Hot to Trot

Reminders…….

• Should anyone require our assistance, the Afterhours Emergency Line Phone number is (605) 281-0493. If you are calling after hours, please use the Emergency Line first, and then call the office as we may be working on a call at the clinic. THANKS! WE APPRECIATE YOUR BUSINESS!!

• CHECK US OUT ON THE WEB www.hosmervetclinic.com AND ON OUR FACEBOOK PAGE!!

• RETURNS—we are very limited on the items we can return to our distributors. Therefore, all returns will be considered on a case-by-case basis. We are sorry for the inconvenience.

EQUINE HERPESVIRUS (EHV-1 AND EHV-4)

“EHV-1” and “EHV-4” are closely related equine herpesviruses that can cause respiratory disease, abortion and neurological disease, with a dormant lifelong infection which can suddenly re-activate and result in renewed viral shedding (“EHV-3” is a venereal form of herpes, and is mainly transmitted via sexual intercourse.) EHV-1 and EHV-4 are endemic or present in most horse populations throughout the world. Like EHV-4, EHV-1 can cause respiratory symptoms, but EHV-1 is also noted for its greater ability to cause abortions and neurological disease. EHV-1 neurologic or paralytic disease, also known as “EHN” (equine herpes virus myeloencephalopathy) is unique from other equine neurologic diseases, in that it can be spread by aerosol transmission directly from horse to horse without a vector such as a mosquito, making it a disease for concern for race tracks and horse shows.

EHV-1 and EHV-4 can be directly transmitted between horses by nasal or ocular discharges, aborted fetal material, contaminated hands, equipment or tack, or simply by direct aerosol transmission of the viral particles from horse to horse. The virus has an uncanny ability to lie dormant within an animal, so that no symptoms may be seen until some type of stress triggers its reactivation. Once a horse is an EHV-carrier, it remains infected with the virus throughout its life, which means that the virus can suddenly reactivate and cause disease symptoms, or that the horse can shed the virus sporadically, infecting nearby horses. These “silent carriers” who shed the disease without displaying symptoms can be a danger to any unprotected horse.

Vaccination has been proven to reduce clinical signs, and to decrease viral shedding. The reduction in viral shedding may reduce the incidence of abortion. Although no vaccine has been proven effective against equine herpesvirus neurologic disease, it is important to take measures to prevent or reduce the transmission of EHV-1 as part of a comprehensive protection strategy. Management is a very important aspect in controlling EHV disease, coupled with a total herd vaccination program. The main aims of EHV control should be the reduction of clinical respiratory signs and the reduction of virus shedding.

The incubation period of EHV-1 infection is HIGHLY VARIABLE, depending on the host, on the virulence of the virus, and on environmental and other factors such as stress. The AVERAGE incubation period is 4 to 7 days, with the majority of cases being 3 to 8 days, but with some taking up to 14 days. When neurological disease occurs, it is typically 8 to 12 days after the primary infection involving fever. In most cases, horses exposed to EHV-1 will develop a fever and possibly nasal discharge and then go on to recover.

All new horses entering farm premises should be quarantined for 14-21 days, and their temperatures taken daily. Hygiene is essential, as viral particles could be transmitted between horses if they are carried on equipment, or on handler’s clothing. Following transportation, all trailers should be disinfected. Any horses displaying respiratory symptoms should be isolated until diagnosis, as the virus is very labile and cannot travel any great distance. Aborting mares should also be placed in isolation.

The initial clinical signs of the infection may be nonspecific and include fever of 102°F or greater. Fever may be the only abnormality observed. Other presenting signs may be combinations of fever and respiratory symptoms of nasal discharge and cough. Some horses have reddish mucous membranes. Horses with neurological disease caused by EHV-1 infection can soon become uncoordinated and weak and have trouble standing. Difficulty urinating and defecating may also occur. Often the rear limbs are more severely affected than the front. Signs of brain dysfunction may occur as well, including extreme lethargy and coma-like state.

Mares infected with EHV-1 abort precipitously with no impending signs, and evidence of the previous respiratory tract infection is usually not observed. The placenta is expelled along with the fetus that is often still enveloped in its amniotic membrane. At the time of abortion, the fetus has usually just died from asphyxia associated with sudden separation of the placenta from the endometrium that precedes fetal expulsion. The aborted fetus possesses high levels of virus and extensive histopathological evidence of multi-organ infection. Almost all EHV-1 abortions occur during the last four months of gestation. Most abortion occurrences associated with EHV-1 infection involve only one or two mares in a group. However, epidemic abortigenic disease (abortion storms) claiming high percentages of the potential foal crop also occurs. Once a mare has aborted, her future reproductive potential is not compromised; most mares conceive successfully shortly after abortion and foal normally the following year.

If your horse develops fever, respiratory signs or neurological signs immediately notify your veterinarian and do not move the horse or horses in the immediate area. Alert those who have horses in the adjacent area to cease all movement of horses in and out of the facility until a diagnosis is confirmed by testing. If horses are exposed and then travel to a new stable or show, the infection can spread to other horses at that new location.

