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Streptococcus equi subsp. *equi* (Strangles) Infection

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***Streptococcus equi* subsp. *equi* infection (strangles) is an acute, contagious upper respiratory tract disease of predominantly young horses characterized by rhinopharyngitis and lymphadenitis of the submandibular and retropharyngeal lymph nodes. *S. equi* infects the host by attaching to tonsillar epithelium after inhalation or ingestion of infected secretions. Several components of the bacterium, including the capsular M-protein, contribute to virulence and immunogenicity. Nasopharyngeal mucosal and systemic immune responses are important for recovery, and most naturally infected horses become solidly immune for years thereafter. Definitive diagnosis is made by culture or polymerase chain reaction testing of nasal swabs, abscess aspirates, or guttural pouch lavages. Treatment is dependent on the stage and severity of disease. Inapparent carrier horses play an important role in persistence of *S. equi* and its transmission to susceptible populations. Control and prevention depend on isolation of affected horses, appropriate sanitation, identification of carriers, and judicious use of vaccines. Complications from *S. equi* infection include guttural pouch empyema, internal abscesses, purpura hemorrhagica, and myositis.**
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Streptococcus equi subsp. *equi* infection is an acute, contagious upper respiratory tract disease of Equidae that typically affects young animals and is characterized by mucopurulent inflammation of the nasal passages, pharynx, and associated lymph nodes. The disease is also referred to as "strangles" (suffocation), to reflect compression of the upper airway caused by enlargement of the retropharyngeal lymph nodes in severe cases. Infrequent complications of the disease include guttural pouch empyema, internal spread of the bacteria, and immune-mediated vasculitis.

Etiology

Streptococcus equi subsp. *equi* is a Lancefield's group C Gram-positive bacterium that forms long chains of irregularly shaped cocci. The most common isolate is encapsulated, highly virulent, and makes honey-colored mucoid colonies with a wide zone of hemolysis on blood agar. An atypical variant is less encapsulated, less virulent, and produces a matte appearance on blood agar. The M-protein (SeM) found within the bacterial capsule is highly immunogenic, inhibits phagocytosis by neutrophils and macrophages, and prevents opsonization by inhibiting complement deposition.[1-3] Capsular hyaluronic acid is also antiphagocytic and contributes to virulence. *S. equi* produces several toxins that inhibit host cell response, including hemolysins (streptolysins), hyaluronidase, leukocytotoxins, and mitogens that contribute to disease severity.[4-8]

Pathogenesis

Infection occurs by inhalation or ingestion of the organism, followed by attachment to the cells in the crypt of the tonsil and adjacent lymphoid nodules.[8] The hyaluronic acid capsule and SeM, in addition to fibronectin-binding proteins, facilitate adherence to host cells.[9,10] The bacteria release enzymes and toxins that damage surrounding cells and initiate inflammation, causing fever, rhinitis, and pharyngitis. The inhibition of C3 complement deposition onto the bacterium by SeM inhibits opsonization, which is critical for clearance of the organism.[1] The bacteria then translocate within a few hours to the submandibular and retropharyngeal lymph nodes that drain the pharyngeal/tonsillar region. Although neutrophil chemotaxis in response to bacterial peptidoglycan is decreased due to SeM-mediated complement inactivation, a significant number of neutrophils are recruited into the lymph nodes.[1] They are, however, unable to effectively phagocytose and kill the *S. equi* organisms, due to the antiphagocytic properties of SeM and hyaluronic acid capsule, and the organization of bacteria in chains. This results in accumulation of extracellular organisms, degenerating neutrophils, and necrotic tissue (abscess) in the lymph nodes.

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Rarely, lymphatic or hematogenous spread of bacteria occurs during the early, febrile stage and results in "bastard strangles," which refers to abscessing of lymphoid tissue or other organs beyond the head and neck (see complications). Most internal *S. equi* abscesses are located in the abdomen or thorax, although involvement of other locations has been reported, including four cases of brain abscesses associated with *S. equi* infection.[11] Guttural pouch empyema can occur secondary to rupture of retropharyngeal abscesses into the pouch or from local extension of infection from the pharynx through the pharyngeal openings of the guttural pouch.

