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# *Tutorial Article*

## *Type B* botulinum intoxication in horses: case report and literature review

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## Introduction

Botulism in horses has been attributed to toxins in feeds, including brewers grain (malt), oat grain, grass clippings allowed to decay in a pile, water contaminated with carcasses, spoiled vegetables and potatoes, silage, hay and processed alfalfa hay cubes (Switzer *et al.* 1984; Colahan *et al.* 1991; Hailu *et al.* 1991; Radostits *et al.* 1994). **High moisture feed, protein rich material and anaerobic conditions increase the risk of an outbreak.**

The organisms produce potent exotoxins of protein nature. Seven types of toxin (*A, B, C, D, E, F* and *G*) have been recognised on the basis of immunological differences (Hariharan and Mitchell 1977; Radostits *et al.* 1994). Each type of toxin has a different geographic distribution (different types of soil) and species susceptibility (Hariharan and Mitchell 1977; Swerczek 1980; McKay and Berkhoff 1982; Divers *et al.* 1986; McIlroy and McCracken 1987).

*Type B* spores predominate in the eastern United States, Iceland, the UK and much of continental Europe (Dodds 1994). *Type B* strains are encountered most frequently in slightly acidic soil of high organic content (Smith 1984).

*Types C* and *D* are found more frequently in warmer environments (Dodds 1994). The bacteria are not always able to produce toxins where they are able to grow. Toxins in feed can be active for several months (Hariharan and Mitchell 1977) but are susceptible to pH changes and high temperatures (Switzer *et al.* 1984).

When ingested and absorbed, these toxins can disturb neuromuscular transmission by blocking the release of acetylcholine and causing flaccid paralysis. *Types B, C* and *D* have been reported to be responsible for equine botulism (Switzer *et al.* 1984; McIlroy and McCracken 1987).

Outbreaks of botulism are often catastrophic as horses seem to be quite sensitive and the death rates are high. Smith (1984) and Franzen *et al.* (1992) have suggested that horses are very sensitive to *type B* toxin.

In recent years, 3 routes of exposure have been recognised (Swerczek 1980; Hailu *et al.* 1991; Dodds 1994):

- Ingestion of preformed toxin is the best known and the most common cause of botulism.
- Colonisation and production of toxin in the intestines, as described for infant botulism.
- Growth and production of toxins in wounds, as described in toxicoinfectious botulism (Shaker foal syndrome).

Clinical signs usually appear 3–7 days after the animal gains access to the toxic material (Switzer *et al.* 1984). The severity of the disease and the period until first signs appear, depend on the dose ingested. Signs of botulism in horses are predominantly related to progressive muscular paralysis, particularly of the pharynx (dysphagia), tongue, jaw, lower lip and limbs (Swerczek 1980; Ricketts *et al.* 1984; Haagsma *et al.* 1990; Franzen and Gunnarsson 1992). Saliva may drool from the mouth. Food falls from the mouth or is observed at the nostrils. There may be muffled vocalisation (dysphonia) and respiratory stridor, the latter caused by dorsal displacement of the soft palate.

A dull sleepy appearance is frequently a prominent sign. The body temperature is usually normal. The appetite is often remarkably good. The discovery of swallowing difficulties may be delayed because the affected horse is often observed chewing. Muscle weakness and paralysis commonly start in the hindquarters and progress to the forequarters then the head and neck. Abnormal gaits are often observed such as shuffling, stilted gait and toe dragging (Ricketts *et al.* 1984; Radostits *et al.* 1994; Colahan *et al.* 1991). Hanging head and neck below the horizontal are common. Mydriasis and slow pupillary reflexes are frequently observed.

Intestinal hypomotility is common. Rectal examination may reveal dry mucus covered faeces. Sometimes colic, generalised sweating and muscle tremors are also observed (Ricketts *et al.* 1984).

Peracute cases show very few characteristic signs. Once mature horses with botulism become recumbent they seldom rise again. Breathing becomes laboured and paralysis of the costal and diaphragmatic musculature is the most common cause of collapse and death (Ricketts *et al.* 1984; Colahan *et al.* 1991).

Death rates have been reported in naturally occurring cases to be in the order of 69–90% (McKay and Berkhoff 1982; Colahan *et al.* 1991). Death may occur within hours or take several days. The neuromuscular blockade seems to be permanent but in cases of recovery, new junctions are created. Recovery may take weeks or months but is complete (Colahan *et al.* 1991). Muscular weakness and tremors are characteristic findings of the toxicoinfectious botulism in foals (Swerczek 1980).

**In the following 2 outbreaks of a serious disease in horses, *type B* botulism was suspected to be the cause.**

## Case histories

### *Outbreak 1*

On 3, 4 and 5 January 1994, 5 out of 7 Icelandic horses, held on a small pasture on the southeast coast of Iceland, became anorexic and weak. Four of the affected horses went into recumbency, followed by respiratory failure and coma. Death occurred 18–48 h after first signs were noticed. The fifth horse (the eldest one) became unsteady and stumbling but able to stand. Nevertheless, it was subjected to euthanasia on humane grounds 7 days after it showed the first symptoms. Two horses showed no obvious signs. Cyanosis was obvious at the terminal stage in 3 cases and the body temperature was normal in most cases.

