Guidelines for dealing with Strangles outbreaks

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Strangles is a notifiable disease in Victoria under the Livestock Disease Control Act 1994, and must be reported within seven days.

What is strangles?
Strangles is a highly contagious infectious disease of the upper respiratory tract, caused by the bacterium *Streptococcus equi* subsp. *equi* (*S. equi*). Horses, ponies and donkeys may be affected. Infection occurs when susceptible horses come into contact with infected horses, either through direct contact or via indirect contact with contaminated materials such as feed and water buckets, tack, transport vehicles, stables, as well as in contact personnel’s hands and clothing. Strangles is an endemic disease that is more common in younger horses, but can occur in horses of any age. Outbreaks may follow the mixing of groups of horses or the introduction of new horses onto a property.

Clinical Signs
Strangles is usually readily recognised, and is characterized by an acute fever followed by the development of lymphadenopathy and mucopurulent nasal discharge. Disease severity varies depending on the immune status of the horse: younger horses are more likely to develop severe lymph node abscessation, whereas older horses usually exhibit milder clinical signs. Horses will usually have an elevated temperature that persists while lymph node abscesses are forming. Dysphagia, reluctance to eat and depression are common. Nasal discharge may be serous initially but rapidly becomes mucopurulent. The submandibular and retropharyngeal lymph nodes are commonly involved and become swollen and painful usually within a week after infection. Early signs of generalised painful oedema with serum oozing from the overlying skin may be apparent before the lymph node abscesses mature and rupture. A presumptive diagnosis is often made based on the characteristic clinical signs and control measures should be instigated before a definitive diagnosis is made. Guttural pouch empyema may occur secondary to retropharyngeal lymph node abscessation, which ruptures into the guttural pouch. In severe cases, retropharyngeal lymphadenopathy may result in tracheal compression and difficulty breathing. Affected horses do not typically cough. Occasionally, haematogenous or lymphatic spread of the bacterium may result in dissemination to other locations.
resulting in metastatic lymph node abscessation in the thorax and abdomen, or any other peripheral node ("bastard strangles").

**Epidemiology**

The first clinical sign of disease is an elevation in temperature which occurs 3 to 14 days after exposure, depending on the infective dose (the typical incubation period is 2-6 days). Nasal shedding of *S. equi* usually begins within 2-3 days of the onset of pyrexia and persists for 2 to 3 weeks in most cases. Therefore, as shedding does not begin until a day or so after the onset of pyrexia, newly identified cases (i.e. horses with elevated temperatures) should be isolated immediately before shedding starts. Guttural pouch infection is usually associated with much longer shedding - these cases typically become subclinical carriers which can shed intermittently for months to years. Clearance of the bacterium requires both systemic and local immune responses and usually occurs 2 to 3 weeks post infection. Although older horses may develop milder signs of disease, they will still shed virulent *S. equi* that can produce severe disease in younger animals. Disease severity is also dependent upon the challenge load and duration or exposure. In susceptible populations, disease morbidity is high (nearly 100%) but mortality is low (<10%). After recovering from disease, approximately 75% of horses develop solid, long lasting immunity (5 years or longer). Foals nursing from immune mares are usually resistant to *S. equi* infection until 3 months of age, thus infection in foals usually occurs after weaning.

The most obvious source of transmission is the characteristic purulent discharge, or contact with the contents from burst lymph node abscesses. Transmission may be via direct contact or indirect via fomites unless appropriate precautions are undertaken. Recovering horses and subclinical cases can also transmit disease. A recovered horse may be a potential source of infection for a minimum of six weeks after resolution of its clinical signs. Carriers are horses which are fully recovered but intermittently shed *S. equi*: these long term subclinical carriers are an important source of infection for susceptible horses. The source of bacterium in these carriers is usually the guttural pouch – development of chondroids (accretions of inspissated pus) are common in such cases. The bacterium does not survive more than a week in the environment, especially on surfaces exposed to direct sunlight. *S. equi* does not persist well with other soil borne flora, but the exact length of environmental persistence is unknown. Laboratory tests suggest environmental persistence on materials such as wood may be 48-63 days, with longer persistence in colder climates, although these results were not obtained from field trials.

