

## A Review on Equine Strangles

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**Abstract:** Strangles is one of the most common equine respiratory diseases and the most common bacterial infection of horses caused by a bacterium *Streptococcus equi subspecies equi*. The bacterium typically infects the upper airway and lymph nodes of the head and neck. The infection is highly contagious in horse populations and can recur on farms with previous outbreaks. It is one of the most commonly diagnosed and economically important contagious diseases of the horse worldwide. The primary source of recurrent infections is most likely asymptomatic carrier horses. Strangles is transmitted by direct contact from infected horse and indirectly a horse can be exposed to the bacterium on tack, feed, water buckets, or on brushes that have been contaminated by infected horse. Although strangles is not generally fatal, there are the occasional complications that can lead to death like bastard strangles, purpura hemorrhagica and pneumonia. Infected horses show variable clinical signs depending on the stage and severity of the infection. Common signs include: Copious nasal discharge, decreased appetite, fever, listlessness and swelling of the lymph nodes in the neck/throat area. The diagnosis of strangles is based on the clinical signs like fever, nasal discharge and lymph node swelling, the isolation of *S. equiequi* through the culture of nasal swab, nasal lavage and pus from abscess or lymphoid tissues (Cult) and PCR and serological tests. Strangles is treated with penicillin with good supportive treatments. Vaccination and hygiene are the most important tools when guarding against strangles.

**Key words:** Equine • Strangles • *Streptococcus equisub spp* • *Equi*

### INTRODUCTION

Strangles is a highly contagious and serious disease of horses and other equids caused by the gram positive, pyogenic and haemolytic bacterium, *Streptococcus equisub spp. equi* (*S. equiequi*). The disease is characterized by severe inflammation of the mucosa of the head and throat region with extensive swelling and often ruptures of lymph nodes [1].

Strangles was described in early veterinary science literatures. It was first reported by Jordanus Ruffus in 1251 [2]. The name strangles was given by Bourgelat in 1791 [2]. In the 19<sup>th</sup> century, many researchers focused on the bacteriological origin and the contagious nature of strangles. Inoculation of fresh cultures by Lowenthal has been demonstrated exclusively that *Streptococcus equisub spp. equi* was the causative agent of strangles [3].

Strangles is an epizootic disease of young equids and is worldwide in distribution. Strangles occurs as outbreak when the movement of horses in and out of the herd

increases and when the climate is cold. The mortality rate reaches up to 10% with the morbidity rate reaching up to 100% [4].

*S. equiequi* is an obligate bacterium of horses' upper respiratory tract and all infections are attributable to transmission from infected horses either directly from horse to horse contact or indirectly from fomites. Transmission from fomites occurs by inhalation or ingestion [5].

The early clinical signs of the disease are fever, nasal discharge and lymph node swelling. The lymph nodes below the jaw and neck become enlarged, hot and painful. As these lymph nodes abscessate, they may begin to ooze and eventually rupture and drain externally or internally [6]. Chronic infection of the guttural pouches can lead to drying of the pus and formation of solid, concretions called chondroids [7]. In complicated cases of strangles, several conditions can be developed including pneumonia, guttural pouch empyema, bastard strangles and purpura hemorrhagica (Small hemorrhage in the skin).

In complicated cases of strangles, the lymph node swelling may exert pressure on the esophagus and pharynx which results in difficulty swallowing. If the lymph node swelling obstructs the airway, the horse will develop difficult respiratory and require emergency attention, hence the origin of the term “Strangles” [8].

Diagnosis and treatment of strangles depends on history of exposure and clinical signs like fever, nasal discharges and lymphadenopathy. Isolation of the bacteria from nasal discharges, pus from abscess and lymphoid tissues by culture or PCR is the most effective diagnosis for strangles. Detection of the serum protein M antibodies by ELISA is also possible to diagnose strangles [5]. Strangles is treated with penicillin although treatment is rendered by the stage, feature and severity of the disease. Supportive treatments with penicillin are effective when the disease is painful and complicated [9].

Control and prevention of strangles can be achieved by reducing the movement of horses in and out of the herd, quarantine of the newly introduced horses, vaccination, cleaning and disinfection of housings, utensils, pastures and grooming brushes. Personnel should wear protective boots and clothes to avoid contamination [2].

