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Pharyngeal Lymphoid Hyperplasia

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Pharyngeal lymphoid hyperplasia (PLH) is a condition in horses involving proliferation of one of the equine tonsillar structures. It has been diagnosed more frequently in the past few years due to a greater awareness of its existence, a developing prevalence of endoscopic examination, and/or an increased incidence of the syndrome. We will discuss the following in this paper as they relate to PLH:

1. The age and breed predilection
2. The equine tonsillar anatomy
3. The causative agents
4. The clinical signs and the differential diagnosis based on them
5. The endoscopic appearance
6. The treatments of PLH.

In a one and a half year case load study at New Bolton Center, 1,4576 horses were endoscopically examined. Pharyngeal lymphoid hyperplasia was diagnosed in 207, or 5% of the cases. The majority of these horses were between 2 and 4 years; 95% of them were standardbreds and thoroughbreds. However, PLH has been found in all breeds.

There are five tonsillar structures in the horse. They are:

1. The lingual tonsil consisting of follicles at the root of the tongue and in the area of the glossoepiglottic fold;
2. The palatine tonsil being a 2cm wide × 10 cm long, flat, follicular tonsil on the floor of the pharynx, extending caudally from the glossoepiglottal fold to the base of the epiglottis;
3. The tonsil of the soft palate being an oval, slightly elevated follicular tonsil located rostrally on the ventral surface of the soft palate;
4. The tubal tonsil being a triangular portion of lymphoid tissue usually found between the pharyngeal openings of the auditory tube and as solitary nodules in the lateral walls of the nasopharynx and the dorsal surface of the soft palate; and
5. The pharyngeal tonsil being an accumulation of tonsillar follicles at the caudal end of the nasal septum and in the vicinity of the choanae.

Of primary concern are the tubal tonsil and the pharyngeal tonsil. These lymphoid-filled follicles on the dorsum of the pharynx are most often seen in young horses, their number and size decreasing at 2 to 3 years of age, and only a few being present at 4 or 5 years.3 This age-related decrease in the amount of lymphoid tissue explains the predilection of PLH for 2 to 4 year old horses.

The pharyngeal tonsil is affected first when upper respiratory infections occur. This is due to 1) its common access to pathogens either inhaled from the nasopharynx or ingested through the oropharynx, and 2) its diffuse nature and large surface area which increases the likelihood that it may be traumatized by pathogens, air turbulence, or smoke inhalation.

The etiology is non-specific, including viral and bacterial agents, air turbulence, and caustic substances. The viral agents considered are Equine Herpes I (the rhinopneumonitis virus), Equine Herpes II (the LK virus), the rhinovirus, and in the past, the parainfluenza virus.4 However, a consistent clinical syndrome has not uniformly been associated with infection.7 Regardless, there is general concurrence that viral infections...
cause acute inflammation of the larynx which may become a chronic inflammation if the animal's training is continued and/or secondary bacterial invasion follows. Chronicity is achieved by the initial pharyngeal inflammation causing a decrease in the diameter of the air passage which in turn produces a greater negative pressure during inspiration. The outcome is a downward pull on the dorsal wall of the pharynx and an elevation in the soft palate—further narrowing the pharynx. The end result is an increased resistance, a change in the airflow pattern, and air turbulence which mechanically irritates the mucosa.

If bacterial invasion occurs, culture results in growth of *Streptococcus equi* or *Streptococcus zooepidemicus*. These bacteria are considered to be present as secondary invaders only, not as the etiologic agent.

In the initial stages the horse is febrile and presents with a nasal exudate. As it progresses the two most common complaints are 1) a marked reduction in exercise tolerance and 2) noise heard on inspiration—both leading to an unsatisfactory record in the racing animal. Differentials include a list of upper airway obstructions:

1. pharyngeal lymphoid hyperplasia
2. epiglottal entrapment
3. pharyngeal or subepiglottal cyst
4. retropharyngeal cyst
5. gutteral pouch infections
6. elongation or paresis of the soft palate
7. evagination of the cricotracheal ligament
8. pharyngeal paralysis
9. pharyngeal foreign body
10. tracheal collapse or stricture
11. neoplasms, etc.

