PLASVACC FOR LIFE®

Equiplas® B

Licensed by the USDA and available in both foal and adult doses, Equiplas® B is antitoxin to Clostridium botulinum, Type B.

BOTULISM TYPE B

Botulism is a disease caused by the neurotoxins produced by *Clostridium botulinum*. Direct ingestion of preformed toxin in food (hay, grain, water or silage); ingestion or inhalation of spores (toxicoinfectious form); or in rare cases, clostridial wound infections can cause botulism. Basically, botulism causes flaccid paralysis affecting the peripheral nervous system.

Causative Organism

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

Disease

Type B is the most commonly found spore in the soils of Kentucky and the Eastern Seaboard, while Type A 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.

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 systemically formed, it is absorbed into 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
- Dypsnea 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 the mouth may be seen, or the animal may have a fetid odor of the 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, West Nile Virus, white muscle disease, rabies, tetanus, leukoencephalomalacia, and any other disease or toxin that affects the nervous system.

TREATMENT

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 cannot 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 (used for up to a month to feed horses)
- Laxatives (cathartic of choice: 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 has an ability to bind circulating toxin, 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

Plasvacc USA Inc. is the manufacturer of USDA licensed 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 native 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 Plasvacc's other equine plasma products. It is available in an adult dose of 20,000 IU and as a foal dose of 5,000 IU.

IMPORTANT: Before starting any treatment, please consult with your veterinarian.

PURITY & SAFETY

Our plasma is never blended. We use single-source donor tracing and cell-free plasma technology to reduce the risk of adverse reactions. Each donor animal is carefully screened, meticulously cared for, and quarantined. Our high gamma globulin, hyperimmunized plasma helps patients heal more quickly, with less drug intervention. All plasma is frozen immediately after collection and stored at –17°C to -40°C. Safety and sterility tests are performed on every serial to check for bacteria and fungi.

ORDERING & SHIPPING

Contact your local veterinary distributor to order. Plasma is shipped frozen, via overnight delivery service, and packed in insulated boxes.

PLASMA STORAGE

Store frozen up to 3 years at –17°C to -40°C until required. Re-freezing plasma that has been warmed to body temperature is not recommended.

SUGGESTED READING

Ricketts, S.W., et.al., 1994.
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Toxicoinfectious botulism in foal and adult horses. J . Am.Vet.Med.Assoc. 176: 217–220.

Whitlock, R.H. and Buckley, C., 1997.
Botulism. Vet. Cl. of No. Amer. - Equine Practice. 13(1):107–128.

Wichtel, J., 1991.
Botulism associated with feeding alfalfa hay to horses. J.AM.Vet.Med. Assoc. 199(4):471–472

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