



EQUIPLAS[®] B

BOTULISM AND ITS PREVENTION

BOTULISM TYPE B

Botulism is a neurotoxic disease caused by the neurotoxins produced by *Clostridium botulinum*. Direct ingestion of preformed toxin in food (hay, grain or silage) or water; ingestion or inhalation of spores (toxioinfectious form); or in rare cases, clostridial wound infections can cause Botulism. Basically, botulism is a flaccid paralysis that results from the neurotoxin acting on the peripheral rather than the central nervous system.

CAUSATIVE ORGANISM

Clostridium botulinum, one of the most potent toxins known, is a gram positive, anaerobic, spore-producing rod.

DISEASE

Clostridium botulinum Type B is most commonly (but not exclusively) found in the soils of Kentucky and the eastern seaboard, while Type C is more commonly found on the West Coast. Shipments originating out of area containing contaminated feedstuffs can result in outbreaks in atypical locations. Horses are extremely sensitive to Type B and Type C botulism toxin. Levels of circulating neurotoxin able to kill a horse are often too low to be detected by standard assay methods (mouse serum neutralization), making the disease difficult to definitively diagnose. (Specimens recommended for bioassay include serum, liver tissue, GI tract contents, and feces.)

As little as 50 to 100 grams of hay contaminated by a decomposing carcass can be lethal to a horse. Shaker Foal syndrome is due to colonization of type B spores in the stomach. Toxicoinfectious botulism is also seen in adults, especially those with necrotic stomach ulcers and occasionally in those having a wound contamination.

Once the toxin is ingested or manufactured, it is absorbed into the circulation and carried to the neuromuscular junction. There it blocks the release of acetylcholine by attacking the ties between the neurotransmitter-filled vesicles and the neuronal membrane. A lag phase between toxin binding and clinical signs can be 12 hours to more than 60 hours depending on the toxin load. Self-immunization may occur with prolonged low dose exposure; therefore, naive horses (animals never having been exposed to botulinum organism) shipped to a state with a high incidence of botulism may be at greater risk than the native population.

CLINICAL SIGNS

Clinical signs of botulism can vary from peracute respiratory distress, paralysis and death to more slowly progressive muscular paralysis.

Clinical signs in foals occurring as early as two weeks of age and up to about eight months of age:

- Stilted gait progressing to generalized muscle tremors and collapse
- Drooling milk due to pharyngeal paralysis
- Reduced tail and eyelid tone
- Sluggish pupillary response
- Constipation and ileus
- Increased respiration and heart rate culminating in respiratory failure.

Symptoms are similar in adult horses and include:

- Reduced tongue and tail tone
- Muscular weakness including a stilted shuffling gait, toe dragging, drooping head and neck
- Trembling, especially of shoulder muscles
- Dull appearance due to absence of facial expression and drooling saliva
- Lingual and pharyngeal paralysis, inability to retract the tongue, quidding, or food in nostrils, muffling of vocal sounds
- Colic symptoms due to ileus
- Sluggish pupillary light response and mydriasis
- Inability to rise
- Inhalation pneumonia
- Dyspnea and respiratory failure.

Symptom progression is highly variable. It has been reported to take from one to seven days from the onset of signs to the inability to rise. Temperature and heart rate usually remain normal until acute respiratory distress occurs. The appetite is often normal but chewing is slow and swallowing is difficult. Horses may stare at and 'play' in water. Attempts may be made to drink but they are unable to do so. Foaming at mouth may be seen or the animal may have a fetid odor of mouth.



DIAGNOSIS

Definitive diagnosis requires finding toxin in the serum or spores in the GI tract. Detecting serum toxin is extremely difficult. Spores reportedly can be found in almost 80% of the feces of foals and in 20–40% of the feces of adult horses. Presumptive diagnosis may be made on finding the toxin or organism in feedstuffs and on the clinical signs observed. Differential diagnoses include equine protozoal myelitis, white muscle disease, rabies, tetanus, leukoencephalomalacia, and any other disease or toxin that affects the nervous system.

THERAPY

Neutralization of circulating toxin with antitoxin is the first objective. Once an animal exhibits progressive clinical symptoms of botulism it is very difficult to treat, since the toxin can not be displaced from the nerve endings. Mortality is often 70–90%.

Supportive treatment includes:

- Stall confinement to reduce physical activity
- Tube feeding (A high protein, low residue slurry of alfalfa meal, dextrose, cottage cheese and electrolytes has been used for up to a month to feed horses)
- Laxatives (Cathartic of choice is mineral oil)
- Pain medication / sedation
- Fluids
- Attention to animal's physical safety
- Other nursing procedures as needed.

Anti-microbials may be given for specific secondary complications. Avoid using drugs that potentiate neuromuscular blocking, such as aminoglycosides, procaine penicillin and tetracyclines. Oral antibiotics may encourage the overgrowth of the *Clostridium botulinum* organism.

Botulism antitoxin can bind circulating toxin hence preventing further transmitter disruption. Owners should be warned not to expect immediate results. The patient may appear to worsen after administration of antitoxin because the toxin that is already bound to motor end plates is not affected and symptoms continue.

IMMUNOPROPHYLAXIS

Botulism Type B toxoid is available from Neogen Corporation (800) 477-8201. Mares can be vaccinated near term to encourage passive transfer of botulism Type B antibodies to their offspring. Alternatively, foals may be vaccinated starting at two weeks of age. An animal who has not been vaccinated but who is at immediate risk due to either location or possible imminent exposure will benefit from administration of Clostridium Botulinum Antitoxin available from Plavacc USA Inc.

Plavacc USA Inc. has been granted a license by the USDA to produce EQUIPLAS® B (Clostridium Botulinum Type B Antitoxin, equine origin) for prophylactic administration to horses. When an individual is suspected of having contracted botulism, the source of infection is usually unknown. It is therefore advisable to prophylactically treat any horses that may have been exposed to the same food or water source as the affected animal. Antitoxin administered intravenously has the ability to immediately bind circulating toxin. If a horse has ingested the toxin, but is not yet manifesting clinical symptoms, prophylactic administration of antitoxin will prevent or greatly reduce signs of the disease.

Antitoxin should be administered to foals from botulism naive areas that are being shipped to areas of known risk for Shaker Foal Syndrome. Prophylaxis with the Clostridium Botulinum Type B Antitoxin is effective for a minimum of four weeks and up to 3 months.

EQUIPLAS B has been produced to the same high standards as Plavacc's other plasma products. It is available in an Adult Dose of 20,000 IU (200ml) and as a Foal Dose of 5,000 IU (50ml). USDA Licensed Product.

SUGGESTED READING:

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