STRANGLES; THE CONTINUOUS CHALLENGE TO PRACTITIONERS (A REVIEW ARTICLE)

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ABSTRACT

Streptococcus equi is the etiologic agent of strangles a highly contagious disease of horses that is manifested by respiratory signs such as fever, nasal discharges and inflammation of lymph nodes in the head region. The disease is widely distributed around the world, however very little information is available regarding its incidence of the disease in the region. The disease causes significant impact on the horse sport industry. This impact is a result of the direct loss of the activity of race horses, disturbance of exercising schedule, cost of treatment, disinfection and other management requirements. Treatment is directed toward the use of appropriate antibiotic such as procaine penicillin G and trimethoprim sulfonamide and supportive care. Vaccination that is based on conventional preparation or genetically modified live organism, is available. However extra-cautions should be exercised when live vaccines are used. Additional measurements are required for prevention of introduction of affected horse into a strangle-free herd.

Streptococcus equi causes strangles, an acute highly contagious disease of horses that is manifested by local respiratory signs such as, thick mucopurulent nasal discharge, inflammation of nasal and pharyngeal mucous membranes with swelling and abscessation of submandibular, submaxillary, and reteropharyngeal lymph nodes as well as depression, increased body temperature and reduced appetite. The name strangles was originated from the upper airway stenosis and compression that is induced by enlarged lymph nodes in the pharyngeal region.

Strangles was described for the first time by Ruffus J. in the thirteenth century. Solleysel thought that strangles was contagious and recommended isolating infected horses (Todd, 1910). Later, Rivalta detected chains of cocci in pus from abscess of horses infected with strangles, however Schutz (1988) was the first one described the organism in details (Todd, 1910, Schutz 1888). Morphologic and cultural characteristics in simple media were described by several authors (Sand 1888, Poels 1888, Baruchello

1908). Finally, Bazeley in 1943 provided detailed microbiologic and immunologic information of the disease in the 1940's.

INTRODUCTION

Microbiology

S. equi is a Gram-positive, ovoid or spherical in shape, 0.6-1.0 _m in diameter (Hardie, 1986). The organism occasionally forms bacilli and grows in pairs leading to the formation of short or long chain. *S. equi* is a member of Lancefield group C streptococcus that is capable of forming capsules in young cultures. In addition, honey-colored mucoid colonies that form wide zone of hemolysis are produced when the bacterium is cultured on blood agar (**Prescott et al.1982**). Matt form, a different form of colonies, is associated with a mild form of the disease and resulted from a phage-associated hyalurinadse that cause collapse of the mucoid colonies (**Timoney et al.1984**). S. equi is an acid producing organism that ferments glucose, sucrose, maltose, salicin and galactose, however, it does not ferment lactose, arabinose, trehalose, raffiose, inulin, mannitole or sorbitole.

S. equi gains its virulence as a result of the presence of several factors. The most important virulence factors are hyaluronic acid capsule and M-protein. Also there are other virulence factors such as hyaluronidase, streptolysin S, streptokinase, peptidoglycan, and IgG Fc-receptor protein.

Hyaluronic acid is a high molecular weight polymer that consists of N-acetyl glucosamine and glucuronic acid alternatively. Hyaluronic acid is a non-antigenic factor and is not involved in the protective immunity, however, it reduces the numbers of streptococci that are killed by neutrophils as well as maintains the appropriate environment for bacterial toxins such as streptolysin O to function. M-protein is a dimer or a trimer molecule with a molecular weight of 58 kDa. The main fragments are 46kDa, 41 kDa and 29-31 kDa (**Galan and timoney,1987**). It is an antiphagocytic factor which acts by binding fibrinogen and complement factor H to the M-protein (**Traore et al. 1991, Baschwitz and Timoney 1994, Erickson and Narcross 1975**). Antibodies against M protein enhance in vitro phagocytosis and killing of bacteria by PMNs. Mutant *S. equi* that express low levels of M protein are much more susceptible to phagocytosis in vitro by equine PMNs and also less virulent for mice (**Wallace et al. 1995**).