Strangle is one of the most economically important infectious disease of horses. This is because of its effect on equine production as well in agriculture indirectly [1]. Therefore, the objective of this seminar is: to review the epidemiology, diagnosis, treatment, control options and the economic importance of equine strangles.

**Etiology:** Strangles is a highly contagious and serious infection of horses and other equids caused by a gram positive, hemolytic, pyogenic and lancefield group C bacterium, *Streptococcus equisubsp.equi* from genus streptococcus. It is characterized by severe inflammation of the mucosa of upper respiratory tract with extensive swelling of retropharyngeal and submandibular lymph nodes and often ruptures of lymph nodes produces large amount of thick creamy pus [1, 5].

**Morphology, Growth and Staining Characteristics:** *S.equiequi* is spherical in shape and 0.8 to 1mm in diameter. It has the appearance of chain during division and it is encapsulated. It is gram positive. *S.equiequi* has fairly exacting growth needs media containing blood or serum. After 24 hours of incubation at 37°C, it produces clear and mucoid colonies usually less than 4mm in diameter. *S.equiequi* is a catalase negative and facultative anaerobic bacterium [10].

## Epidemiology

**Occurrence:** Strangles occurs mostly in horses and rarely in donkeys and mules worldwide. Outbreak is seen frequently on breeding farms, in racing stables and in horses taken to fairs and riding schools when the infection is introduced by new arrivals that are often asymptomatic [1]. The disease affects mostly young horses (Three months to five years). Outbreak can occur at any time of the year but most likely to happen in cold weather [11].

In Ethiopia, horse taxi owners 48% reported respiratory disease (Strangles), mid rift valley 79% horse cart owners reported respiratory disease as a major constraint [12].

Sources of infection and transmission: *S. equiequi* is an obligate bacterium of horses and all infections are attributable to transmission from infected horses. Nasal and abscess discharges from infected horses are the most common sources of infection [13].

Horses that recover from the clinical disease may have persistent infection of *S.equiequi* in pharynx and guttural pouches for many months and horses with clinically inapparent disease are sources of infection [14]. Environmental existence of the agent is limited but in water and scalding it may exist for some weeks to months [15].

Transmission occurs through contact with purulent nasal discharge from horses with active and recovering strangles. Direct or indirect transmission can occur. Direct transmission involves horse to horse contact, nose to nose touching or coughing of infected secretions into the next stall [16]. Indirect transmission involves sharing of contaminated housings, water sources, feed or feeding utensils, tack, or handlers and their clothing. Apparently, healthy animals may transmit the disease either during the incubation phase or during the recovery phase [8].

## Risk Factors

**Animal Risk Factors:** strangles is more common in young (Three months to five years) and naïve horses although the disease occurs in any age group. Horses that have previously infected are less likely to develop disease on subsequent exposure. Resistance to the disease is associated with the production of serum and mucosal IgG antibodies to the streptococcal M protein [1]. The movement of horses has more influence on the occurrence of outbreaks. Horses recovered from strangles and clinically inapparent horses are sources for infection [17].

**Agent Risk Factor:** a variety of virulence factors like a non-antigenic hyaluronic acid capsule, streptolysin S, streptokinase, pyogenic mitogens and surface protein M of the agent are involved to induce a disease. These virulence factors play a role in agent attachment, killing of neutrophils and production of inflammatory reaction [18].

**Environmental and Climatic Factors:** Environmental persistence of *S. equiequi* is rare because it is sensitive to bactericin from environmental bacteria and is less competent with other bacteria but in water and scalding it persists for weeks to months. Outbreak occurs most likely in cold weather [4, 18].

**Pathogenesis:** *S. equiequi* enters via the mouth or nose and attaches to tonsil crypt cells and adjacent lymphoid nodules with the help of M protein, fibronectin binding protein, fibrinogenbinding protein and hyaluronic acid capsule [19]. Adherence triggers internalization and subsequent localization in the subepithelial spaces. Pyogenic exotoxins initiate an acute inflammatory response. Capsule and protein M protect *S. equiequi* from opsonization and phagocytosis. Streptolysin S and streptokinase also contribute to abscess development and lyses of host cells. Migration of neutrophils in to lymph nodes causes swelling and abscessation [18].