EHV-1 does not persist in the environment for a long time, but disinfection of premises, stalls, trailers and so forth is indicated. If you handle a horse with EHV-1 and don’t wash hands or change clothing, you may spread the infection to other horses. A solution of

DID YOU KNOW? While exercising, oxygen consumption of the horse increases 30X compared to a horse at rest.

DID YOU KNOW? Inhaled breath of the horse at 44 degree F. outside the nostrils warms to 87 degrees F. within 1/100th of one second.

March 2014

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Strangles is characterized by abrupt onset of fever followed by inflammation of the upper respiratory tract lining. There is also mucopurulent nasal discharge and acute swelling with subsequent abscess formation in submandibular (lower jaw) and retropharyngeal (throat latch area) lymph nodes. The name strangles was coined because affected horses sometimes were suffocated by enlarged lymph nodes that obstructed the airway. Severity of disease varies greatly depending on the immune status of the animal. Older horses often exhibit a mild form of the disease characterized by nasal discharge, small abscesses, and rapid resolution of disease, whereas younger horses are more likely to develop severe lymph node abscessation that subsequently opens and drains.

Fever is the first clinical sign and persists as lymph nodes enlarge and the abscesses mature. Inflammation of the pharynx causes difficulty eating and the affected horses may become reluctant to eat and often stand with their neck extended. Attempts to swallow food and water may be followed by reflux of these and it coming back out the nostrils. Depression and listlessness are common signs. Pharyngitis, Laryngitis, rhinitis may occur and contribute to bilateral nasal discharge, which is serous initially and rapidly becomes mucopurulent.

Increased lymph node size is a major clinical sign. The submandibular and retropharyngeal lymph nodes are about equally involved in strangles infections and become swollen and painful about 1 week after infection. The first sign of lymph node swelling is often hot, diffuse, painful edema. Serum may then ooze from the overlying skin for several days as the lymph node abscesses mature before rupturing to drain creamy pus, which does not have a foul odor. Other lymph nodes of the neck are also frequently involved and may also abscess. Retropharyngeal lymph nodes may drain into and cause empyema of the guttural pouch. Natural draining of these deeper abscesses to the skin may take several days or weeks, and the swelling can exert pressure on the pharynx, larynx, trachea and esophagus, causing severe dyspnea, stridor, and dysphagia. Periorbital abscesses can cause marked swelling of the eyelids. Abscesses of the lymph nodes at the thoracic inlet can cause severe tracheal compression, asphyxia, and death. Coughing is not a significant feature in many cases, although some horses develop a soft, moist cough that becomes more productive and increasingly severe as the disease progresses. Expansion of large quantities of pus from the nose or mouth with coughing usually indicates empyema of the guttural pouch.

Although strangles predominately involves the upper airway, including the guttural pouches and associated lymph nodes, metastasis to other locations occasionally occurs. Spread may be through the bloodstream or via lymphatic channels, which results in abscesses in lymph nodes and other organs of the thorax and abdomen. This form of the disease has been known as “bastard strangles.” Metastasis to the brain has also been recorded.

Nasal shedding of strangles usually begins 2 to 3 days after onset of fever and persists for 2 to 3 weeks in most animals. Some animals never shed and in others, shedding may persist much longer should infection persist in the guttural pouch. Approximately 75% of horses develop a solid, enduring immunity to strangles after recovery from the disease. Milk from mares that have recovered from strangles contains IgGb and IgA with specificities similar to those found in nasopharyngeal mucus of convalescent horses. Suckling foals therefore benefit from the protective effects of this antibody until weaned. Colostral antibodies ingested during the first 24 hours of life recirculate to the nasopharyngeal mucosa, thus providing an additional source of protection to the foal during its first weeks. Foals that suckle immune mares are usually resistant to infection until weaning.

Appropriate treatment of horses with strangles usually depends on the stage and severity of the disease. Veterinary opinion as to whether or not to use antibiotic treatment remains markedly divided. However, the majority of strangles cases require no treatment other than proper rest and a dry, warm stall and provision of soft, moist, and palatable food of good quality while letting the disease run its course. Food and water should be easily accessible to the horse. The overall complication rate with strangles is about 20%. A variety of complications can occur as a result of strangles. These can be generally grouped as:

- Those associated with the spread of infection from the head and neck region to other locations.
- Immune-mediated processes, including purpura hemorrhagica and myopathies.
- Agalactia

Control of outbreaks it is suggested that:

- All movement of horses on and off the affected premises should be stopped and segregation and hygiene measures implemented immediately.
- Cases of strangles and their contacts should be maintained in well-demarcated “dirty” quarantine areas.
- Rectal temperatures should be taken at least once daily during an outbreak to detect, promptly segregate, and possibly treat new cases.
- The aim of the control strategy, following bacteriological screening, is to move horses from the “dirty” to “clean” areas where non-affected and noninfectious horses are kept.
- Every care should be taken to ensure very high hygiene standards throughout the premises and for the duration of the outbreaks.

1 part chlorine bleach to 10 parts water is effective for decontaminating equipment and environment.