Four Horses Down In A Matter Of Days

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The case described is loosely based on a case submitted to UVDL. Details have been changed and added or omitted by the author.

A horse owner recently lost all four of his horses. Three died and one was euthanized. Clinical signs in these horses as described by the owner included rapid recumbency in at least two of the animals, slow eating, quivering, weakness, tongue hanging out, teeth grinding and tremors. The horses that were recumbent did not have the strength to raise their heads. The first horse became sick on March 4, the second on March 8. The third animal was euthanized and submitted to the UVDL, Spanish Fork for necropsy on March 10. The last horse died on March 17.

No gross lesions were seen in the horse necropsied. A complete set of tissues was taken for histopathology (including the brain and spinal cord, to rule out other neurologic diseases). There were no significant histologic lesions in the horse necropsied including in the brain and entire spinal cord. The only change in management was feeding a new batch of alfalfa cubes to the horses starting on February 22. No new animals had come to the farm recently and none of the horses had moved off the farm for any reason in the past several months. Vaccinations for tetanus and West Nile virus were up to date.

Case Summary

Diagnosis: Botulism

Colon content and a sample of the hay cubes were submitted to the Botulism Diagnostic Laboratory at the University of Pennsylvania for testing. The lab no longer does the mouse bio-assay test (the classic test for botulism, it is still performed at National Veterinary Services Laboratory and at the California Animal Health and Food Safety Lab System, San Bernardino Lab). A real-time PCR assay for the toxin gene has been developed and validated and offered commercially by the University of Pennsylvania. The horse GI contents were positive for Type C botulism by real-time PCR (and negative for types A and B) and the hay cubes were negative for Types A, B, and C by real-time PCR. From the Botulism Lab website at the University of Pennsylvania: “Our laboratory tests all diagnostic samples using a real-time PCR assay for the detection of Clostridium botulinum types A, B and C toxin genes in vegetative cells after a culture enrichment step. Finding C. botulinum organisms or spores in a fecal or GI sample of a horse or cow, or in a feed sample, is strongly supportive of a diagnosis of botulism in animals exhibiting compatible clinical signs… samples from normal animals are rarely positive.”

In conclusion, the diagnosis was botulism and it is likely that the other three horses also had botulism. Type C botulism is less common than type B in horses in the US and has been associated with alfalfa hay cubes. We do not have confirmation that the alfalfa cubes that the horses were eating were the source of the toxin, but it is the suspected source. There is a report in JAVMA in 1991 (Kinde H, et al., Vol 199, No.6, September) of Clostridium botulinum type-C intoxication in horses in California associated with consumption of processed alfalfa hay cubes in horses. Pieces of animal body parts were found in the cubes and type C botulinum toxin was detected in the cubes and in horses (liver or GI content). In the cases described here, botulism was suspected and the hay cubes were no longer fed to the horses as of March 7, but the damage had already been done.

This recent case of botulism diagnosed in a horse highlights some of the important features of the disease in horses. In general, botulism is caused by a potent

neurotoxin that is produced by the anaerobic Gram-positive spore-forming bacterium Clostridium botulinum. It affects a wide variety of animals and birds worldwide. There are nine types of C. botulinum based on toxins produced. Types C and D cause most outbreaks in domestic animals in the United States, but not in horses. Of course, botulism affects human beings too, and is the reason that infants under 1 year of age should not be fed honey. The neurotoxins produced by this bacterium are the most potent biological toxins known. The toxins prevent release of acetylcholine at neuromuscular junctions resulting in flaccid paralysis. Usually, the toxin is ingested preformed (e.g. feed contaminated with rotting carcasses, spoiled food, etc).

Horses are highly susceptible to botulism, only a tiny amount of toxin is enough to cause severe disease. Approximately 85% of cases of equine botulism in the U.S. is type B botulism. Type B is endemic in mid-Atlantic states and Kentucky. Type A occurs more commonly in the western US. The case described here was type C botulism. Most adult horses acquire botulism by ingestion of preformed toxin. Foals with Shaker Foal Syndrome ingest the bacterial spores which germinate in the intestine and then release toxin (toxicoinfectious botulism). There is a third way that horses may acquire botulism: wound contamination with C. botulinum bacteria (less common). Type C botulism in horses has been associated with ingestion of forage contaminated with carcasses of dead animals.

Typical clinical signs appear a few hours to several days after exposure to toxin. Affected horses become weak with decreased tail tone and tongue tone (will not withdraw tongue into mouth easily), drop food, have difficulty drinking, dilated pupils, drooping eyelids, and usually progress to recumbency and death due to respiratory failure. Recumbent horses are often alert and continue to want food and drink. Sometimes, treatment with type specific antitoxin (very expensive) and supportive care may help horses recover if they are not yet recumbent. Remember that toxin binding is irreversible and the antitoxin only neutralizes unbound toxin. Antitoxin for horses is available as a monovalent antitoxin against type B and a polyvalent antitoxin against types A to E. The only vaccine licensed for use in horses is a type B botulinum toxoid. The use of type C toxoid that is licensed for mink is not recommended for use in horses.

Unfortunately, there is no equine vaccine against type C botulism available. Treatment is expensive and usually only effective in horses that are still standing: in this case, the polyvalent antitoxin would have had to be administered rapidly because the horses’ clinical signs rapidly progressed to recumbency.

References

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