Although strangles predominantly involves upper airways and associated lymph nodes, metastasis to other body parts through hematogenous or lymphatic channels occurs and results abscess in lymph nodes and other organs of thorax and abdomen [15].

Nasal shedding of *S. equiequi* usually begins after 4-14 days of infection and ceases between three and six weeks after the acute phase but in some horses shedding may continue to harbor infection in their guttural pouches after clinical recovery which can be a source of contagion for susceptible horses [13].

**Clinical Findings:** Horses affected by strangles develop a clinical sign varying from acute form to a complicated form depending on the immune status of the horse, virulence of the agent and the environmental factors facilitating the disease process [20].

**Acute Form:** In acute form strangles is characterized by sudden onset of fever followed by mucopurulent (Copious) nasal discharges and acute swelling in the submandibular and retropharyngeal lymph nodes, depression, lethargy and loss of appetite within an incubation period of 3-14 days [1].

Pharyngitis and lymphadenopathy may develop which makes swallowing difficult and affected horses become anorexic or reluctant to eat and stand with neck extended. A soft cough and slight but painful swelling between the mandibles occurs [3].

With the progression of the disease, abscesses develop in the submandibular (Between the jaw bones) and/or retropharyngeal (At the back of the throat) lymph nodes. The lymph nodes become hard and very painful and may obstruct breathing (Strangles). The lymph node abscesses will burst in 7-14 days releasing thick pus heavily contaminated with *S. equiequi*. The horse will usually rapidly recover once abscesses have ruptured [15].

**Chronic Form:** Classic strangles is a severe infection that can be fatal usually because of a variety of complications that occur. Strangles in about 20% of the cases is complicated which significantly increase the mortality rate [21].

**Guttural Pouch Empyema (Filled with Pus):** which may be concurrent with classic strangles, or follow in the immediate convalescent period. The two guttural pouches are large mucous sacs; each is a ventral diverticulum of the Eustachian tube [20]. They present only in Equidae and are situated between the base of the cranium dorsally and the pharynx ventrally. They open into the nasal pharynx and each has a capacity of about 300 ml [19]. Persistent infection in the guttural pouch may lead to inspissations (Drying) of pus and, in some cases, the formation of a solid, stone-like, concretion called a chondroid. It is extension of the infection in to guttural pouches as a result of rupture of retropharyngeal lymph nodes in to the medial compartment [1].

**Bastard Strangles:** This describes the dissemination of infection to unusual sites other than the lymph nodes draining the throat. For example, abdominal or lung lymph nodes may develop abscesses and rupture, sometimes weeks or longer after the infection seems to have resolved. A brain abscess may rupture causing sudden death or a retropharyngeal lymph node abscess may burst in the throat and the pus will be inhaled into the lung [2].

**Post Strangles Myocarditis (Inflammation of Heart Muscle):** which may follow strangles in a small proportion of horses. An electrocardiogram (ECG) can determine that a horse can return to heavy work or to training after an episode of strangles [7].

**Suppurative Necrotic Bronchopneumonia:** occurs secondary to the aspiration of pus from the ruptured abscesses in the upper air way and is the most fatal complication of strangles [22].

**Purpura Haemorrhagica:** this is an immune-mediated acute inflammation of peripheral blood vessels that occurs within four weeks of strangles, while the animal is convalescing [23]. It results from the formation of immune complexes between the horse's antibodies and bacterial components. These immune complexes become trapped in capillaries where they cause inflammation, visible in the mucous membranes as pinpoint haemorrhages. These haemorrhages lead to a widespread severe edema of the head, limbs and other parts of the body. Purpura haemorrhagica can also be a complication of routine vaccination [4].

**Anemia (Low Red Blood Cell Count):** occurs during the convalescent period because of immune-mediated lyses of red blood cells [8].

**Myopathic Syndrome:** muscle infarction and rhabdomyolysis have been reported. Muscle infarction is manifested from purpura hemorrhagica and often results in infarction in the gastrointestinal tract, skin and lung. Rhabdomyolysis occurs due to cross reactivity of antiserum M antibodies with myosin which results in stilted gait and elevated serum activity of creatine kinase [15].