Streptokinase is an enzyme that interacts with the c-terminal serine protease domain of equine plasminogen to produce active plasmin which hydrolyses fibrin (**McCoy et al 1991**). This may help in dispersion of the organism in tissues. Streptokinase may also provide low-molecular

weight nitrogenous substrates for bacterial growth through complement activation. Streptolysin S causes the beta hemolysis through a mechanism that is based on creating transmemebrane pores (**Flangan et al., 1998 and Carr et al., 2001**). On the other hand, streptolysin O is inhibited by oxygen reversibly and the other member of this family of toxins. Streptolysin O forms hydrophilic channels in membranes that contain a high a mount of cholestero) (**Timoney and Mukhtar 1993**). It is highly antigenic; however it is highly conserved and is capable of lysing erythrocytes of different hosts. It is also toxic for other cells and cell structures including PMNs, cardiac muscles, platelets and cytoplasmic lysosomes (**Bhakdi et al.1985**).

Peptidoglycan potentially activates the alternative complement pathway causing release of chemotactic factors (C3a, C5a) for equine PMNs. This may explain the presence of large number of PMNs in the infected lymph nodes and on the upper respiratory mucosa during strangles. It is also responsible for the febrile condition in S. equi infected horses by inducing the release of pyrogenic cytokines as interleukin-1, Il-6, Il-8 and tumor necrosis factor-alpha from leukocytes (**Oikawa et al.1994**). The role of the *S. equi* surface associated IgG *Fc* receptors is not completely understood. They may help the organism to avoid the host cellular recognition mechanisms via binding of the host plasma protein to the surface of the organism which may prevent access of C3 or specific antibody to target sites on the organism.

Epizootiology :

Strangles continues to be the most frequently reported disease worldwide according to the Equine Disease Quarterly. Numerous outbreaks were reported from Denmark, Ireland, South Affrica and Switzerland (**Equine diseases quarterly 2008**). The disease has been reported in the United Arab Emirates, however it involved quarantined horses arriving for horse events (Wrenery, Personal communication). Very little information regarding strangles in the Kingdom of Saudi Arabia is available.

Equidea is the only animal group affected by *S. equi*. The large distribution of horses around the world makes the disease world wide in distribution (**Rossdale and Ricketts 1974, Blood et al., 1983**). Foals less than six months are more susceptible to the disease due to the lack of acquired immunity (**Fallon, 1969**). However any age animal can be affected unless a vaccination program has been used or previously exposure has occurred. Inhalation and ingestion are the most common route of infection. The disease can be transmitted via direct oral or nasal contact or by the aerosal route. It also can be transmitted by indirect contact through transfer of purulent discharge in feed, water, water bucket, bedding, handlers, flies, veterinarians and other animals. Although the organism requires the presence of the host for its survival, it may persist in

the environment for up to 63 days if favorable moisture and temperature conditions are present (**Jorm, 1992**).

The morbidity rate is high, ranging from 30% to as high as 100%. However the mortality rate is low usually and does not exceed 10%. Recovered horses can shed the organism for several months but usually it is shed from 4 to 5 weeks. Long term carriage of *S. equi* may occur if the organism remains in the guttural pouch. *S. equi* can be recovered more easily from the guttural pouch using a guttural lavage. The horse may remain an asymptomatic carrier for up to 39 months (**Newton et al., 1997**).

The continuous introduction of new horses to a farm can allow the disease to become endemic in the population. Predisposing factors that enhance introduction, maintenance and spread of the disease include: overcrowding, movement and mixing of horses from different sources at breeding farms, boarding stables, training centers, race tracks and shows as well as introduction of affected animals either incubating the disease, clinically ill or recovering from the disease (**Timoney 1993a**).

Pathogenesis :

S. equi enters the body through the mouth or the nose and becomes attached to the cells in the crypt of the tonsil and surrounding lymph nodules. Shortly, it migrates below the mucosa into the local lymphatics and reaches the lymph nodes that drain the pharyngeal and tonsillar region. Extracellular multiplication in the lymph nodes takes place leading to formation of long chains of the organism. Neutrophils are attracted to the infected site by chemotactic factors. Antiphagocytic factors such as hyaluronic acid capsule, M-protein and leukocidal toxins released by the organism reduce the efficacy of phagocytic PMNs and promote abscess development. During abscess development the flow of lymph fluid is restricted causing enlarged lymph nodes and subsequently swelling of the submandibular area and possible occlusion of the upper respiratory tract (**Timoney, 1993a**).

The disease is usually restricted to the upper respiratory airways and surrounding tissues, however systemic spread can occur through hematogenous and lymphatic channels. This results in abscesses in lymph nodes and other organs in the thorax and abdomen and sometimes in the brain (**Sweeny et al., 1987**). This form of the disease is known as "bastard strangles".