**Diagnosis**

The definitive diagnosis of strangles is based on detection of *S. equi* by culture of nasal (or nasopharyngeal) swab samples, nasal washes or pus from abscesses. Nasopharyngeal swabs or washes are preferred over nasal swabs obtained from just within the nasal passages. The main bacteriological differential diagnosis is *S. zooepidemicus*. Nasal washes are more effective than nasal swabs to detect small numbers of bacterium. False negatives may occur in the incubation and early clinical stages as *S. equi* is not normally present on the mucosa until 24-48 hours after the onset of pyrexia. Culture can take several days before a result is available. Polymerase chain reaction (PCR) detects a specific gene sequence from the *S equi* genome. This rapid screening test is available at the Centre for Equine Infectious Disease at the University of Melbourne. Concurrent PCR and culture testing is recommended, as PCR does not distinguish between live and dead organisms. Although PCR is more sensitive than culture, false negatives can still occur. PCR of nasopharyngeal samples will tend
to be negative before guttural pouch lavages, as mucociliary clearance usually clears both organisms and DNA at the same time. Therefore to detect asymptomatic carriers, PCR of guttural pouch lavages is recommended. PCR can also be used as a screening tool to detect infection status before introduction of new horses onto a property, but the sensitivity of this practice is low. Serology (dual antigen ELISA) is useful for managing an outbreak of strangles. In an outbreak, horses should be divided into exposure groups based on clinical signs. Horses with clinical signs of strangles should be classed as exposed (RED), horses that have been in contact with horses with clinical signs should be grouped into a likely exposed (AMBER) group, while horses not exposed to clinically affected horses should be grouped into an unexposed (GREEN) group. ELISA testing can be used to test horses in the amber and green groups to determine if they have been exposed to the pathogen during the outbreak. ELISA testing can also be useful in diagnosis of bastard strangles and immune mediated complications such as purpura haemorrhagica. This test is available at the University of Melbourne laboratory. Carriers that are persistently infected but lack clinical signs can also be indirectly identified via ELISA. A blood sample taken on arrival onto a property can identify recently exposed or persistently infected horses. If negative, a second blood sample should be taken 2 weeks later to identify exposed horses that have seroconverted.

Vaccination and outbreaks

Development of immunity after vaccination takes approximately two weeks so protection may not be provided quick enough during an outbreak. Vaccination during an outbreak remains controversial and should be restricted to healthy horses in the green group that have not been exposed to infected horses (including their nasal discharge and associated fomites). Horses known to have had strangles within the previous year should not be vaccinated. As a general rule, it is best practice to not vaccinate in the face of an outbreak as this confuses the ELISA results and slows down control of the outbreak. As well, vaccinating horses with high antibody levels may contribute to the development of purpura haemorrhagica.

Control of outbreaks

Effective communication with the owner is vital to obtain a detailed history, determine the number of affected animals, prior movements on and off the property, geographical relationships of cases on a property and management practices that may impact disease control.