Epidemiology

S. equi infection has been referenced since the 13th century in Europe and was described in 1888.[12,13] The disease has since been prevalent worldwide. In a susceptible population, yearlings and young adults are most at risk, followed by weanlings and then adults.[14,15] Typically, yearlings are the most severely affected with a longer duration of clinical signs.[15] Foals up to 3 months of age born to immune dams are usually protected.[16] The morbidity is 85% to 100% in susceptible populations, and the mortality is 4% to 8%.[14,15] Predisposing factors for disease transmission include overcrowding, mixing of horses from different areas, and various "stressors," including weaning, traveling, severe weather, concurrent illness, and improper nutrition.[17,18] The disease can become enzootic on properties with multiple, ongoing risk factors. A strangles outbreak may last up to 4 to 6 months on a farm with an inadequate isolation protocol and a highly susceptible population.[15]

Unlike *S. equi* subsp. *zooepidemicus*, *S. equi* is not part of the normal nasopharyngeal bacterial flora. Most infected horses begin shedding *S. equi* 2 to 3 days after the onset of fever and continue shedding for up to 4 weeks after resolution of clinical signs.[4,17] Infected secretions can be transmitted directly or via fomites (feeders, water buckets, tack, handlers, flies, etc.). A small but significant proportion (1% to 10%) of infected horses fail to clear the organism within 4 weeks and continue to shed the *S. equi* in nasal secretions for a prolonged period. Shedding has been documented for up to 39 months after resolution of clinical signs,[19,20] and can contribute significantly to outbreaks in naïve horses on well-managed farms. Most of these carriers harbor the organism in the guttural pouch, which may or may not be evident externally or on endoscopic examination of the guttural pouches.[19,21,22] If the purulent material becomes inspissated in the form of chondroid masses, it is unlikely to drain from the guttural pouches. The organism does not survive for a prolonged period in the environment because of its susceptibility to heat, sunlight, dessication, and many disinfectants, including povidone iodine, chlorhexidine, and glutaraldehyde.[4,16] Although survival of *S. equi* in a laboratory setting for up to 9 weeks has been reported,[23] it is unlikely that the organism persists in the environment on typical equine facilities for more than a few weeks.

Immunology

Production of local antibody directed against the SeM is thought to be the most important protective immune response to *S. equi* infection, although systemic IgG appears to confer some protection.[24,25] Nasopharyngeal mucosal IgA and IgG levels are high following natural infection[24] and, regardless of systemic antibody levels, immunity is excellent following natural infection in most horses.[26] Approximately 75% of horses are protected for at least 4 years following natural infection,[13,24] whereas 25% fail to mount an appropriate immune response and are susceptible to reinfection within 6 to 12 months.[27] Most foals born to immune mares are protected for up to three months, as a result of passively acquired *S.equi*-specific IgG in their serum and IgA on their nasopharyngeal mucosa. Colostral antibodies specific to SeM coat the oropharyngeal and nasopharyngeal mucosa during suckling and IgA can also redistribute hematogenously to the nasopharyngeal mucosa after being absorbed from the gastrointestinal tract.[28] Although SeM has been shown to be highly antigenic, other bacterial proteins may also stimulate production of antibodies that may be involved in the protective immune response.[29]

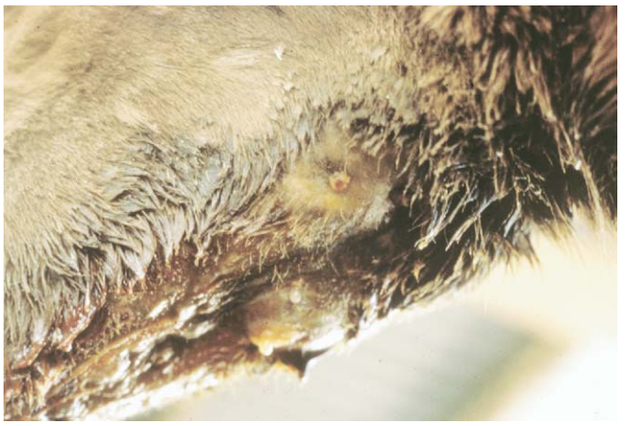

Figure 1 Ruptured and draining submandibular lymph node abscesses. (Color version of figure is available online.)

Clinical Signs

Signs become apparent after an incubation period of 3 to 8 days, and the clinical course usually lasts 3 to 4 weeks.[15,17,30,31] Marked fever (103-106°F) develops during the acute phase and may subside until the lymph nodes abscess, at which time a second wave of fever may develop. Affected horses typically become anorexic, depressed, and develop bilateral, serous to mucoid nasal discharge within 24 hours of onset of fever. The discharge becomes mucopurulent as the disease progresses, and a moist cough may develop in some cases. Plasma fibrinogen concentration and leukocyte counts usually increase at this time. The submandibular lymph nodes are involved most often and become enlarged, firm, and painful (Fig. 1). The retropharyngeal lymph nodes may also be affected, and can induce dysphagia if they become markedly enlarged. The abscessed lymph nodes typically rupture 7 to 10 days after the onset of clinical signs and, in uncomplicated cases, recovery is complete 1 to 2 weeks thereafter.[16] A short course of illness with mild fever and slight nasal discharge may develop in horses infected with less virulent atypical *S. equi* variants.

If abscesses in the retropharyngeal lymph nodes or other

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