Update on *Streptococcus equi* subsp *equi* Infections

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KEYWORDS

• Lymphadenopathy • Guttural pouch • Empyema • Strangles
• *Streptococcus equi* subsp *equi*

KEY POINTS

- The classic form of strangles includes fever and lymphadenopathy that develop within 3 to 14 days of initial exposure. Less common complications include metastatic abscesses, purpura hemorrhagica (PH), myositis, and other immune-mediated conditions.
- Samples (purulent debris or nasopharyngeal wash samples) should be submitted for both routine culture and polymerase chain reaction (PCR) testing to provide the highest diagnostic sensitivity.
- Chronically infected horses, with no outward clinical signs, are a likely source of new strangles outbreaks. Identification of these horses requires screening with upper airway endoscopy.
- Application of good biosecurity measures is integral to the successful resolution of a strangles outbreak.

PATHOGENESIS AND EPIDEMIOLOGY

The clinical syndrome referred to as strangles is caused by infection with *Streptococcus equi* subsp *equi*, a β-hemolytic, Lancefield group C *Streptococcus*. Unlike other *Streptococcus* sp (and particularly *S equi* subsp *zooepidemicus*), this gram-positive agent is not considered a normal commensal in the equine respiratory tract and is generally associated with disease.

Several microbiologic traits of *S equi* subsp *equi* contribute to its pathogenicity. Unlike many other β-hemolytic *Streptococcus* sp, *S equi* subsp *equi* is able to evade phagocytosis. This avoidance is specifically associated with a hyaluronic acid capsule and SeM surface protein.[1,2] Several cell surface antigens are also thought to contribute to virulence and can assist with serologic diagnosis. Significant experimental efforts have been directed toward leveraging these various cell surface antigens for better diagnostic testing.

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Successful infection begins with bacterial entry to the oral or nasal passage of the horse. Bacteria access pharyngeal tonsillar tissue and directly colonize deeper tissue via this location. Bacterial cell surface antigens mediate the entry of bacteria into tonsillar epithelial cells. Within a few hours of initial colonization, *S equi* subsp *equi* is no longer evident on the epithelial tissue but can be found within subepithelial cells and the lymph nodes responsible for draining the pharyngeal region.[3]

Bacterial arrival into the local lymph nodes stimulates an influx of neutrophils, but because of evasion of phagocytosis these cells are generally unable to prevent bacterial multiplication and colonization of the node. These accumulated neutrophils eventually contribute to the typical lymph node abscesses seen with the disease. Streptolysin S and streptokinase also seem to contribute to cell membrane damage and the ultimate formation of abscesses.[4] Although uncommon, distant or metastatic infection and abscessation can occur after lymphatic or hematogenous spread.

The time delay between exposure and initial colonization of local lymph nodes is short. Fever develops within 3 to 14 days of exposure. Bacteremia has been demonstrated for 6 to 12 days after experimental infection with a virulent strain.[5] After the onset of fever, nasal shedding can be expected within 2 to 3 days and typically persists for 2 to 3 weeks. Shedding can continue beyond this point, particularly in animals that develop an infection of the guttural pouch (GP).

Although relatively few organisms are present at the time of initial colonization, there is evidence of substantial bacterial propagation by the time of onset of fever. Abscessation and subsequent rupture of the abscesses allows for easy contamination of the environment and infection of other horses.

CLINICAL SYNDROME

The classic signs of strangles include fever and lymphadenopathy, which develop within 3 to 14 days of initial exposure to disease. Submandibular and retropharyngeal lymph nodes are most frequently affected, but any node in the head and neck is theoretically susceptible. Periorbital swellings are reported, and involvement of the lymph nodes at the thoracic inlet may result in substantial and dangerous restriction of tracheal airflow. Lymphadenopathy progresses to abscesses that, if allowed to mature, generally rupture and drain a tenacious purulent material. Depending on the direction of rupture (retropharyngeal lymph nodes can drain into the GP or pharynx) purulent material may drain via the nasal passages or directly externally. Retropharyngeal lymph nodes that drain into the GP may also establish a chronic empyema. Ocular discharge may also be present. The occasional horse develops such significant lymphadenopathy that pharyngeal or tracheal airflow is restricted.

Fever typically occurs before maturation and rupture of abscesses and resolves after the establishment of drainage. Some horses become depressed and inappetant at the initial stage of infection; this is most likely because of fever, but dysphagia can also occur. Cough is occasionally present and may be worsened by secondary pharyngitis or laryngitis.

Routine blood testing is not required for straightforward cases of strangles. Complete blood cell count typically reveals a hyperfibrinogenemia and mature neutrophilia.[6] Blood chemistry is generally unremarkable.

Complications

Most strangles cases progress as described above and resolve within several weeks after rupture of the abscessed lymph nodes. However, various sequelae can occur