Clinical signs :

The incubation period for strangles is 4-10 days and is followed by an elevated body tempera-

ture of more than 40°C, depression, anorexia and restlessness (**Sweeny 2002**). Nasal discharge is serous in the beginning but becomes mucopurulent and eventually purulent. The intermandibular region is painful with some swallowing difficulties and head extension due to lymphadinitis of regional lymph nodes. Local edema may develop. External rupture of affected lymph nodes may occur in 1-2 weeks as well as internal rupture of lymph nodes into the pharynx. Aspiration pneumonia and guttural pouch empymea may occur following internal rupture of lymph nodes. Nerve damage may lead to dysphagia or hemiplegia of arytenoid. The animal may show signs of cough which is soft and moist and then becomes more productive and severe. Systemic spread of the organism or metastasis to other lymph nodes and organs such as liver, kidney, spleen, lungs, and brain may occur. This form of the disease is called bastard strangles. Therefore signs referable to involvement of these organs may develop. After rupture of abscesses, fever, depression, and anorexia subside and recovery can occur if no other abscesses develop.

In addition to the clinical and economic impacts caused by strangles, it may lead to more complicated clinical conditions that require further clinical management and may result in additional economic loss (**Sweeny et al., 1987**). Some of these complications are guttural pouch empyema that might result in chronic nasal discharges or acute severe upper airway obstruction. Subsequently cranial nerve neuropathy may develop and be expressed as laryngeal hemiplagia, facial nerve paralysis and Horner's Syndrome. Guttural pouch empyem may be managed by systemic antibiotics, local irrigation or surgical drainage of the guttural pouch.

Purpura hemorrhagica usually affects mature horses but in one study it was reported in a yearling. This may also occur following vaccination with a bacterin or an M-protein vaccine. Purpra hemorrhagica is characterized by edematous swellings that occur due to the vasculitis that occurs with the disease. The vasculitis is a manifestation of a streptococcal hypersesitivity. It is treated by antibiotics, anti-inflammatory drugs and supportive care.

Bastard strangles occurs when there is metastatic spread of the bacterial infection of lymph nodes in the head and upper neck region. The infection can spread to the lungs, liver, kidney, brain and mesenteric mediastinal, periorbital and perivertebral lymph nodes. Signs due to involvement of these lymph nodes and organs may then occur. Brain and spinal cord involvement may cause hyperasthesia, incoordination, seizures, nystagmus, proprioceptive defect of the limbs, head tilt circling and quadraparesis. Purulent pleuritis or peritonitis may develop due to rupture of mediastinal and mesenteric lymph nodes. Cutanous abscessation, cellulitis and ulceration can occur in the facial, periorbital, perianal regions and on the extremities. Preputial abscessation can cause marked preputial swelling and dysuria. Paravertebral abscessation can cause pain in the neck and restrict motion. Mesenteric abscessation may lead to colic, recurrent

fever, chronic weight loss, depression, reduced appetite and occasionally death.

Upper airway obstruction as a result of abscessiation of the pharyngeal and mandibular lymph nodes may cause asphyxia and lead to death. Tracheostomy may be required to relieve asphyxia. Pneumonia and pleuritis are not common in uncomplicated cases but may be seen in cases with aspiration of the organism through inhaled exudate or by metastatic spread of the organism to the lungs and mediastinal lymph nodes. Purulent bronchopneumonia rarely causes massive intrapleural hemorrhage or acute epistaxis. Laryngeal or pharyngeal dysfunction may result from retropharyngeal abscessation or guttural pouch empyema which leads to damage of cranial or recurrent laryngeal nerves. Agalacti may be found in mares before parturition although mammary glands have normal physical appearance. Agalactia might be due to high fever, anorexia and depression associated with the infection. It is not life-threatening to the mare but can become a management problem in feeding the foal.

Cardiac conditions such as endocarditis of the right atrioventricular valves and thrombus formation in the right atrium leading to heart murmurs were reported. Myocarditis due to immune mediated inflammation may result in a disturbance in the conduction system which results in significant changes on the electrocardiograms. Cardiac failure may result. Therefore giving the horse rest for several weeks after experiencing strangles is essential. The presence of nodules in atrioventricular valves and ruptured chordae tendinea has been reported as a result of bacterial endocarditis which may occur with strangles.