### *Outbreak 2*

On 13 and 14 January a new **outbreak** occurred on a different pasture on the same farm. Eight of 14 horses showed signs less acute than those in the first **outbreak**. Three horses were subjected to euthanasia and the rest recovered. Six horses showed no obvious signs.

## Clinical signs

Clinical signs in **outbreak 2** and those of the subacute case in **outbreak 1** were quite similar. The most important findings were normal rectal temperature, dysphagia and a dull sleepy appearance. The horses tried to eat and drink. Saliva dribbled from the mouth and ingested food was sometimes seen in the nostrils. The horses were also observed on several occasions with their lips in drinking water and, in a few cases, paresis of the lips was seen. Keeping the head above water for long periods, without any attempt to drink, might have been an attempt to moisten the mouth and the throat.

The horses usually brightened up when stimulated, although they soon returned to their previous sleepy appearance when left alone. Signs involving the legs were rather mild and seriously affected horses were still able to get to their feet without much effort.

## Treatment

When the second outbreak started, **the sick horses and horses without signs** were given purgatives in an attempt to inhibit toxin absorption from the small intestine. All the sick horses in **outbreak 2** were treated with antibiotics (procaine penicillin) and fed with thin porridge. Some of them also received parenteral nutrition i.v.

## Epidemiology

The feed, rather than a contagious pathogen, was immediately suspected. The horses were held on pasture and ate silage directly from a big bale. The distance between *feeding place 1* and *feeding place 2* was approximately 2 km. Nothing abnormal could be found at these feeding places.

On 11th January, a dog was seen grasping the carcass of a bird, 10–15 m from *feeding place 1*. The carcass was very squashed, smelled of silage and was tentatively identified as belonging to an oyster catcher (sea pie). The horses associated with **outbreak 2** were also fed with big bale silage that had been prepared from the same meadow as in the first case. After the second outbreak the farmer stopped feeding horses with bales from this particular meadow but continued feeding sheep. These animals showed no signs of botulism.

The grass silage was harvested from a meadow located near a lake where gulls and other waterfowls were commonly observed on the meadow. The lake is connected to the ocean and tidal variations occur daily. The silage was prepared without preservatives. Nothing was left of the suspect bale and nothing abnormal was observed in other bales from that meadow (the dry content was probably higher than 25%).

### *Laboratory results*

The carcass of the bird was sent to the Institute for Experimental Pathology at Keldur, University of Iceland for further analysis. An extract from the carcass was analysed by the mouse bioassay for botulinum toxin. *Type B* toxin was demonstrated by mice inoculations, followed by neutralisation tests with antitoxin.

## Discussion

### *Diagnosis*

On the basis of the signs, negative *post mortem* findings and the demonstration of *type B* botulinum toxin in the carcass, the horses in **outbreak 1** probably suffered from botulism caused by spot contamination of toxin in a bale. On the basis of the signs and similarity with **outbreak 1**, **outbreak 2** was probably caused by *type B* toxin contamination of a big bale. There are several possibilities for the botulin bacteria to make toxin in silage. Birds may deposit their faeces containing *Clostridium botulinum* on the meadow which contaminates the grass cut for silage. Very often the source is eaten and cannot, therefore, be identified.

Dysphagia seemed to be a constant and prominent sign in our cases as described for other *type B* outbreaks in horses (Haagsma *et al.* 1990; Hailu *et al.* 1991; Franzen and Gunnarsson 1992). A dull, sleepy appearance was also a constant sign. In subacute cases, the lip wetting tendency was obvious. The signs of normal rectal temperature, dysphagia, dull sleepy appearance and the lip wetting might be pathognomonic for *type B* botulism in horses.

Dry mouth, tongue and throat is described in cases in man, most frequently in *type B cases* (Dodds 1994). In some outbreaks of *type C* botulism, throat function seems to have been quite normal but signs involving dysfunction of limbs were more prominent (Kelly *et al.* 1984; Hailu *et al.* 1991).

## *Differential diagnosis*

Listeriosis may present signs similar to botulism including dysphagia, salivation and depression. When muscular tremors and weakness are observed, hypocalcaemia or grass sickness should be considered.

Poisonings resulting from agents, such as lead, organochlorines, organophosphates and ragwort may present similar clinical pictures. Horses affected by leucoencephalomalacia (mycotoxins, mouldy corn grain) can show marked depression of consciousness, swallowing inability and weakness (Radostits *et al.* 1994). Some virus infections, for example, equine encephalomyelitis, may give rise to similar features and azoturic myopathy may cause signs involving the hindlimbs.

**The lack of exposure to other pathogens and specific histopathological and clinicopathological findings should lead to the consideration of a diagnosis of botulinum intoxication** (Hailu *et al.* 1991).

An accurate diagnosis is seldom achieved. It is established by the demonstration of botulinum toxin in serum, liver or other tissues.

