STRANGLES (EQUINE DISTEMPER)

ETIOLOGY

. Streptococcus equi subsp. equi (S. equi) is a Gram-positive coccobacillus that produces a beta-hemolysin , Strangles occurs in horses, donkeys, and mules worldwide, Strangles can affect horses of any age, although the morbidity rate is usually greater in younger horses such as foals and weanlings.

Source of infection and transmission

1- Nasal and abscess discharge from infected animals that contaminates pasture.
2- grooming equipment, and hands and clothes of grooms and veterinarians is often the source of infection for susceptible horses.
3- Direct transmission from infected animals to susceptible animals occurs through contact.
4- Horses with clinically inapparent disease, such as some cases of guttural pouch empyema, may shed the organism for over 3 years

Pathogenesis

Virulence of S. equi is attributable to the presence of M proteins on the surface of the bacteria associated with adhesion and invasion the tissue, a hyaluronic acid capsule and the production of a leukocidal toxin.

The capsule of S. equi is associated with resistance to non-immune phagocytosis and pathogenicity. Strains of S. equi that do not produce a capsule do not induce disease.

Following exposure of the oral and nasopharyngeal mucosal surfaces to S. equi, bacteria lodge in the pharyngeal and tonsillar lymphoid tissues where they multiply rapidly, Release of streptolysin S and streptokinase

may contribute to tissue damage by directly injuring cell membranes and indirectly through activation of plasminogen. Bacteremia may occur. Migration of neutrophils into the lymph nodes causes swelling and abscessation, with associated disruption of lymph drainage and development of edema in tissues drained by the affected nodes. Swelling of retropharyngeal lymph nodes may interfere with deglutition and respiration. Nasal shedding of S. equi usually begins 4-7 days after infection, or 2 days after onset of fever, and persists for 2-3 weeks in most horses, Death is usually due to pneumonia caused by aspiration of infected material, other causes of death include asphyxiation secondary to upper airway swelling and impairment of organ function by metastatic infection.

Metastatic infection of the heart valves, brain, eyes, joints, and tendon sheaths or other vital organs may occur and cause a chronic illness and eventual death. Metastatic infection may occur because of bacteremia or extension of infection along chains of lymph nodes. Purpura hemorrhagica may occur as a sequela to S. equi infection and is associated with high serum antibody titers to SeM.

CLINICAL FINDINGS

Acute disease

The acute disease is characterized by

1- mucopurulent nasal discharge and abscessation of submandibular and retropharyngeal lynlph nodes.
2- After an incubation period of 1-3 weeks the disease develops suddenly with complete anorexia, depression, fever (39.5-40 C)
3- a serous nasal discharge, which rapidly becomes copious and purulent.

4- Lymphadenopathy becomes apparent as the submandibular lymph nodes enlarge and palpation elicits a painful response.
5- The pharyngitis may be so severe that the animal is unable to swallow and there is a soft, moist cough. The head may be extended.
6- The affected nodes become hot, swollen, and painful.
7- Swelling of the retropharyngeal lymph nodes may cause obstruction of the oro- and nasopharynx with subsequent respiratory distress and dysphagia.
8- If the infection is particularly severe, many other lymph nodes, including the pharyngeal, submaxillary, parotid and retro bulbar nodes, may abscess at the same time.
9- Death by asphyxiation may occur at this time in severe cases.

Complications

1- **Suppurative necrotic bronchopneumonia**, which pro-bably occurs secondary to the aspiration of pus from ruptured abscesses in the upper airway, or metastatic infection of the lung.
2- Extension of the infection into the gut-tural pouches, usu ally as a result of rupthe medial comparhnent, causes empyema.
3- Metastatic infection ('bastard strangles) results in the formation of abscesses in any organ or body site but most commonly in the lungs, mesenteric lymph nodes, liver, spleen, kidneys, and brain.
4- Purpura hemorrhagica can occur as a sequela to S. equi infection deposition of complexes of antigen and immunoglobulin in the walls of capillaries and small blood vessels. The disease appears to be immune complex-mediated and due to a type III hypersensitivity reaction. The common association of the disease is with Strep. equi infection of the upper respiratory tract. The high

concentrations of antibodies to Strep. equi M protein in affected horses, and the presence of complexes of IgA and streptococcal M protein in sera are evidence that the disease is associated with an immune reaction to streptococcal protein.

5- Two myopathic syndromes occur with S. equi infection in horses.26 Muscle infarction, which may be extensive, is assumed to result from immune-mediated vasculitis associated with purpura hemorrhagica. Rhabdomyolysis and subsequent muscle atrophy.

CLINICAL PATHOLOGY

1- Hematological abnormalities during the acute phase of the disease include leukocytosis with a neutrophilia reaching a peak as the lymph nodes abscess.
2- Hyperfibrinogenemia is characteristic of both the acute and chronic disease.
3- Leukocytosis with a hyperproteinemia in Metastatic and chronic phase.
4- A commercial test that measures the serum IgG antibody titer to SeM has been developed and has been used to determine response to vaccination, Slutability for vaccination and presence of metastatic infection.
5- PCR testing is useful to detect shedding of S. equi DNA. The test is reported to be more specific than culrure for detection of S. equi shedding.
6- Culture of nasal, pharyngeal, guttural pouch, or abscess discharge will usually yield S. equi in horses with active disease or in carriers.

Samples for confirmation of d iagnosis

Bacteriology - swab of abscess wall, enlarged lymph node (CULT) .

DIAGNOSTIC CONFIRMATION

Confirmation of strangles depends on the isolation of S. equi from nasopharyngeal I swabs and discharges from abscesses.

Differential Diagnoses:

• Epizootic Lymphangitis
• Ulcerative Lymphangitis
• Dourine
• Melioidosis
• Equine Viral Rhinopneumonitis
• Equine Influenza
• Equine Viral Enteritis

TREATMENT

1- The environment for clinically ill horses should be warm, dry, and dust-free.
2- Warm compresses are applied to sites of lymphadenopathy to facilitate maturation of abscesses.
3- Facilitated drainage of mature abscesses will speed recovery. Ruptured abscesses should be flushed with dilute (3%–5%) povidone-iodine solution for several days until discharge ceases.
4- NSAIDs can be administered judiciously to reduce pain and fever and to improve appetite in horses with fulminant clinical disease Non-steroidal anti-inflammatory drugs (phenylbutazone 2.2 mg/kg orally or IV every 12 hours, or flunixin meglumine 1.1 mg/kg

orally or IV every 12 hours) may reduce inflammation and providesome analgesia..

5- Tracheotomy may be required in horses with retropharyngeal abscessation and pharyngeal compression.

6- The specific treatment of choice for S. equi infection of horses is penicillin procaine penicillin G (22 000 IV/kg intramuscularly every 12 h) or potaSSium or sodium penicillin G (22 000 IV/kg intravenously every 6 h).

7- Tetracycline (6.6 mglkg intravenously every 12-24 h) and sulfonamide-trimethoprim combinations (15-30 mg/kg orally or intravenously every 12 h) may be efficacious but should only be used if penicillin cannot be administered.

8- Treatment the Purpura hemorrhagica by corticosteroids such as dexamethasone (0.05-0.2 mg/kg, IV or 1M every 24 hours) or prednisolone (0.5-1 mg/kg, 1M or IV every 24 hours) .