Arthritis, glomerulonephritis and myositis may develop during or after clinical strangles. These conditions may be attributed to immune complex deposition in blood vessels of these tissues. Chronic nasal discharge may continue as an ongoing problem for a prolonged period. Mortality rate is low at 5-10% and is usually due to systemic complications. Finally, pneumonia is one of the most common causes of death.

Immunology :

Horses affected with strangles may recover following production of specific serum antibodies against the M-protein and streptolysin O (**Timoney 1993a**). Recovered animals also develop solid immunity following natural disease mainly due to production of mucosal secretory IgA. This local mucosal immunity plays a significant role in protection against experimental reinfection before detection of serum opsonic antibody. The produced immunity is not life-long in duration therefore, additional infections may occur. Field exposure with or without developing clinical signs can produce a high titer against *S. equi* Further exposure, either naturally or through vac-

cination, induces a rapid immune response. However exposure to a natural infection might produce the disease by overwhelming the circulating antibodies. At time of abscessation circulating antibody titers are low. Some defect in phagocytosis would be suggested if antibody titers were found to be high and accompanied by disease. Animals may be carriers in the face of type specific immunity.

Diagnosis :

Diagnosis can be made as following: 1) clinical signs including fever, lymphadenopathy and nasal discharge; 2) laboratory diagnosis including CBC showing elevated PMNs numbers up to 25,000 cells/ l, increased fibrinogen of ≥ 0.5 g/dl, and positive *S. equi* bacterial culture from nasal swab or nasal wash, swabs from ruptured abscesses or aspirate from intact abscesses. A positive culture is considered the definitive diagnosis. However negative results do not rule out S. equi because only 50% of draining lymph nodes will give positive results. Polymerase chain reaction (PCR) to detect S. equi bacterial DNA utilizing primer sequences from SeM in samples similar to those used for bacterial culture is a more advanced method to detect the organism and has been developed to give result within 6 hours (**Newton et al., 2000, Timoney and Artiushin 1997**).

The clinical manifestation of the disease may lead to miss-diagnosis with other disease that have similar clinical signs. Fever, nasal discharge and anorexia should be differentiated from viral disease such as influenza or rhinopneumonitis using CBC and bacterial culture. Lymph nodes abscessation should be differentiated from S. zooepidemicus with bacterial culture. Infections by Actinobacillus, Streptococcus and Corynebacterium pseudotuberculosis cause abscessation and differentiation from *S. equi* is by the nature of the purulent drainage and by culture. Anaerobic infections due to foreign body penetration or external puncture may be differentiated from strangles based on tissue necrosis, foul odor and necrosis. Retropharyngeal lymph node swelling is differentiated from *S. equi* associated guttural pouch empyema by radiography and endoscopy. An enlarged pharynx may also be due to thyroid adenoma, parotiditis, parotid melanoma, stalocoele, guttural pouch tympany, hematoma, cellulitis or any traumatic lesion.

Treatment :

Management concerns include isolation and segregation of clinical cases, due to the highly contagious nature of strangles. Complete rest and good nursing care including keeping horses warm and dry with good feed and water. The disease significantly impact treatment approach

(Sweeny et al.,2002). If horses were exposed to strangles showed no signs, antibiotics may be used to reduce chances of development of clinical disease. However, if early clinical signs such as fever and nasal discharges started to appear, the use of antibiotics may prevent further development such as lymph node abscessation.

High doses of procaine penicillin G is the drug of choice, 22,000 IU/Kg two times per day for 10-14 days or until clinical signs subside. Additional antibiotics that may be used include ampicillin, trimethoprim sulfonamide, erythromycin, oxytetracycline, benzathine penicillin. The argument that poor immunity and bastard strangles may develop due to antibiotic treatment is not supported by reliable data.

Further treatment may be required based on the severity of the disease. Application of hot-packs, poultices or liniments enhances maturation of affected lymph node. After maturation, lymph nodes are lanced, drained and flushed with povidone-iodine until discharge ceases. Non-steroidal antiinflammatory drugs (NSADs) such as phenylbutazone may be used to reduce fever, pain and swelling. If severe dyspnea developed, tracheostomy is required. Guttural pouch treatment by flushing or surgical incision is recommended since it is a very suitable site for long carrier. Stomach tube feeding may be needed. Cases that develop bastard strangles will have a poor prognosis and abscessation of internal organs and lymph nodes require prolonged antibiotic treatment.