**Inapparent Form:** Although the disease process described above is classic, some horses (Especially older horses) will develop a mild, short lasting disease without or with minor lymph node abscessation [24]. This is thought to be the result of partial immunity although this may also result from infection by *S. equiequi* of relatively low virulence. Affected horses have a transient fever for 24-48 hours and profuse nasal discharges. Moderate enlargements of mandibular lymph nodes occur in only half of the affected horses [14].

**Clinical Pathology and Necropsy Findings:** During the acute phase, hematological abnormalities like leukocytosis with neutrophilia reaching a peak as the lymph nodes abscessate occurs [22]. Hyperfibrinogenemia both in acute and chronic form is also common. Leukocytosis with a hyperproteinemia attributable to a polyclonal gammaglobulinemia is a characteristic of metastatic and chronic abscessation. Serum IgG antibody titer to serum protein are very high in horses with metastatic infection or purpura hemorrhagica [25].

Necropsy examination usually reveals suppuration in internal organs especially in the liver, spleen, lungs, pleura, peritoneum and mesenteric lymph nodes [11].

### Diagnosis

**Clinical Diagnosis:** Clinical signs like fever, mucopurulent nasal discharges and lymphadenopathy in a history of recent exposure of susceptible horses are presumptive diagnostics for strangles [26]. Endoscopy is used to diagnose guttural pouch empyema and/or chondroids and also used to take sample from pouch contents [20].

**Laboratory Diagnosis:** The gold standard for the diagnosis of strangles is isolation of *S. equiequi* through the culture of nasal swab, nasal lavage and pus from abscess or lymphoid tissues (Cult). In culture, *S. equiequi* forms a large mucoid colony surrounded by a wide zone of hemolysis in blood agar. *S. equiequi* is differentiated from other lancefield group C Streptococci by its sugar fermentation in peptone water containing serum [10, 13].

The Polymerase Chain Reaction (PCR) test is a way to magnify and detect very small amounts of DNA from a variety of infectious agents. It is available for Strangles, by the amplification of the specific gene of the agent, providing a very useful and rapid diagnosis of the *S. equiequi* infection [23].

Also PCR is considered useful for detection of carrier animals not showing signs of infection, establishing *S. equiequi* status prior to transport, following transport and prior to commingling, determining the success of elimination of *S. equiequi* from the guttural pouch and Prevention [16].

Serology is one of the most diagnostic methods for diseases. ELISA for antibodies to serum protein M is useful in the diagnosis of strangles. It is also useful for the diagnosis of horses predisposed to purpura hemorrhagica and metastatic abscesses [5].

**Differential Diagnosis:** In the early stage of the disease, it may be confused with equine viral pneumonitis, equine adenovirus, equine viral arteritis and equine influenza but in these diseases there is usually no marked enlargement of lymph nodes [1].

**Treatment:** Treatment of horses with strangles depends on the stage, severity, feature and epidemiology of the disease. The drugs of choice for strangles are penicillin either as procaine penicillin or potassium (Sodium)

penicillin intramuscularly or intravenously and sulfonamides-trimethoprim combination orally or intravenously [5]. NSAIDS are used as supportive treatment to reduce swelling and to provide pain relief [17].

Horses with early signs including fever, anorexia, depression and purulent nasal discharges should be isolated and treated with a therapeutic dose of penicillin for at least five days and horses with signs of prolonged fever, anorexia, depression, lethargy or dyspnea are also responsive for systemic treatment with penicillin [1].

Treatment of horses with lymph nodes abscessation should be directed toward enhancing maturation and drainage of abscess. The recommended procedures include isolation of sick horses, application of hot packs and poultices to the abscess and possible lancing of the ventral surface of the abscess [19]. After draining of the abscess, it should be flushed regularly with 3% to 5% povidine iodine. Systemic administration of penicillin and application of petroleum ointment on the surrounding skin to prevent scalding should be used [17].

Horses with metastatic infections require systemic penicillin in combination with specific therapy for complications. Pulmonary and mesenteric infections are problematic because they are not amenable to surgical drainage and prolonged penicillin therapy is required to effect a cure [27].

