Descriptive study of botulism in an Austrian dairy herd: a case report

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ABSTRACT: An outbreak of botulism was suspected at an Austrian dairy farm in June 2010. Six Simmental cows, out of a herd of 29, were affected and showed the typical signs. The affected cows included either animals suffering from sudden recumbency and reduced tongue tone or others which developed paresis, which gradually led to recumbency. Most of the affected animals died. Two cases were submitted to the Clinic for Ruminants at the University of Veterinary Medicine Vienna in order to clarify the cause of illness. The animals had reportedly been fed recently with silage possibly polluted by discarded cat carcasses spread onto the pasture used for the silage in question. The two referred cases both showed recumbency and swallowing difficulties. Both cows had to be euthanized. One of the two cows was in the late stage of pregnancy and a healthy calf could be delivered by Caesarean section. The outbreak of botulism was diagnosed clinically as well as by confirmation of *Clostridium botulinum* neurotoxins C and D by mouse bioassay. To the authors' knowledge, this is the first report in which a live calf has been delivered out of a cow suffering from acute botulism.

Keywords: botulism; herd case; flaccid tongue; recumbency; mouse bioassay

Botulism is caused by *Clostridium botulinum* neurotoxins (BoNT). *Clostridium botulinum* (*C. botulinum*) is a gram-positive anaerobic rod that forms spores under aerobic conditions. Its spores can survive for almost 30 years and germinate under warm and humid conditions. *C. botulinum* pathogens occur worldwide and are associated with soil, decaying and rotting vegetative material or cadavers. *C. botulinum* produces eight different types of neurotoxins: A, B, C_{α}, C_{β}, D, E, F and G (Galey et al. 2000; Bohnel and Gessler 2010). Botulism in cattle is most often caused by BoNT B, C and D. BoNT C and D are mostly associated with the ingestion of carcasses and broiler litter (Dirksen et al. 2006; Radostits et al. 2006). BoNT B is mostly connected with rotten vegetation or feedstuff (Rings 2004; Hogg et al. 2008).

The latent period ranges from 24 hours to seven days and depends on the amount of ingested toxins (Radostits et al., 2006). Infection can occur by way of three routes: wound contamination, ingestion of the preformed toxin and by the proliferation of the neurotoxins within the intestinal tract (toxicoinfectious botulism). The toxins are distributed via the blood stream, travel retrograde along the nerve cells to the neuromuscular junction and interfere with the release of acetylcholine. This results in characteristic progressive flaccid paralysis that commences with a stiff gait, mostly starting in the hind limbs and advancing further cranially. Further clinical signs are anorexia, weakness, reduced ruminal contractions, difficulties in rising or cattle already in recumbency with heads tucked in their flanks. Faeces consistency varies from dry and hard to diarrhoea. Affected cows have difficulties swallowing, show salivation and their tongues protrude from their mouths, which results in decreased feed intake. The prognosis depends on the clinical stage of the disease. Recovery has been reported in patients with mild clinical signs. In acute cases, death is caused by respiratory paralysis. In cattle, the disease is caused most often by the ingestion of the preformed BoNT. Phosphorus deficiency may result in pica. Cattle with pica tend to chew on cadavers and bones in order to balance

their mineral deficiency which means a high risk of BoNT ingestion (Braun et al. 2005; Dirksen et al. 2006; Radostits et al. 2006). Toxicoinfectious botulism is well documented in human infants (Arnon and Chin 1979; Bartram and Singer 2004), in equine shaker foal syndrome (Radostits et al. 2006), and has been suspected in foals suffering from equine grass sickness (Wylie and Proudman 2009). In bovines, the clinical appearance is different from other species (Bohnel et al. 2001). The symptoms of toxicoinfectious botulism in cattle are seen in the periparturient time and consist of indigestion, non-infectious acute laminitis, engorged veins with a positive venous pulse, oedema in the legs, udder, dewlap, hard abdomen, forced respiration and unexpected death (Bohnel et al. 2001). These symptoms are typical for the chronic form of toxicoinfectious botulism and are specified as a chronic "visceral form of botulism" (Bohnel et al. 2001). A final diagnosis of botulism is elusive, as toxin levels might be too low to detect, or all toxins might be bound at the neuromuscular junctions. In addition, the source may have already passed through the ingestion and been excreted or the diagnostic methods, such as the mouse bioassay, might not be sensitive enough (Hogg et al. 1990; Galey et al. 2000; Rings 2004; Sharpe et al. 2008). The present paper describes an outbreak of botulism in an Austrian dairy herd. Furthermore, this is the first description of a healthy calf delivered from a cow suffering from acute botulism.

