Equine Strangles: A Brief Overview

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INTRODUCTION

Equine strangles is an enzootic disease of horses characterized by a regional or generalized suppurative lymphadenitis associated with an upper respiratory infection. The etiologic agent is a beta-hemolytic streptococcus, *Streptococcus equi* (*S. equi*). Reports indicate that no primary viral infection or abrasion of the respiratory mucosa is needed to produce the disease. The disease occurs in mostly young, non-exposed, non-vaccinated horses. Morbidity is as high as 100% while mortality is quite low, from 2 to 5%. All ages are affected in non-enzootic herds. This paper is a review of the pathological characteristics of the infection and the various methods of treatment, control, and prevention of strangles outbreaks.

PATHOGENESIS

Experimentally it has been found that the incubation period of strangles is 3 to 6 days. First, there will be anorexia accompanied by a fever ranging from 100 to 105°F. The respiratory and heart rates will remain within normal limits. There will be a leukocytosis, coughing, and often the nasal mucosa appears dry, but any mechanical irritation will induce a copious nasal discharge. About 7 to 9 days post-infection the horse may appear to be uncomfortable, standing with its neck outstretched. This is associated with the development of abscesses in the pharyngeal, intermandibular, and retropharyngeal lymph nodes. In the majority of cases, the abscesses drain about two weeks after their onset and then proceed to heal.[1] In a few instances there is the formation of retropharyngeal abscesses which do not show externally. This is associated with inspiratory dyspnea and possible tracheal collapse due to external pressure. In these cases a tracheotomy is necessary, as they will suffocate or 'strangle' due to occlusion of the larynx without it.[2]

"Bastard" strangles, the disseminated form of the disease, is an infrequent but possible complication of the routine strangles outbreak. The occurrence of this condition seems to be related to animals with an inadequate immune response to the initial infection. Often times the animal will present with signs of 'colic' or peritonitis and may have a history of chronic weight loss. Differential diagnoses include pleuritis, neoplasia, chronic hepatic disease, chronic malabsorption, renal disease, and severe parasitism. In these animals with an apparent chronic disease process, clinicians may see: depression anemia, increased plasma protein concentration, leukocytosis with a left shift, hypergammaglobulinemia, hypoalbuminemia, and/or hyperfibrinogenemia. In one study it was shown that the fibrinogen concentration may have some prognostic value. In that study, 6 of 15 horses (40%) with a concentration of greater than 800 mg/dl died or were euthanized, but only 2 of 10 horses (20%) with a concentration of less than 800 mg/dl died or were euthanized.[3]

Those animals felt to be acutely colicy due to a case of "bastard" strangles may be elected to be managed with phenylbutazone to reduce fever, relieve pain and anxiety, and allow the animal to eat and drink. Surgery is not a very practical method of treatment for these animals since it is so difficult to provide adequate drainage of the abscess without contaminating the peritoneal space. The location of the abscess, often in or very near vital organs, makes it hard to excise, and often it is quite friable and will rupture with handling.[3] In general, the animal suspected of having "bastard" strangles is given a very poor prog-

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nosis. The animal will usually die due to extensive involvement of the central nervous system or viscera.

Other possible sequelae to a routine bout of strangles include laryngeal hemiplegia and equine purpura hemorrhagica. The laryngeal hemiplegia is thought to be due to an abscess forming in an anterior cervical lymph node that happens to lie in close proximity to the recurrent laryngeal nerve. The purpura hemorrhagica is thought to be a result of an exaggerated immune response to *S. equi*. Each individual attack varies in severity, but the animal generally presents with petechial hemorrhages of the nasal mucosa, edema of the head and distal limbs, and in some cases, fever. The skin is not broken but serum may exude.[1]

TREATMENT

The treatment of strangles is a highly controversial topic. It can be divided into two main categories—conservative and medical. Those supporting the conservative mode of treatment think the animal should be monitored closely and treated symptomatically. They advise the owner or herdsman to observe for animals exhibiting signs of upper airway obstruction. Should this occur, either a tracheotomy is performed or the obstructing abscess is lanced and drained. It is felt that in other than an emergency situation the abscesses should be allowed to come to a head on their own or be hot-packed to a head and then incised, drained, and lavaged. It is especially emphasized that environmental stress be minimized. This means maximum rest for the animal, high quality feed, good shelter, etc.[4] These veterinarians advise against the use of antimicrobials due to the action of certain antibiotics such as penicillin, which acts to inhibit cell wall production. Because *S. equi* antigens necessary to a protective immune response are located on the cell wall, they argue that the use of penicillin in effect reduces antigen exposure and results in a deficient immune response, increasing the chances of inducing "bastard" strangles. The more conservative practitioners also suggest that the practice of restricting use of antimicrobials until the abscess ruptures, followed by treatment for 7 days is unncessary. They point out that once the abscess drains, the horse appears better whether treated thereafter or not.