Prevention :

Prevention and control measurements must be strictly followed to minimize the spread of the disease. Newly arriving horses should be segregated and carefully monitored for two weeks prior to being introduced to the herd. Isolation of suspected cases immediately for 14-21 days, rectal temperature should be taken 2x/day and other health abnormalities such as nasal discharge, cough, dysphagia and dyspnea should be recorded. Culture or PCR of nasal swab or washes and isolation of clinically affected or positive cases for 6-weeks should be implemented. Separate pails for feeding and watering of affected animals are needed. Complete disinfection or cleaning of stalls and stables with chlorohexadine or glutaraldehyde. Stalls should be left empty for drying for 4 weeks. Fly control by installation of screens, insecticides and electric fly killers is recommended in controlling spread of the disease. Newly introduced animals should be isolated for 3-4 weeks. Farm workers that have worked with diseased animals should not work with unaffected animals without thorough washing and disinfection. Finally, equipment such as brushes and halters should not be shared between affected and non affected horses.

There are a number of S. equi vaccines were developed including conventional and molecular type vaccine. Conventional included whole bacteria or components. Bacterins which are killed S. equi that is given as 2-ml dose intramuscular three times at 2-4 weeks interval then followed by annual booster dose **(Bazely 1943)**. This vaccine caused marked vaccine reactions such as inflammation, abscess formation and muscle pain **(Smith 1994)**. This led to the development of subunit vaccines include the M-protein vaccine produced by hot acid treatment or mutanolysin were introduced in the USA during the 1970s and 1980s. Protein-based vaccine decreases the clinical signs of strangles in young horses to a level similar to what occurs in adults **(Srivastava and Barnum1983)**. However the safety of this vaccine was questionable therefore limiting its wide use in the field (Hoffman et al.,1991). Finally, a recombinant S equi hyaluronate associated protein failed to provide protection **(Waller and Jolley 2007)**.

Advancement in the understanding of the molecular bases of S equi resulted in the production of more sophisticated vaccines. An intranasal live vaccine, genetically modified, non-capsulated, Pinnacle IN, by Fort Dodge, has been introduced to the market in the USA **(Walker and Timoney 2002)**. It has been shown to stimulate a mucosal antibody response. However, adverse reactions such as nasal discharge, lymphadenitis, abscesses of lymph nodes in the head and purpura haemorrhagica were remarkable **(Al-Ghamdi et al., 2001)**. In Europe, a live attenuated vaccine TW 928, Equilis StrepE, Intervet, was developed **(Kelly et al., 2006)**. This vaccine is given under the mucosa inside the upper lip. It produces immunity for short period of time about three months that can be boosted. Nonetheless the use of these live vaccine should be exercised with great caution.

Acknowledgment :

The Author would like to thank Dr. Taha Fouda for critical of this article.

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الملخص العربى

خناق الخيل : التحدى المستمر للأطباء الممارسين

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٣١٩٨٢، ص. ب. ١٧٥٧، المملكة العربية السعودية

خناق الخيل هو أحد الأمراض الوبائية التى تصيب الخيول نتيجة الإصابة بالميكروب العقدى حيث تتمثل الأعراض الإكلينيكية فى ظهور بعض العلامات التنفسية مثل الإفرازات الأنفية، صعوبة التنفس، إرتفاع درجة حرارة جسم الحيوان المصاب بالإضافة إلى إلتهاب وتضخم العقد الليمفاوية فى منطقة الرأس.

كما ينتشر هذا المرض فى العديد من بلدان العالم ولكن هناك القليل من المعلومات المتوفرة لدينا فيما يتعلق بانتشار المرض فى المنطقة، ولهذا المرض آثاراً سلبية على رياضة الخيول نتيجة لعدد من العوامل منها فقدان الحصان لنشاطه وحيويته، خلل فى نظام التدريب بالإضافة إلى تكاليف العلاج واحتياجات التعقيم وطرق الرعاية الأخرى، وتتلخص برامج العلاج لمثل هذه الحالات فى إستعمال المضادات الحيوية مثل البروكايين بنسيلين وكذلك مركبات الترای میثوبريم والسلفوناميد بالإضافة إلى الرعاية الجيدة والأدوية المنشطة.

كما تتوافر لهذا المرض اللقاحات التى تعتمد على تجهيز واستخدام الميكروبات الحية والمماثلة جنبا مع توخى الحذر التام عند إستعمالها، بالإضافة إلى ذلك هناك بعض الإجراءات الهامة التى يجب مراعاتها والأخذ عند إدخال الخيل المصاب إلى المزارع السليمة والخالية تماماً من المرض.