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Botulism is a form of food poisoning, which occurs in man, animals, and birds as a result of ingesting *Clostridium botulinum* toxins in contaminated food, water, mud, animal carcasses, and bones. *C. botulinum* was discovered in 1897 in salted ham that was involved in the death of three people in Belgium (Pal et al., 2014).

11.1 Etiology

Clostridium botulinum is a gram-positive, motile, catalase-negative, strictly anaerobic, spore-forming, rod-shaped bacterium found in the soil and marine sediments throughout the world. It may contaminate vegetations, and colonize the gastrointestinal tract of fish, birds, and mammals. Furthermore, carrion carcasses and decayed organic matter provide a moist, warm, and low-oxygen environment for botulinum spores to germinate and produce toxins.

C. botulinum encompasses a diverse group of bacteria which were initially classified on the basis of their ability to produce botulinum toxin and are currently classified into four distinct phenotypic groups I–IV; these groups, along with some *C. butyricum* and *C. baratii* strains, are capable of producing some types of botulinum toxins (Smith & Sugiyama, 1988).

While *C. botulinum* Group IV has not been shown to cause disease in man or animals, Groups I and II are responsible for most cases of human botulism whereas Group III mainly causes botulism in animals.

C. botulinum organisms produce at least eight, serologically distinct, neurotoxins (A–H). These toxins are produced by botulinum endospores under strict anaerobic conditions and are the most powerful toxins known to mankind. Following their ingestion by people or animals, they are absorbed from the intestinal tract and carried by the bloodstream to neuromuscular endings, eventually reaching the axon terminal and blocking excitatory synaptic transmission, thus resulting in severe flaccid paralysis and death (Halpern & Neale, 1995).

Botulism often occurs in areas where the soil, plants, and consequently animals, are deficient in phosphorus. In this situation, animals sometimes tend to consume bones or soil, or even cadavers, in an effort to compensate for the deficiency, thereby increasing their risk of being exposed to botulinum toxins.

Among farm animals, botulism is primarily found in cattle especially in South Africa and South America, although a few cases are sometimes met with in equines and small ruminants as well.

11.2 Modes of Transmission

Very little information is available on camel botulism. In 1975, Provost et al. (1975), described a devastating outbreak of botulism (Type C) in dromedary camels in Chad, presumably after drinking from a well contaminated with a cadaver, which was the source of toxin. The affected herd consisted of 150 camels of which 45 were dead and 40 were severely ill at the time of reporting.

More recently, Bushara and Musa (2012) reported botulism in livestock, particularly camels, from Northern Darfur State in Western Sudan. They investigated many carcasses from various animal species, as well as water sources, soil, and animal tissue samples from the affected areas. Cultural and biochemical methods were used to isolate and identify *Clostridium botulinum* (Type C) while botulinum neurotoxin was identified by mouse bioassay and typing of the isolates by PCR. These authors stated that inadequate feeding of the animals leads to ingestion of soil and they suggested vaccination and improvement of feeding to reduce the risk of botulism.

11.3 Clinical Picture and Pathology

Signs of botulism include muscular weakness and flaccid paralysis of the hind legs, which may extend to other parts of the body including muscles of the face, jaw, and tongue resulting in dysphagia and drooling. In the case of camels, Provost et al. (1975) stated that the affected animals exhibited difficulty in standing, hind quarter paresis, collapse, and rapid death apparently due to respiratory or cardiac failure. Also, during botulism outbreaks in Darfur, Sudan, the predominant clinical sign in camels and other livestock was paralysis of the hindquarters (Bushara & Musa, 2012). There are no specific post-mortem lesions in botulism. Sometimes, abnormal material, such as bones or sand, may be found in the animal's digestive system.

11.4 Diagnosis

Botulism is initially suspected on the basis of clinical signs and differentiation from other conditions causing motor paralysis, e.g., rabies, certain toxic plants, poisoning by organophosphorus compounds, Ca deficiency, etc. Laboratory methods are used to detect bacteria, spores, and toxins. Detection of botulism toxin in suspected

material is the most reliable test method. Samples should be taken from any potential source as soon as botulism is suspected. In peracute cases, the toxin may be detectable in the blood by mouse inoculation tests but usually is not detectable in the average field case in camels and other farm animals. The PCR can be used for typing the toxin. Other detection methods include ELISA, complement fixation test (CFT), and immunodiffusion.

11.5 Treatment and Prevention

There is no specific treatment for botulism; *C. botulinum* toxoid and Guanidine hydrochloride may be tried but the results are inconsistent.

The source of the toxin should be removed as soon as it is identified, and the carcasses of dead animals should be properly disposed of. Proper nutrition and salt licks should be provided, while P or any other deficiency in the diet should be corrected. Rotten silage and plants should be removed and the contamination of feed and water with bird or rodent carcasses or bird litter should also be prevented.

Vaccines (types C and D) developed for cattle in some countries may be considered for use in camels in endangered areas.

11.6 Notification

Botulism should be reported to relevant public health authorities as it can sometimes be foodborne.

References

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