Blood samples should be taken immediately when an animal shows clinical signs because toxin cannot usually be detected in horse serum at a late stage (Switzer *et al.* 1984). These samples should be frozen and submitted to a diagnostic laboratory for toxin detection. When horses are affected with toxico-infectious botulism, it is suspected that serum contains only minute amounts of toxin that cannot be detected by conventional methods (Swerczek 1980). The circulating blood dilutes the toxin far beyond the concentration necessary to kill adult mice.

Finding the bacteria (or spores) in the faeces or intestinal tract is of limited diagnostic value. However, finding toxins in the gastrointestinal tract, with or without spores, is regarded as a more reliable sign (Hailu *et al.* 1991). Toxins may be produced *post mortem*. The demonstration of toxins in suspected feed stuffs is of great importance but too seldom achieved because the suspected feed or any other cause of botulism has disappeared.

When examining feed, samples should be collected from different locations and preferably from moist areas. All samples should be refrigerated immediately or frozen and sent to the laboratory as soon as possible. Even with optimal handling of samples the demonstration of toxins may prove difficult.

## *Treatment*

Treatment is mainly based on intensive nursing care and supportive administration of fluids and nourishment (Colahan *et al.* 1991). Specific or polyvalent antitoxins may be used early in the disease. A reduction in death rate from 90 to 25% due to use of specific antitoxin in field outbreaks of botulism, has been reported (Colahan *et al.* 1991; Crane 1991). If antibiotics are used, those that may potentiate neuromuscular weakness, such as aminoglycosides, tetracyclines and procaine penicillin should be avoided (Divers *et al.* 1986; Colahan *et al.* 1991). Potassium penicillin is recommended, especially when toxico-infectious botulism is suspected.

Mineral oil should be given initially and thereafter as needed to avoid or treat constipation. Difficulties may be encountered passing the stomach tube in affected animals. If gastric emptying and intestinal motility are adequate, placement of an indwelling nasoesophageal tube ensures adequate hydration and nutrition. In an attempt to reduce the absorption of toxin from the small intestine, all horses exposed to the suspect feed should be treated with purgatives.

Vaccination with toxoid vaccine is sometimes recommended. This must be type specific or combined, because cross immunity does not occur between *types B* and *C*. All animals should be vaccinated including animals that have recovered from the disease (Hariharan and Mitchell 1977; Hailu *et al.* 1991). The reason is that the immunising dose is much higher than the lethal dose. The variation which occurs in geographical distribution of the various *types* is an important factor when considering prophylactic vaccination programmes (Radostits *et al.* 1994).

## *Silage as a possible epidemiological factor*

Ensilage is very often used as horse feed in Iceland and many other countries; the anaerobic conditions favour the growth of the botulinum bacteria. It should also be taken into consideration that endospores are more tolerant to heat, irradiation, oxygen and antibiotics. After packing, the $O_2$ content of the silage drops almost immediately and a fall in pH soon follows (Jonsson 1989). Within a dry matter range of 15–25% the corresponding pH is usually 3.7–4.6 units (Jonsson 1989; Kjus 1991; Randby 1992). A pH lower than 4.6 inhibits growth of *C. botulinum* (Notermans *et al.* 1979; Dodds 1994), but several factors influence the acid tolerance of the bacteria, including strain, substrate, temperature, the presence of preservatives and water activity ($a_W$). It has been shown that proteolytic strains of *C. botulinum type B* can produce toxins in grass silage (Notermans *et al.* 1979). Noncarrion-associated botulinum (forage poisoning) of animals is reported and *type B* has frequently been demonstrated in such outbreaks (Divers *et al.* 1986).

Wilting may inhibit growth of *C. botulinum* as the vegetative bacteria is quite sensitive to high osmotic pressure, but safe values are difficult to estimate (Jonsson 1989; Hailu *et al.* 1991). If the dry matter content of the silage is low (15% or below), the pH may fall in the beginning of the process but rise again as lactate-forming bacteria lose their dominance. Such conditions reduce the feed quality and greatly increase the possibilities of growth and toxin production (Notermans *et al.* 1979; Jonsson 1989; Dodds 1994).

When used correctly, silage preservatives have been shown to improve feed quality. Silage preservatives inhibit the growth of *C. botulinum* by lowering pH and/or inhibition by nitrite (Dodds 1994). The microbiological environment in a carcass or manure is, however, different

from the rest of the silage and when such contamination occurs, preservatives have limited ability to protect against botulism. It has also been shown that the effect of low pH and low $a_w$ in limiting toxin production is less in a meat than in a grass medium (Notermans *et al.* 1979).

Prevention of toxin production in silage may, therefore, prove difficult. Further research should be performed on the growth of *C. botulinum* and toxin production in carcasses in silage prepared with or without preservatives. Preservatives containing nitrites are of special interest.

## *Botulism in Iceland*

To the author's knowledge, treatment of horses with botulinum antitoxins has not yet occurred in Iceland. Since 1995 all horses on one farm in South Iceland (where botulism was suspected in 1993 and 1994) are vaccinated with *type B* specific toxoid vaccine (Gunnarsson 1997). Botulism seems to be a greater problem for horses than cattle or other ruminants in Iceland.

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