Complication like guttural pouch empyema requires surgical drainage or repeated flushing of the affected pouch through the pharyngeal openings with sterile and non-irritating isotonic solutions (0.9% NaCl) and combined topical and systemic administration of potassium benzyl penicillin is also used. Purpura hemorrhagica is treated with systemic penicillin and corticosteroids [2]. Horses with upper air way obstruction may require replacement of short term tracheotomy [1]. Oral or IV fluid therapy is given for horses unable to eat or drink [14].

### **Control and Prevention**

**Hygienic Measures:** In a group of infected horses; all movements should be stopped and an infected quarantine area should be separated from both clean and contaminated area with strict hygienic activities [26]. Infected horses should be isolated and treated, newly introduced horses should be quarantined for three weeks and observe for signs of strangles, horses with elevated temperature should have nasopharyngeal or guttural pouch swab cultures, recovered horses never be mingled with susceptible horses for a period of several months and infection status should be determined by three PCR examination and cultures at weekly interval [5].

All potential sources of fomites including pails, brooms, housings, feed utensils, water troughs and pasture and grooming brushes should be thoroughly cleaned and disinfected; people who care of infected horses must avoid contact with susceptible horses if not they must wear protective boots and clothes. Pastures on which infected horses have stayed should rest for four weeks [2,12].

**Vaccination:** Strangles continued to be a serious and common epidemic disease throughout the world. Vaccination is one of the important control and prevention method [6]. With strangles, vaccination will most likely reduce the severity of disease in the majority of horses infected after they are vaccinated. Available vaccines can be administered by intramuscular, intravenous, subcutaneous and intranasal routes. Extract vaccine (m Protein rich extracts) and live attenuated vaccines are widely used [9].

Vaccination is generally not recommended during an outbreak of strangles. If there are horses on the farm with no clinical signs of infection (Fever, nasal discharge) and no known contact with sick horses, vaccination may be considered [12]. Horses that have had the disease within the previous year also do not need to be vaccinated. Once recovered from an active infection, 75% of horses have immunity for one to two years [16]. Vaccination of horses recently exposed to strangles (That have high antibody levels) may result in purpura hemorrhagica. Vaccination is only recommended in healthy horses with no fever or nasal discharge [27].

Naïve horses and foals should be vaccinated with extract vaccines IM or IV or subcutaneously at an interval of two weeks with two to three doses. It elicits serum antibody responses within seven days [12]. Horses can also be vaccinated by live attenuated vaccines through intranasal or submucosal routes which stimulate mucosal and systemic antibodies response. Booster doses are given once annually [9].

Vaccination of brood mares one month before foaling increases colostral IgG antibodies and presumably serum and mucosal immunoglobulin concentration in their foals [15].

**Economic Importance:** Strangles is one of the most important diseases of horses in the world accounting up to 30% of reported infectious disease episodes [1]. The disease is important not only because of death but more significantly the disruption of the management of commercial horse establishment, the time and cost necessary to treat infected horses and esthetic

unpleasantness of the running nose and draining abscesses. Strangles is important not only on horse production but also indirectly on agriculture [2].

### CONCLUSION

Strangles is a highly contagious and serious infection of horses and other equids caused by a gram positive, hemolytic, pyogenic and lancefield group C bacterium, *Streptococcus equisubsp. equi*, in the world and is highly contagious. The primary reason for concern is how quickly it can be spread within the horse population and also that some recovered horses will become 'carriers' and therefore able to spread the disease to others even though they themselves appear clinically healthy either by direct contact or indirectly from fomites on equipment or personnel. Strangles begins as rhinitis and pharyngitis and is characterized later by abscesses in the lymph nodes. In rare instances, strangles may be generalized (Complicated) and terminate in death. Strangles is continued as a serious problem of horse farms and is one of the limiting diseases in equine production directly and on agriculture indirectly.

Based on the above conclusion the following recommendations are forwarded:

- All infected horses and any horses that have been in contact with them should be in strict isolation.
- Monitoring of animal movements should be performed.
- Screening before purchase ought to be performed to reduce the spread of strangles.
- If shared water troughs are utilized, they should be routinely disinfected. When traveling to shows, minimize your horse's exposure by bringing your own feed, buckets and equipment.
- Horses should be vaccinated annually or as recommended during outbreak since vaccination reduce the severity of the disease as well its occurrence.
- Since the disease is highly contagious and cause complications in prolonged time, early diagnosis ought to be performed.
- Need to create awareness among horse owner's.

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