

Botulism in Cattle



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Abstract

Botulism is an important disease of all mammalian, avian, and fish species that may lead to death. The disease is caused by the neurotoxins produced by *Clostridium botulinum*. There are several clinical findings of the disease and the treatment chance is low, thus vaccination measures should be taken. The purpose of this mini review is to present information related to etiology, pathogenesis, clinical findings, and diagnosis of botulism in cattle.

Keywords: Botulism, Cattle, Intoxication, Paralysis

Introduction

Botulism is caused by neurotoxins of a gram-positive, spore-forming, anaerobic bacteria called *Clostridium botulinum*. Botulism is an important disease that affects all mammalian and avian species as well as fish [1,2]. The disease emerges as a progressive, flaccid paralysis that affects the whole body [3].

Etiology

C. botulinum is an anaerobic, gram-positive, spore-forming, rod-shaped bacteria that can produce neurotoxins. Produced spores are resistant to heat. Eight types of botulinum toxins were identified and classified as A, B, C₁, C₂, D, E, F, and G, according to their antigenic specificity [1,3,4]. Type A, B, E, and F toxins lead to botulism in humans, while type C and D cause botulism in animals. Particularly wild animals, avian species, cattle, horses, and some fish species can be significantly affected by the disease [4]. Type C and D rarely type B causes intoxication in cattle [2,5].

C. botulinum is widely found in the soil. Anaerobic conditions, alkaline and neutral pH, particularly rotting animal carcasses provide the best median for the growth of bacteria and toxin production [1,6]. Bacteria can also produce toxins on rotten vegetables and feed that are contaminated by rotten substances [1,3,6]. Inadequate and non-balanced feeding, poor phosphorus uptake with the feed may lead to botulism epizootics. In animals with phosphorus deficiency, allotriophagia and tendency to eat animal carcasses, and osteophagia in animals feed with protein-

deficient feed may be observed. These animals may eat rotten carcasses which leads to the disease [1,2].

Pathogenesis

Intoxication occurs by several routes. The toxins can be orally ingested and the production of bacteria on anaerobic wounds may also lead to intoxication. When bacteria rapidly grows and produces toxins in the gut, the toxins may enter the bloodstream and spread by blood circulation [3,6]. Botulinum toxins can lead to paralysis except for type C₂. These toxins are ingested orally and resorbed in the intestines. Following the spread by the blood circulation to cholinergic neurons, these neurotoxins irreversibly bind to presynaptic regions of neuromuscular joints and lead to inhibition of acetylcholine secretion at the presynaptic and cholinergic neurons. As a result, impulses are blocked between efferent neurons and muscles, which causes paralysis on neuromuscular conjunctions, parasympathetic end-plates, cholinergic ganglia of the sympathetic nervous system, and adrenal glands [1,2,3].

Clinical Findings

Botulism is a sporadic disease; however, it can also endemically occur in grazing animals that tend to eat rotten carcasses. Disease progression is acute to subacute and the incubation period is 1-6 days [2,4]. There are several clinical findings reported in cattle with botulism. These are stiff gait, lateral recumbency,

hyporeflexia, bradycardia, abdominal type respiration, reduction in tongue strength, decrease in muscle tone in tail muscles and rumen, loss of pupillary and anal reflexes, mydriasis, ataxia, paralysis progressing from the hind part of the body to the front, hypersalivation, difficulty in swallowing, constipation and having dry-hard feces [1,2,5,6]. Death occurs due to respiratory paralysis [4,6].

Diagnosis

The disease is diagnosed according to anamnesis and clinical findings and should be differentiated from listeriosis, the paralytic form of rabies, tick paralysis, hypocalcemia, hypomagnesemia, mycotoxin toxicosis, lead poisoning, nitrate poisoning, and organophosphate poisoning. For definitive diagnosis, it is required to detect neurotoxins in serum, tissues, and gastrointestinal contents [1,4,6]. If suspected of botulism, rumen, and intestinal content, as well as feed samples, should be analyzed. These samples can be inoculated to mice for definitive diagnosis. To determine toxin type, specific botulinum antitoxin, and sample extracts are intraperitoneally injected and neutralization tests are applied [1]. In cattle, toxin concentration is generally low for analysis, and toxins may be resorbed or exposed to bacterial destruction which can avoid detecting toxins [2,4].

Treatment

Recovery is difficult [1]. Bivalent toxoid vaccines contain type C and D cultures are available to use in cattle for prophylaxis.

Vaccination can be performed twice every six months. Pregnant cows can also be vaccinated [7].

References

1. Catik S, Akgul G, Mecitoglu Z, Senturk S (2013) Botulism in a dairy herd. *Uludag Univ J Fac Vet Med*,32(2):53-56.
2. Duru SY, Sahal M, Beskaya A, Gazyagci S (2016) Investigation of botulism cases in a cattle herd in Bolu province. *Ataturk Univ J Vet Sci* 11(3): 332-338.
3. Galey FD, Terra R, Walker R, Adaska J, Etchebarne MA, et al. (2000) Type C botulism in dairy cattle from feed contaminated with a dead cat. *J Vet Diagn Invest* 12(3): 204-209.
4. Rodloff AC, Krüger M (2012) Chronic Clostridium botulinum infections in farmers. *Anaerobe*, 18(2) :226-228.
5. Aytakin I (2020) Evaluation of serum total sialic acid levels and some biochemical parameters in cows with botulism. *Ataturk Univ J Vet Sci*, 15(2):151-155.
6. Kelch WJ, Kerr LA, Ringle JK, Rohrbach BW, Whitlock RH (2000) Fatal Clostridium botulinum toxicosis in eleven Holstein cattle feed round blade barley haylage. *J Vet Diagn Invest* 12(5): 453-455.
7. Altug N, Ozdemir R, Cantekin Z (2013) Preventive medicine in ruminants: I. Vaccination program. *J F c Vet Med Univ Erciyes*, 10(1): 33-44.



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