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Botulism in Horses

***Botulism is a fatal neurologic disease caused by toxins* Clostridium botulinum produces.**

Overview

Botulism is a fatal neurologic disease caused by toxins the anaerobic, spore-forming, soil-dwelling bacterium *Clostridium botulinum*. Eight different toxins are produced by *C. botulinum* produces, but type B is associated with the majority of botulism cases in North America. Botulinum toxins are extremely potent, and horses are particularly sensitive to these toxins. Thus, clinical signs of disease (even death) can occur very rapidly post-exposure.

Horses are exposed to the toxins in one of three ways:

1) Ingesting the toxins directly. This is also called forage poisoning and is the primary form of botulism in adults.

2) Ingesting spores and absorbing toxins from the gastrointestinal tract. This is also referred to as toxicoinfectious botulism or shaker foal syndrome. Toxicoinfectious botulism is the second most common form of botulism in horses, and this arises when horses ingest the bacterium from soil, and it colonizes the gastrointestinal tract. As it grows inside the body, the bacterium produces the toxin, and signs of disease become apparent as toxin is absorbed into the bloodstream from the intestinal tract.

3) Contamination of wounds with *C. botulinum* spores and absorbing the toxins systemically.

Regardless of the route of exposure, once the botulinum toxin enters the bloodstream, it circulates throughout the body and enters various motor nerve cells (neurons). The toxins then travel to the end of these neurons (where they innervate muscle tissues) and prevent the transmission of the nerve impulses to the muscle cells. As a result, the horse becomes weak and potentially paralyzed.

MATHEA KELLEY

When forced to stand, foals with botulism rapidly develop muscle tremors (hence the term 'shaker foal') and flop to the ground in lateral recumbency.

Clinical Signs

Early clinical signs of botulism in adult horses include drooling, dropping food, dysphagia (difficulty swallowing), and inappetance/anorexia, as well as pacing, weakness, exercise intolerance, muscle tremors, depression, lying down more frequently or for longer periods of time than normal, and recumbency (inability to rise). A characteristic sign of botulism prior to onset of recumbency is reduced tongue strength and slow or absent tongue retraction.

In foals clinical signs include an increased amount of time lying down. When forced to stand, they rapidly develop muscle tremors (hence the term "shaker foal") and flop to the ground in lateral recumbency. Additional signs include drooling, weak eyelid tone, a tongue that is easily pulled from the mouth and only slowly retracted, constipation, and ileus (lack of intestinal contractions). Affected foals are otherwise bright and alert. Foals are typically between 2 and 5 weeks old, but cases in foals as old as 8 months have been reported.

If left untreated the toxin blocks nerve transmission to other muscles throughout the body—including the diaphragm—resulting in respiratory arrest.

Diagnosis

Call your veterinarian immediately so he or she can examine any horse exhibiting neurologic signs. There are multiple diseases that cannot be easily differentiated from botulism based on clinical signs alone, including rabies—a potentially fatal neurologic disease capable of infecting humans. Other diseases to consider are equine protozoal myeloencephalitis (EPM), the viral encephalitides (e.g., Eastern, Western, and Venezuelan equine encephalitis), the neurologic form of equine herpesvirus-1, West Nile virus, wobbler syndrome (cervical vertebral myelopathy), white muscle disease, and hyperkalemic period paralysis, among others.

A definitive diagnosis of botulism can only be achieved by demonstrating the presence of the botulinum toxin in the bloodstream, finding *C. botulinum* spores in the gastrointestinal tract and/or feedstuffs, or identifying an antibody response to *C. botulinum*.

Since each test is challenging and time consuming, botulism is generally diagnosed by ruling out other potential diseases. Complete blood counts (CBCs) and serum biochemistry generally show few or no changes early in the disease. Response to treatment is also a common method of "confirming" a diagnosis of botulism.

Treatment

Animals with a slow onset and mild course of disease can survive without treatment, but botulism is usually fatal if left untreated. The treatment of choice is the intravenous administration of a single dose of botulism antiserum. The antiserum contains neutralizing antibodies that bind to the botulinum toxin circulating in the horse's bloodstream. The antiserum has no

effect on the toxin that has already blocked the junction between neurons and muscle cells. As such, intensive nursing care is required for affected horses until new neuromuscular junctions are created, which takes approximately 10 days.

Nursing care involves confining the horse to a padded stall, providing food and water (or milk to foals) via nasogastric intubation, muzzling horses with an appetite to minimize the chances of aspiration pneumonia, administering mineral oil as a cathartic (empties the bowels) to prevent a large colon impaction, catheterizing the urinary bladders of horses unable to urinate, maintaining recumbent horses in a sternal position, and minimizing the development of pressure sores.

Prognosis

Prognosis depends on the dose of botulinum toxin and whether or not the horse is recumbent. Horses that remain standing often regain the ability to swallow within three to seven days after antitoxin administration. A full recovery for these horses typically occurs in about one month.

Once a horse has been recumbent for 24 hours, the prognosis is poor. Recumbent foals have a better prognosis than adult horses, and many affected foals can stand again seven to 10 days after treatment, assuming intensive nursing care is provided. Horses and foals that recuperate tend to have a full recovery and show no residual neurologic deficits.

Prevention

At present there is only one vaccine available against *C. botulinum* type B. The American Association of Equine Practitioners recommends vaccinating broodmares annually four to six weeks prior to foaling. The antibody levels in a vaccinated mare's colostrum are thought to protect the foals against shaker foal syndrome for up to eight weeks post-partum. Foals that are considered at high-risk of contracting botulism can be vaccinated. Three doses administered four weeks apart can be given as early as 2 weeks of age. Work with your veterinarian to develop the most appropriate vaccine schedule for your horse(s).

Vaccination against *C. botulinum* type B does not protect against the other seven botulism toxins.

FAST FACTS

- ***Botulism*** is a fatal neurologic disease of horses caused by *Clostridium botulinum*-produced toxins.
- **C. botulinum *type B*** is associated with the majority of equine botulism cases.
- Horses are exposed to the botulinum toxin through forage contamination, ingestion of the toxin producing spores, and wound contamination.
- ***Classic clinical signs*** include an inability to swallow, weakness, and recumbency.
- ***Diagnosis*** is challenging and often reached after ruling out all other potential causes of neurologic disease.
- Affected horses are treated with a single intravenous dose of botulism antiserum.
- ***Prognosis*** is variable and depends on the dose of toxin the horse received and severity of disease.
- ***A vaccine*** against *C. botulinum* type B is commercially available and recommended for at-risk horses and foals.