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Strangles

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Strangles is a bacterial disease caused by *Streptococcus equi subsp equi*, affecting horses, donkeys, and mules. The disease is highly contagious and often leads to outbreaks in groups of horses. Clinical signs are very variable, depending on infectious dose and individual immune status. This issue of *Large Animal Veterinary Rounds* discusses the pathogenesis, clinical presentation, treatment, and prevention of strangles in horses, based on a recent consensus statement published by the American College of Veterinary Internal Medicine (ACVIM). In addition, two cases of strangles are presented that illustrate diagnostic and treatment approaches to complicated cases.

Case 1

A 2.5-month-old Arabian filly is presented to the Western College of Veterinary Medicine (WCVM) for evaluation of strangles. There have been several cases on the farm and the filly has a history of nasal discharge and submandibular lymph node swelling of several days duration. She had been improving until the morning of presentation. The filly apparently has a good appetite, but regurgitates milk when suckling.

Physical examination reveals a filly in good body condition. She is febrile (38.9°C) and has a mildly elevated respiratory rate of 20 bpm. She holds her head and neck stretched out and has mucopurulent nasal discharge mixed with feed material (Figure 1). The filly exhibits respiratory stridor and distension of the right guttural pouch is visible and palpable in Viborg's triangle. Auscultation reveals a tracheal rattle and tracheal sounds referred over the lung field, but no abnormal lung sounds. Submandibular lymph nodes are enlarged and painful on palpation and a scab is evidence of a previous abscess rupture.

Significant bloodwork changes include:

- Leukocytosis $12.5 \times 10^9/L$ (normal range [NR] $4.3 - 11.5 \times 10^9/L$)
- Neutrophilia $8.75 \times 10^9/L$ (NR $1.8 - 7.2 \times 10^9/L$) and a mild left shift
- Mild monocytosis $0.75 \times 10^9/L$ (NR $0.1 - 0.5 \times 10^9/L$)
- Hyperproteinemia 83 g/L (NR 60 - 74 g/L)
- Mild hypoalbuminemia 30 g/L (NR 31 - 48 g/L)
- Hypergammaglobulinemia 53 g/L (NR 21 - 39 g/L).

Upper airway endoscopy shows moderate inflammation and swelling of the pharynx, as well as bilateral guttural pouch empyema. Upon distension of the right guttural pouch with lavage fluid, respiratory effort increases markedly and the procedure has to be interrupted. A tracheostomy is performed and an indwelling tracheal tube is placed. This relieves respiratory distress and allows for a thorough lavage of the guttural pouches with sterile saline. Because of the inspissated nature of the guttural pouch contents and the necessity for repeated lavage, 20 French Foley catheters are placed in both guttural pouches under endoscopic control. A semi-rigid plastic stylet is used to facilitate placement. The catheters are secured by inflating the balloon and suturing the catheter ends to the nose (Figure 2). The catheters are well tolerated over the next several days. Mild sedation causes the filly to lower her head during lavage, minimizing the risk of aspiration. The filly is treated with ceftiofur (Excenel®, 4 mg/kg IM BID) and flunixin meglumine (Banamine®, 1 mg/kg IV SID), daily guttural pouch lavage and regular cleaning of the tracheostomy tube and site. Re-evaluation by endoscopy on day 6 reveals a small amount of purulent material remaining in the left guttural pouch, while the right guttural pouch appears normal. The foal is discharged on day 7 for continued treatment with ceftiofur at home. At this time, mild dysphagia persists, but respiratory signs have resolved. Re-evaluation on day 16 shows a filly in good condition and growing normally. Endoscopy demonstrates a moderate amount of scarring in the left guttural pouch, while empyema has resolved completely in both pouches. The tracheostomy site is healing well. The filly is discharged with recommendations to discontinue antimicrobial treatment and re-evaluation for continued shedding of *S. equi* after 1 month.



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Figure 1: Severe mucopurulent nasal discharge in an Arabian foal (Case 1). Distension of the throat-latch area is visible.



Case 2

A 16-year-old Arabian mare is presented to the WCVM for evaluation of weight loss, nasal discharge, and abscessation of the face and udder. The horse was boarded at a breeding facility where strangles had occurred 3 months prior to presentation and has shown clinical signs since returning from the breeding facility.

