant upon early recognition and prompt intervention by attending clinicians. Identification of cases presenting with clinical signs of epistaxis or fungal plaques overlying major vessels should be considered an emergency and the owners, trainers or keepers informed of the potentially fatal outcome. Affected animals should be moved to a suitable establishment where treatment is available.

Transarterial coil embolisation techniques are the currently accepted gold standard for treatment, and using this technique Lepage and Piccot-Crézollet (2005) reported a survival rate of 87%, with 71% of horses returning to a satisfactory level of exercise. Mycotic lesions generally show complete regression within 4 months of treatment with this technique.

A poor prognosis should be given if there are clinical signs of neurological disease, particularly dysphagia, prior to treatment. In some horses with neurological deficits, including evidence of left recurrent laryngeal nerve dysfunction and Horner’s syndrome, prior to treatment, complete resolution does occur but can take up to 18 months.

**Summary**

In summary guttural pouch mycosis is an uncommon but potentially fatal disease affecting horses of all ages, breeds and sex. The most common presenting signs include epistaxis and dysphagia. The condition is caused by invasion of the wall of one or both guttural pouches by *Aspergillus* spp. fungus, which leads to damage of vital vascular and neurological structures. Surgical treatments carry the highest and most predictable success rates and involve occlusion of the damaged blood vessels.

Further investigation into the causative agent is needed if we are to understand why this normal inhabitant of the airway becomes pathological in some horses, what the predisposing factors are and to allow early diagnosis to more effectively prevent fatal haemorrhage and the development of career and life-threatening neurological derangements.

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**References**