The use of antibiotics in the face of an outbreak is the most controversial means of treatment. Some practitioners feel that the infection can be handled successfully with specific antibiotics given prior to the formation of abscesses in the lymph nodes. In fact, in one study it was shown that in 82% of the cases of strangles tested, excellent to good responses resulted with the use of ampicillin.[5] Other studies, using antibiotic discs, show all strains of streptococcus to be sensitive to penicillin, chloramphenicol, and ampicillin. Tribrissen is also shown to be effective. At the same time, it has been found that the streptococcal infections are resistant to such antibiotics as oxytetracycline, dihydrostreptomycin, sulphadimidine, and neomycin.[6,7]

Other veterinarians feel that *any* use of antimicrobials (especially inadequate doses) in the treatment of strangles enhances the chance of developing internal abscesses. It is believed by some authors that the hematogenous spread of the organism is underway by the time superficial abscesses appear and that all lymph nodes are inflamed in *S. equi* infections. The penicillin suppresses the immune response, as mentioned above, and thus allows the organism, already spread throughout the body, to grow out of control. They feel, therefore, that if antimicrobial therapy is initiated in horses exhibiting respiratory catarrh and lymphoid abscessation, the treatment should be continued at levels of 40,000 to 100,000 units/kg body weight, divided into two doses per day for at least 10 days and possibly for 5 to 6 months following cessation of all clinical signs.[2,3]

PREVENTION AND CONTROL

In most cases, outbreaks of equine strangles can be prevented by closely observing new arrivals to a facility and keeping these animals strictly quarantined (especially water and feed troughs) for two weeks. In the face of an outbreak, the enforcement of rigid sanitation and isolation of affected animals is recommended, until all clinical evidence of infection has subsided and for as long a time afterwards as is feasible. Cold temperatures apparently preserve the organism for a time so the environment may be a source of infection during cold weather. Normal cleaning and disinfection of feeding equipment, tack, and stalls should eliminate the organism.[7]

Some practitioners favor the use of an autogenous or commercial bacterin to limit the extent of an outbreak and prevent the spread

of infection.[8] The first work concerning countering *S. equi* infections was done by Bazeley in 1942. He found that antiserums, produced from the immunization of rabbits or horses with heat or formalin-killed young *S. equi,* possess potent opsonophagocytic activity. The antiserums were able to passively protect mice against homologous challenges. He also found that antiserums to one strain would protect against challenge by each of the 32 strains of *S. equi* in his collection. This has been supported by more recent studies which show that the different strains of *S. equi* are indeed serologically uniform and will protect against *Streptococcus zooepidemicus* infections.[1]

Bazeley's findings in the 40's resulted in the production of a marketable vaccine that became commercially available in Australia. A comparable product was developed by Englbrecht in the United States in 1969. Englbrecht found that a 30-ml dose given in three 10-ml intramuscular (IM) injections 1 week apart would result in a serviceable degree of immunity to challenge.[3] This initial regimen was to be boostered with a single injection the following year. Research was done to test the efficacy of the vaccine by vaccinating 3152 horses that were not exhibiting signs of disease and were from non-enzootic herds. Only 24 horses developed strangles and the disease was controlled on each farm. In contrast, it was found that on farms where strangles was epizootic, without vaccination the disease became enzootic once the infection ran its course.