Physical examination reveals a thin horse with firm, pitting, ventral edema cranial to the udder (Figure 3). Submandibular lymph nodes show scabs suggestive of previous abscessation. There is a bleeding wound at the site of a previous abscess on the right temple. Mucopurulent nasal discharge is present bilaterally. The left half of the mammary gland is firm, warm, and painful on palpation and a ruptured abscess is present high in the flank adjacent to the udder. Secretions expressed from the gland are white, thick, and contain blood clots. Secretions from the right half of the mammary gland are straw-coloured and clear. Rectal examination does not reveal any abnormalities; the mare is not pregnant.

Complete blood cell count (CBC) shows moderate leukocytosis ($14.3 \times 10^9/L$, NR = $4.3\text{--}11.5 \times 10^9/L$), neutrophilia, and monocytosis. Total solids (100 g/L , NR = $59\text{--}73 \text{ g/L}$) and

Figure 2: Foley catheters in place for repeated guttural pouch lavage (Case 1). The foal has a temporary tracheostomy tube in place due to severe respiratory distress on presentation.



Figure 3: 16-year-old Arabian mare with weight loss, mastitis, and guttural pouch empyema caused by strangles (Case 2). Notice ventral edema associated with mastitis.



fibrinogen (6 g/L , NR = $1\text{--}4 \text{ g/L}$) are elevated. Serum chemistry reveals hypoalbuminemia (25 g/L , NR = $31\text{--}48 \text{ g/L}$) and hyperglobulinemia (79 g/L , NR = $21\text{--}39 \text{ g/L}$).

Ultrasound examination of the udder shows fluid pockets (presumably abscesses) in the left half. A culture of milk obtained from the left teat yields *S. equi*, while culture of the right udder contents is negative. Abdominal fluid is collected by abdominocentesis to investigate the possibility of abdominal abscessation as a cause of weight loss. Abdominal fluid is cloudy and has a mildly elevated total protein concentration of 26 g/L . Cytology reveals mild nonseptic to pyogranulomatous inflammation with evidence of prior hemorrhage. A culture of abdominal fluid is negative; however, endoscopy of the guttural pouches shows firmly inspissated purulent material in the left guttural pouch and empyema in the right pouch.

The mare is treated with intravenous sodium penicillin ($20,000 \text{ IU/kg IV QID}$) and flunixin meglumine (1.1 mg/kg IV SID). The udder is cold-hosed and the left half of the udder stripped out once daily. On day 3, intramammary treatment of the left udder with a combinatory preparation of penicillin, dihydrostreptomycin, and hydrocortisone (Special Formula 17900-forte[®]), is also performed. The facial wound is cleaned daily and treated with topical hibitane ointment. Both guttural pouches are lavaged with sterile saline.

The mare's comfort and appetite improves slightly while at the WCVM and she is discharged at the owner's request on day 4. Treatment at home is continued with intramuscular injections of ceftiofur and oral phenylbutazone. Cold-hosing and daily stripping of the udder, as well as care of the facial wound, is further recommended. Dietary recommendations include senior feed with the addition of oil to increase caloric intake. Two weeks after discharge, the mare is doing well and gaining weight; however, another abscess has ruptured above the left teat. A follow-up examination is recommended. The mare is reportedly doing well 7-months following presentation, but has not returned for re-evaluation.

Etiology and pathogenesis

Strangles is a highly contagious bacterial infectious disease caused by *S. equi* that affects horses, donkeys, and mules. The primary mode of transmission is the transfer of large numbers

of *S. equi*, shed in respiratory secretions of infected horses. Data on environmental survival of the organism under natural conditions are lacking. When applied as a pure culture, viable bacteria could be recovered for 63 days at 2°C from a wooden surface, and for 48 days at 20 °C from a glass or wooden surface.¹ The potential effects of interaction between *S. equi* and other environmental organisms were not investigated in this study; however, *S. equi* is sensitive to bacteriocins from environmental bacteria and may not readily survive in the presence of other soil-borne flora.²

Infection with *S. equi* occurs via the respiratory tract and rapidly spreads to the regional lymph nodes, causing upper respiratory catarrh and lymphadenitis. The bacteria cause massive influx of neutrophils (abscess formation), but resist phagocytosis such that resolution of infection is dependent on abscess drainage. Hematogenous and lymphatic spread to additional sites (eg, intra-abdominal lymph nodes, lungs, liver, spleen, kidney, and brain) are potential complications of *S. equi* infection. The notion that antimicrobial treatment suppresses immune response and favours the development of such “bastard strangles” is widely held, but has not been substantiated.