- If *S. equi* is suspected, horses with clinical signs or an elevated temperature should be immediately isolated (RED group) to minimize the risk of transmission to other susceptible animals. The remaining animals in the group (AMBER) should remain isolated in case they are incubating the disease.
- Ideally, movement of horses on and off the property should cease until the outbreak is controlled.
- Hygiene measures should be instigated, and horses segregated into RED, AMBER and GREEN areas. RED areas are contaminated areas where affected horses and animals in contact with infected horses are maintained. Avoid nose to nose contact between groups of animals. AMBER areas are for those horses that may have had contact with infected animals and could be incubating the disease. If common facilities must be used (e.g. yards and vet crush facilities during the breeding season) then animals should use the facility in order of GREEN, AMBER and RED groups and then thorough disinfection should be carried out.
- Monitor rectal temperatures twice daily to aid in the detection of new cases. Pyrexic horses should be isolated and tested serologically.
- Use effective hygiene practices. Personnel should wear protective clothing (including boot covers) and gloves. Wash hands between horses, soap free hand sanitisers can also be used. Particular care should be taken with hygiene measures during an outbreak to prevent inadvertent transfer from infectious horses and potential subclinical carriers to susceptible horses. Preferably have different people caring for infected and susceptible horses, if this is not possible deal with susceptible non-infected horses first. Have dedicated equipment, feed and water buckets for infected animals and disinfect.
- Disinfect equipment, tack and stables with appropriate disinfectants e.g. Virkon S®. The bacterium is susceptible to most disinfectants such as phenol, povidone-iodine, chlorhexidine and glutaraldehyde. Clean surfaces first and remove organic material and manure from stables first, then soak in disinfectant and allow to dry. Manure and waste materials including left over feed from infected areas should be isolated and composted (the bacterium are destroyed by heat). Don’t forget to disinfect water and feed buckets and troughs (daily disinfection is recommended), as well as transport vehicles and fences.
- Screen convalescent cases after clinical recovery using nasopharyngeal swabs or lavages at weekly intervals for several weeks following recovery with both PCR and culture. At least 3 sequential weekly nasopharyngeal swabs or lavages should be used to determine whether recovered horses are still infectious. Horses consistently negative (after 3 weekly tests) can then be moved into clean areas. Serology can be useful to determine that these horses have not been recently re-exposed by contact with a carrier animal.
- If a horse is outwardly healthy but positive on PCR or culture off nasopharyngeal swabs or lavage, check the guttural pouches. Scoping is recommended to determine if chondroids are present, and guttural pouch lavage can be used to obtain samples for PCR and culture.
- If a clinically normal horse tests positive on blood ELISA, guttural pouches should also be checked. Also, if the seroprevalence in a group of convalescent horses remains high then that group is likely to contain an asymptomatic carrier and the group should be investigated.
- Screen infected horses with blood ELISA starting 3 weeks after the resolution of all clinical signs to identify potential carriers. Unexposed horses can also be tested by sequential ELISA tests.
- Infected horses need to be isolated for approximately 6 weeks.
- Rest pastures used by infected animals for 2 weeks where practical. Before using potentially contaminated paddocks, drain and scrub all water troughs and feeders with disinfectant to remove potentially contaminated material.

Treatment

Appropriate treatment depends on the stage and severity of disease. Opinion is divided as to the need for antimicrobials. Mild cases often only need rest, good husbandry and nursing care. During an outbreak, immediate antibiotic treatment horses with fever may prevent focal abscessation but these horses can remain susceptible to reinfection and may not develop protective immunity. Antimicrobial use in otherwise clinically healthy horses with lymph node enlargement may delay lymph node maturation and rupture. Other symptomatic treatment such as with non-steroidal anti-inflammatories is often required. Horses with severe airway obstruction may require emergency procedures such as
a temporary tracheostomy and antimicrobial treatment is indicated in such cases. Penicillin is generally considered the drug of choice as *S. equi* shows consistent susceptibility to penicillin. Other antimicrobials such as cephalosporins and trimethoprim sulphonamides are generally less effective, and *in vitro* susceptibility to trimethoprim sulphonamides does not necessarily equate with susceptibility *in vivo*. Resistance to trimethoprim sulphonamides has recently been detected. Horses with gulletal pouch chondroids need repeated flushing and removal of the chondroids as well as both topical and systemic antimicrobials.

**Complications of Infection**

Approximately 20% of affected horses develop complications. Common sites of metastatic abscessation ("bastard strangles") include the lung, mesentery, liver, spleen, kidney and brain. This is more difficult to diagnose and antibody titres can be helpful in such cases. Extension of infection into the gulletal pouch is important as this is the most common site for prolonged shedding of the bacterium of organism. Immune mediated complications are also possible. Purpura haemorrhagica is an aseptic necrotising vasculitis characterised by oedema and petechial or ecchymotic haemorrhages. This occurs due to immune complex deposition in blood vessel walls, with horses with high antibody titres at most risk after both clinical infection and vaccination.

**Prevention**

When possible, newly introduced animals should be isolated for three weeks and screened by nasopharyngeal PCR and culture, or tested serologically. Vaccination is recommended but is not fully protective against the development of the disease, although it reduces disease severity. Current recommendations are that 6 monthly boosters are administered as the duration of immunity following vaccination is short.

**References**