The use of commercial vaccines had been extensive in Australia and the United States for many years before studies were done to accumulate definitive data concerning the need for 3 doses in the initial vaccination schedule, the longevity of the immune response produced by the vaccination, and a comparison between vaccination and natural infection immune responses.[9] To determine the need for Bazeley's suggested three-dose regimen, 3 injections of 4ml, 8ml, and 16 ml of vaccine were given intramuscularly at 2 week intervals to animals from different sources. Only one group received a booster at 12 months after the initial schedule. The titers of the animals were measured periodically during the test using passive hemagglutinating antibodies, the most sensitive and reliable test for indicating immunity to strangles. To get a satisfactory antibody response, it was determined that 3 doses of commercial vaccine are mandatory. Also, the booster at 12 months was recommended for up to 3 years of age, due to the duration of immunity which was found to range from 3 to 12 months.[9]

In the unvaccinated animals studied, it was found that immunity is achieved about 3 weeks after infection. At about 9 weeks post-infection the antibody levels are declining with no evidence of longevity of the immune response following natural infection. However, once an animal has recovered from strangles, it is believed that it is immune from further attacks for at least 6 months.[10] Animals exposed to a strangles infection appear to have a quicker anamnestic response than the vaccinated animals. The exposed animal is felt to be sensitized enough that 1 dose of vaccine acts as a booster.[9]

Currently, there are 2 equine strangles vaccines available for commercial use in the United States. Equibac II[a] is a killed *S. equi* product that is for use in healthy members of the equine species that are 3 months of age or older for the prevention of infection with *S. equi.* The recommended schedule is three 2-cc doses at 2-to-4 week intervals, deep in heavy muscle using aseptic technique. A single 2-cc dose is recommended as a yearly booster. Strepvax TM[b] is a concentrated aluminum hydroxide absorbed suspension of purified antigens derived from *S. equi.* The bacterin contains immunogenic protein of *S. equi* but is essentially free of the toxic and irritant substances present in whole cell bacterins.

Often owners will shy away from the use of vaccines because they do not want to risk local reactions to the vaccinations. When a reaction does occur there will be edema accompanied in some by induration. There may also be a transient fever and a pronounced neutrophilia. Abscesses at the site of vaccination are rare and are found to be due to *Streptococcus zooepidemicus* rather than sterile vaccine.[1,2,8,9]

DISCUSSION

Our recommendation for individual treatment of *S. equi* infections is to organize the cases into 2 groups based on severity of the disease and condition of the horse.

Group 1 would contain horses which have been on good health, nutritional, and manage-

[a]Equibac II, Fort Dodge Laboratories, Fort Dodge, Iowa.

[b]Wellcome Animal Health, Division Burroughs Wellcome Co., Kansas City, MO 64108, U.S.A.

ment programs, thus are in good condition. These horses are more likely to weather the disease on their own or with the help of conservative treatment such as non-sterodial anti-inflammatory drugs, hot-packing with eventual drainage, and good nursing.

The second group contains horses which are run down, have had marginal health and nutrition, and are more likely to be anorexic. These horses are going to need help in addition to the above conservative treatment. The treatment should include long term antibiotics (minimum of 10 days) such as penicillin (minimum 20,000 units per kg b.i.d.), ampicillin trihydrate (4-7 mg per kg b.i.d. IM, or if combined with oral usage, less than 3 gms to prevent ampicillin-induced diarrhea), or sulfas such as sulfadiazine or sulfamethoxazole combined with trimethoprim, using a trimethoprim dose of 4 mg per kg orally t.i.d. The tetracylines would not be recommended as a routine due to possible resistances and development of bastard strangles. For the same reasons it is safer to use trimethoprim or penicillin versus ampicillin for long-term therapy.

In the author's (Genetzky) experience the use of Equibac II has appeared clinically to be effective in preventing *S. equi* outbreaks in weanlings and has not resulted in abnormally high abscessation, which is contrary to some reports from practicing veterinarians. The bacterin has always been used in prevention programs which included anthelmintics and available respiratory viral vaccines.

Strepvax™ is new on the market and the author (Genetzky) has not had practice experience with its use. The reports from veterinarians in equine practice indicate the bacterin's use is not resulting in abscessation at the injection sites, but there are some questions as to its helpfulness in prevention of *S. equi* infections.

A typical outbreak of equine strangles can be readily diagnosed. However, the method in which it is handled, medically or conservatively, and the regimen of prevention adopted is equivocal. More work is being completed to develop improved vaccines with more sufficient data to support their efficacy. With additional research into methods of treatment, the uncertainty as to which regimen to follow will hopefully be alleviated.

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