The incubation period is 3–14 days and fever is the first detectable clinical sign. Incubation periods vary in length, depending on the infectious dose and the patient’s immune status.² Shedding of bacteria from infected horses does not begin until 24 to 48 hours following onset of fever. Monitoring of horses at risk and immediate isolation of febrile horses can help reduce infection spread. Shedding typically persists for 2–3 weeks following resolution of clinical signs and bacteria can no longer be isolated after 6 weeks in most affected horses.^{3,4} Horses with persistent guttural pouch infection can shed bacteria for months-to-years and are probably important sources of strangles outbreaks.² Up to 10% of horses showing guttural pouch empyema during the course of strangles may develop chronic guttural pouch infection and carrier status.²

Strangles is often referred to as a “childhood disease” of horses, based on its primary occurrence in younger horses and the often milder or even subclinical manifestations in older horses with better immunity from previous exposure. However, repeated infection with strangles is possible and has occurred within 6 months following naturally occurring disease.⁵ Subclinically infected horses shed bacteria and can contribute to outbreaks in susceptible animals. The importance of chronically infected, clinically healthy horses in the development of strangles outbreaks is being increasingly recognized.²

Clinical signs

Uncomplicated cases of strangles must be differentiated from those in which complications occur. Typical clinical signs of uncomplicated strangles include fever, depression, serous to mucopurulent nasal and ocular discharge, and swelling and abscessation of submandibular lymph nodes. Swelling of retropharyngeal lymph nodes may be apparent during endoscopy of the guttural pouches, but is rarely detected on physical examination. Pain associated with pharyngitis and laryngitis may lead to inappetence or anorexia, dysphagia, and an extended head carriage. Dysphagia, secondary to inflammation of cranial nerves transecting the guttural pouch, may occur in horses with guttural pouch empyema. Treatment other than supportive care and rest is not considered necessary and is not recommended in uncomplicated cases. Prognosis for uncomplicated cases is good if adequate time for recovery is allowed.

Complications of strangles are variable and occur in approximately 20% of infected patients.^{6,7} Complications include those associated with the spread of infection to sites other than the head and neck, immune-mediated processes such as purpura hemorrhagica, myopathies, and agalactia.² Severe guttural pouch empyema, guttural pouch chondroid formation, severe enlargement of retropharyngeal lymph nodes and lymph nodes at the thoracic inlet causing airway obstruction (hence the name “strangles”) or temporary laryngeal hemiplegia due to recurrent laryngeal nerve damage, can also be regarded as complications. Identification of complications warrants aggressive treatment with antimicrobials, anti-inflammatory drugs, and supportive care. Complications worsen the prognosis for affected horses and increase the case-fatality rate.⁶ Horses suffering from strangles should be thoroughly examined to identify and address potential complications as early as possible. Repeated examination is of special importance in horses with prolonged recovery times and persistent clinical or laboratory abnormalities. Chronic guttural pouch empyema may not cause overt clinical signs, but is associated with intermittent coughing and nasal discharge in about 50% of affected horses.²

Diagnosis

Diagnosis of strangles is often based on history and clinical signs; however, definitive diagnosis requires culture of the organism. This is typically achieved by collecting nasal secretions, abscess content (eg, aspiration of submandibular lymph nodes) or by performing nasopharyngeal or guttural pouch lavage. Routine blood work (CBC and chemistry) may be helpful to confirm inflammatory changes, monitor patients during treatment and/or recovery, and to identify potential sites of abscessation (eg, liver). Radiographic and, particularly, endoscopic evaluation is indicated when guttural pouch empyema or chondroid formation is suspected. If spread of infection to other sites is suspected, additional diagnostic tests such as thoracic radiography and ultrasonography, abdominal ultrasonography, evaluation of tracheal or abdominal fluid, computed tomography, and magnetic resonance imaging⁸ may be indicated.