To distinguish between these upper airway obstructions, the history should include the duration of the problem, the presence of odor or discharge, the severity of the signs, and the time of occurrence. Physical exam includes palpation and auscultation of the pharynx; watching and listening to the horse at work; rhinolaryngoscopy; and radiographs.

Endoscopy will be the most useful diagnostic aid. During the acute phase, the follicles will be edematous and hyperemic. Close observation reveals slight ulceration on the most prominent portion of the follicle. The accompanying nasal discharge is initially serous, then mucopurulent. In chronic cases, there is infiltration of fibrous connective tissue and the follicles appear thickened and fibrotic. Some may have lymphoid polyp formation on the pharyngeal wall or in the pharyngeal recess. New Bolton Center has developed a typing system used to classify the lymphoid proliferation, determined by number, size, appearance, and area of distribution.

Grade I—There are a few small, white lymphoid follicles scattered over the dorsal pharyngeal wall, inactive and shrunken. These are a common finding in horses less than 4 years of age and seldom affects their ventilation.

Grade II—There are many small white lymphoid follicles lying close together on the dorsal and lateral pharyngeal walls. Their lateral extent is the pharyngeal openings of the gutteral pouch or just slightly lower. Scattered among these white follicles are a few edematous, pink, active appearing follicles. Clinically, this grade may exhibit enough air resistance to cause increased air turbulence and initiate the development of chronic inflammatory lesions.

Grade III—Pink and white lymphoid follicles lie very close together, covering the dorsal pharynx, the lateral pharyngeal walls below the opening of the gutteral pouch, and even the dorsal surface of the soft palate. In summary, they cover all of the visible surface of the pharyngeal mucous membrane.

Grade IV—The follicles are large and edematous, covering the same area as Grade III plus extending into the gutteral pouches. Numerous polyps may arise from the pharyngeal diverticulum or the dorsal and lateral walls. Clinical signs are usually associated with Grades III and IV but occasionally may not if the diameter of the airway is sufficient to accommodate both the hyperplasia and adequate air. For this reason, it is important not to diagnose pharyngeal hyperplasia on endoscopic findings alone. A thorough examination, especially of the respiratory and cardiopulmonary systems should be done.

**TREATMENT**

The treatment of this condition is symp-
tomatic, the goal being the reduction in the number and size of lymphoid follicles. There are four regimens of treatment: 1) rest, 2) medical consisting of steroids, anti-inflammatory agents, and antibiotics, 3) cauterization using chemical, electrical, or radioactive agents, and 4) immune therapy consisting of repeated vaccination with equine flu and rhinopneumonitis vaccines in an effort to increase local immunity. Follicle development often recurs following withdrawal of therapy or initiation of full work.

In summary, pharyngeal follicular hyperplasia involves the proliferation primarily of one of the five tonsillar structures in the equine. Its anatomical placement and diffuse nature predisposes the pharyngeal tonsil to inflammation. This inflammation begins acutely due to viral agents and progresses to a chronic nature by the addition of air turbulence and mechanical irritation, secondary bacterial invaders, and caustic agents such as smoke inhalation. Clinically, the most common signs are inspiratory dyspnea and exercise intolerance. Endoscopic exam reveals varying grades of hyperplasia, from a few scattered, white follicles to hyperemic, edematous follicles and polyps involving the entire visible pharyngeal mucosa. The methods of treatment include rest, medical suppression of the inflammatory response, stimulation of the immune system, and removal of the hyperplastic tissue with cauterization. The choice of therapy will depend on the use of the animal and the urgency for correction of the situation. No one mode of therapy has consistently provided favorable results. However the majority of cases undergo an age related regression of the hyperplastic lymphoid tissue as the immune system matures.

REFERENCES