Polymerase chain reaction (PCR), based on the *SeM* gene, which encodes for the antiphagocytic M-protein of *S. equi*, is a helpful adjunct to culture.⁹ PCR is more sensitive than culture and has a shorter turnaround time; however, PCR does not differentiate between live and dead organisms. A positive PCR result should be confirmed by culture. Potential applications of PCR testing include detection of asymptomatic carriers, establishment of *S. equi* infection status prior to transportation and following transport prior to commingling, and determination of the successful elimination of *S. equi* from the guttural pouch.²

A commercially available ELISA test (EBI) may be useful in diagnosing recent, but probably not current, strangles infection, to determine the need for booster vaccination, as well as to support a diagnosis of purpura hemorrhagica and metastatic abscessation.² Guidelines for interpretation of ELISA results are available.² Individual immune responsiveness must be considered when interpreting test results and retesting may be required if equivocal results are obtained.

Treatment and control

Treatment of uncomplicated strangles is typically limited to providing supportive care. Horses should be rested and provided

Figure 4: Instruments for guttural pouch lavage. MILA self-retaining guttural pouch catheter kit (top), Chambers catheter (middle), and Foley catheter (bottom).



easy access to good quality feed and water. Soft, palatable, and easily digested feeds should be available for inappetent horses and for those experiencing pain when eating. Pain control and anti-inflammatory therapy may aid in maintaining appetite and attitude, but should be used judiciously. Hot-packing and/or poulticing of abscesses may provide pain relief and hasten abscess rupture. Avoid the premature lancing of an abscess, ie, prior to the development of “soft spots,” because it is of little therapeutic benefit.

Antimicrobial therapy is not indicated for uncomplicated cases of strangles as long as the horse remains comfortable and is eating well. On the other hand, anorexia and severe depression, as well as complications (eg, pneumonia or guttural pouch empyema), are indications for antimicrobial therapy. Penicillin is considered the treatment of choice and, to date, there are no reports of resistance of *S. equi* against penicillin.² Cephalosporins, macrolides, and trimethoprim-sulfa are alternatives to penicillin and, according to anecdotal reports, have been used successfully.² Use of drugs other than penicillin should be based on sensitivity testing of the organism and potential discrepancies between *in vitro* and *in vivo* efficacy should be considered.²

Guttural pouch empyema is best treated by a combination of antimicrobial treatment and pouch lavage. Guttural pouch lavage using endoscopy offers the advantage of visual control and allows for evaluation of treatment progress. Alternatively, blind passage of an appropriate rigid catheter (Chambers catheter) is relatively easy and usually well tolerated under light sedation (Figure 4). The guttural pouch opening is located at the level of the medial canthus. Marking the distance on the catheter from the proximal aspect of the nares to the medial canthus aids in blind placement. A slightly angled catheter tip and careful twisting to open the guttural pouch “flap” is required. Caution should be exercised not to manipulate the catheter excessively once it has entered the pouch, to avoid potential damage to vascular and nerve structures in the pouch. Indwelling catheters for repeated lavage are an excellent alternative to repeated passage of an endoscope or guttural pouch catheter. Self-retaining, coiled guttural pouch catheters are probably the least traumatic (Figure 4). Foley catheters are a cheaper alternative and have been used with good success in our clinic (Case 1). The larger diameter of the Foley catheter may increase the risk of irritation and scarring of the guttural pouch openings. Care should be taken to prevent an excessive length of catheter protruding from the nostril.

Guttural pouch lavage with sterile, isotonic solutions is usually sufficient to remove pus and inspissated material. Use of a gelatin/penicillin mix¹⁰ and 20% acetylcysteine solution for treatment of guttural pouch empyema has also been described. Chondroids may be broken up with transendoscopic devices such as snares or, in rare cases, may require surgical removal.

Treatment of complications depends on the affected site and the overall condition of the patient. Patients with severe lymph node enlargement and resulting airway obstruction may require a temporary tracheostomy until swelling and obstruction have resolved. In select cases, abdominal abscessation may be amenable to surgical treatment by marsupialization or resection; however, prolonged antimicrobial therapy is the treatment of choice in most cases. Prognosis for cases of abdominal abscessation is guarded and long-term complications include adhesion formation and chronic colic. Diffuse peritonitis due to abscess rupture carries a grave prognosis and is usually not amenable to treatment.

Purpura hemorrhagica

Aside from infection spread to sites other than the upper respiratory tract, purpura hemorrhagica is the most common complication of equine strangles. It is an aseptic, necrotizing vasculitis thought to be caused by the deposition of immune complexes in blood vessel walls.² Purpura hemorrhagica typically develops 1-3 weeks following clinical manifestations of strangles, and active *S. equi* infection may still be present when purpura hemorrhagica is diagnosed. Purpura hemorrhagica can also occur following subclinical infection with *S. equi* and in response to vaccination.⁶ Purpura hemorrhagica is not exclusive to strangles; almost 60% of cases in one report occurred secondary to organisms other than *S. equi*, or without an identifiable underlying cause.¹¹

Clinical signs include edema of the distal limbs and ventral abdomen, lameness, mucosal petechiation, anemia, fever, and depression. Edema is pitting, painful, and warm and, in severe cases, oozing of serum and/or sloughing of affected skin may occur. Clinical pathologic abnormalities include leukocytosis with neutrophilia, mild-to-moderate anemia, and hyperfibrinogenemia. Platelet counts are typically normal. Vasculitis can affect internal organs as well as skin, and evidence of organ dysfunction may be present on clinical or clinical pathologic examination. Diagnosis is typically based on clinical signs and history of recent infection with *S. equi*. High antibody titers against the M-protein of *S. equi* or demonstration of active infection by culture can support a diagnosis. Skin biopsy classically shows leukocytoclastic vasculitis.

Treatment includes antimicrobials (penicillin) to decrease antigen load, along with immune suppressive doses of corticosteroids (dexamethasone). Non-steroidal anti-inflammatory drugs may be necessary to control pain, but should be used cautiously in combination with corticosteroids. Supportive care is of paramount importance and includes adequate hydration and nutrition, hydrotherapy of vasculitic limbs, bandaging, and rest. Treatment should be aggressive until clinical signs begin

to improve, at which time, corticosteroid dosage should be tapered gradually as long as clinical signs remain under control. Prognosis is guarded; potential sequelae include considerable sloughing of skin, laminitis, organ failure, and death.

Prevention/vaccination

Prevention strategies against strangles should be implemented in facilities experiencing significant movement of horses, such as breeding and training facilities. Common sense hygiene measures to minimize the potential spread of infectious organisms among horses should be implemented in any multi-horse facility, especially if animals from different sources are housed in close proximity to each other. Strategies to specifically prevent strangles depend on the individual situation, but might include quarantining new arrivals for 3 weeks, screening for *S. equi* infection or carrier status by repeated nasopharyngeal and/or guttural pouch lavage, and culture or PCR.²

Normal mucociliary clearance and mucosal immunity (IgA) are probably very important for protection against strangles; however, both mucosal and systemic antibodies are thought to be necessary for optimum protection.² Most horses develop solid immunity following natural infection with *S. equi* and, in almost 75% of infected horses, immunity lasts for approximately 5 years.² On the other hand, re-infection within 6 months of naturally-occurring disease has been reported in foals.⁵ Immune mares have antibodies in their milk that protect suckling foals until weaning.²

Two vaccines against strangles are currently available in Canada. An extract (bacterin) vaccine (Strepguard[®]) containing the adjuvant Havlogen[®] is sold by Intervet for intramuscular injection. Company recommendations include administration of 2 doses, 3 to 4 weeks apart, and an annual booster, if risk of exposure exists. Foals vaccinated before 3 months of age should receive a third dose when 6 months old; pregnant mares should be given a booster 1-month prior to foaling. Swellings at injection sites have been reported after use of the vaccine. While efficacy of extract vaccines is generally low,² one study reported a 50% reduction in the clinical attack rate of vaccinates receiving 3 injections.¹² Based on evidence of short-lived immunity after natural infection in some horses,⁵ twice-yearly booster shots may be of benefit in horses at high-risk of exposure.

An intranasal, modified live vaccine (Pinnacle IN[™]) is available in Canada. Company recommendations include 2 initial vaccinations, 2 to 3 weeks apart, followed by annual boosters. Because there are reports of abscess formation attributed to the contamination of injection sites, additional injected vaccines should be administered on a different day or, at least, prior to intranasal vaccination. Hands and equipment should further be disinfected after use of the vaccine. No data exist on the efficacy and potential interaction between this and other intranasal vaccines, or on colostral antibody concentrations following intranasal vaccination of broodmares.² Side effects of the vaccine include residual virulence leading to transient upper respiratory signs (nasal discharge and lym-

phadenopathy), as well as purpura hemorrhagica in hypersensitive horses. Shedding of infectious particles following vaccination is also of concern in horses developing clinical signs of strangles subsequent to vaccination. It is possible to discern the vaccine strain from wild-type strains of *S. equi* by genetic testing, and suspected vaccination breakthroughs should be reported to the manufacturer for further investigation.

Vaccination during strangles outbreaks of horses with a history of strangles and/or purpura hemorrhagica are issues frequently faced by equine practitioners. ACVIM recommendations include no vaccination of horses known to have had strangles within the previous year or those with active clinical signs of strangles using the extract intramuscular vaccine.² Little information is available regarding the efficacy and safety of vaccines in outbreak situations, and expected benefits and potential risks should be weighed carefully.

Outbreak investigations and control

Management of a strangles outbreak is challenging and requires close cooperation between veterinarians and farm personnel. Management strategies depend on the individual situation and should be discussed with all people involved to maximize adherence to protocol. Potential limitations should be identified and addressed early on to prevent frustration and a false sense of security. General guidelines for management strategies of strangles outbreaks as published in the ACVIM consensus statement² include:

- Suspend all horse movement on and off the premises and immediately implement hygiene measures.
- Effectively segregate affected and exposed (infectious) from unexposed (noninfectious) animals and prevent cross-contamination between “clean” and “dirty” areas. This includes physical separation of animals to prevent any contact with respiratory secretions; separation of all equipment, including cleaning equipment, tack, and trailers; and disinfection of hands and change of clothing between groups. Ideally, separate personnel should handle infectious and noninfectious horses. Alternatively, noninfectious horses should be handled prior to infectious ones.
- Although rare, infections with *S. equi* in immune-compromised persons have been reported.¹³ Therefore, personnel handling infectious horses should be aware of the potential zoonotic risk and immune-compromised or debilitated persons should not have contact with infectious animals.
- Record rectal temperatures at least once daily; promptly segregate and appropriately treat new cases.
- Establish a bacteriological screening method to decide when horses can be moved from “dirty” to “clean” areas. Ensure that infection is not spread inadvertently during procedures such as nasal swabbing or lavage.
- Evaluate all clinically affected, recovered horses and their contacts for potential shedding of organisms prior to opening the premises following an outbreak. This is best achieved by repeated nasopharyngeal and/or guttural pouch lavage at weekly intervals until 3 consecutive negative cultures and/or PCR tests are available. Endoscopy

and/or lavage and culture of the guttural pouches should be performed in any horse exhibiting clinical signs consistent with guttural pouch empyema, and in horses culturing positive on nasopharyngeal lavage.

- Appropriately treat and continue follow-up evaluations of all animals identified as carriers.¹⁰

- Appropriately clean and disinfect all facilities and equipment following an outbreak. Discarding of contaminated equipment should be considered where financially feasible. A thorough cleaning of surfaces should be followed by treatment with a disinfectant effective against *S. equi*, or by steam treatment and drying. Manure and waste feed from affected animals should be composted in an isolated area to allow for destruction of bacteria by heat. Pastures exposed to infectious animals should be rested for at least 4 weeks.

Summary

Strangles occurs commonly in horses and the severity of clinical signs varies considerably. While treatment may be challenging in individual complicated cases, the bigger challenge lies in the control and prevention of outbreaks, and in the identification and appropriate management of suspect carriers. Vaccination against strangles is widely used; however, there is relatively little evidence available about its efficacy in preventing naturally occurring disease. Potential side effects of vaccination must be weighed carefully against expected benefits.

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