CASE DESCRIPTION

Preliminary report

In June 2010, a local veterinarian in Lower Austria reported a sudden breakdown of several cows in a dairy herd of 29 Simmental cows. Over the course of six days, a total of seven out of twenty-nine cows were affected and showed clinical symptoms. One of these cows showed only mild diarrhoea, while all of the other affected animals could be divided into two groups regarding their clinical signs: one group with sudden recumbency and tongues with reduced retraction strength. These animals died suddenly one day after the first symptoms occurred. The symptoms in the second group developed more slowly starting off with tripping gait and recumbency. Death occurred in these cows after two to three days. On 18 June 2010 the first cow from the dairy herd showed sudden recumbency. The farmer then called the local veterinarian. As the symptoms are typical of hypocalcaemia, treatment with calcium and glucose infusions were administered. For a short period the animal improved slightly. The next day the cow was recumbent again and anorexia was noted. On 20 June 2010 two more cows were affected and the first cow, which was affected (herd case 1), died. The herd case 2 started off with a tripping gait and increased lying time. The treatment carried out by the local vet included B vitamins and antibiotics. The third herd case showed sudden recumbency, anorexia, difficulties in swallowing, and a tongue with reduced retraction strength. This animal died a few hours after the first clinical signs occurred. Two days later, herd case 4 exhibited tripping gait, recumbency, increased unease, cool acra and reduced, but dark, shiny and pasty-like faeces. Two more cows fell ill on 21 June 2010. Herd case 5 had mild diarrhoea but was in good general condition whereas herd case 6 revealed acute signs. These were similar to the symptoms in herd case 3. Herd case 6 died a few hours after the first symptoms occurred. The last herd case occurred on 23 June. Herd case 7 showed mild clinical signs with tripping gait and increased lying time but no problems in swallowing. Herd cases five and seven were the only that survived.

Herd case 2 (hereinafter referred as "case 1") and herd case 4 (hereinafter referred as "case 2") were referred to the Clinic for Ruminants at the University for Veterinary Medicine, Vienna.

Upon further request, the farmer reported that the autumn before he had discarded cat carcasses into the manure and spread it onto the pasture that was used to ensile. The silage in question was produced three weeks before the first herd case was noted. The first clinical symptoms occurred in the herd three days after the first feeding of the silage in question. In addition, the farmer reported that he had found a dead cat in the hay recently.

Clinical study

Thorough clinical and neurological examinations were performed in the two referred cases according the criteria described by Baumgartner (2009).

Laboratory investigations

Samples from herd case one, three and six were taken by the local veterinarian during the out-

break to be tested with mouse inoculation testing for botulism by the Austrian Agency for Health and Food Safety (AGES), Institute for Veterinary Disease Control, Graz, Austria.

At the clinic, blood samples from the jugular vein from case 2 were collected for a complete blood count. As botulism was suspected the blood, faeces, and ruminal content from case 1 were tested for *C. botulinum*, BoNTs, and specific antibodies by mouse inoculation testing and ELISA (type A, B, C and D). The mouse inoculation test and the ELISA were performed by Miprolab, Gottingen, Germany. All results are listed in Table 1.

Medical treatment

Sterile saline solution (NaCl 0.9%, Fresenius Kabi, Bad Homburg, Germany) and 40% Glucose (Glucosteril 40%, Fresenius Kabi, Bad Homburg, Germany) were used for intravenous infusion. For alleviation of pain, Ketoprofen (Romefen 10%, Merial, Lyon, France) was administered in a dosage of 3 mg/kg bodyweight. Ampicillin (Ampivet Suspension 10%, Virbac S.A., Carros, France) was used for antibiotic treatment in a dosage of 10 mg/kg body weight.

Post mortem examinations

Necropsies of case 1 and 2 were carried out at the Institute for Pathology and Forensic Veterinary Medicine at the University of Veterinary Medicine, Vienna.

Gut samples from the small intestines with intestinal contents were collected for further bacteriological investigations from case 2.

RESULTS

Clinical study: Case 1, 2

Case 1 was in sternal recumbency when she arrived at the clinic.

The initial examination showed a poor general condition, a rectal temperature of 38.2 °C, a respiratory rate of 16 breaths/min, a heart rate of 72 beats/min, a weak, irregular pulse rate in addition to weak and poorly filled venous vessels, anaemic oral and nasal mucous membranes and reddened conjunctival mucous membranes. The cow salivated and the tongue protruded from her mouth (Figure 1).

A haematological examination revealed mild erythrocytosis (7.46×10^6 µl), a slightly increased mean corpuscular haemoglobin concentration (34.8 g/dl), mildly increased concentration of segment neutrophils (49.6%), decreased concentration of lymphocytes (38.4%), and increased concentration of monocytes (5.8%). Hepatic enzymes showed an increased value of the aspartat-aminotransferase (AST 173 IU/l) but normal levels of glutamate dehydrogenase (GLDH 9.39 IU/l) and gamma glutamyl transferase (GGT 19.00 IU/l). Calcium, phosphorus, and magnesium were within the physiological ranges (Ca 2.44 mmol/l; P 2.12 mmol/l; Mg 0.84 mmol/l). The animal was treated by an intravenous infusion of a 0.9% saline solution, 40% glucose solution (1 l 40% glucose per 10 l 0.9% NaCl solution) and thiamine (300 mg per 10 l 0.9% NaCl solution) at an infusion rate of 30 ml/kg/day. Ampicillin and ketoprofen were administered, as listeriosis was also a differential diagnosis. Despite the intensive treatment, the cow did not improve over the next days and remained recumbent and

Table 1. Results of microbial investigations

	Mouse bioassay			ELISA	Culture
Agent (Laboratory)	blood	faeces	ruminal content/intestine	blood	intestine
Herd case 1(AGES)	negative	n.t	n.t.	n.t	negative
Herd case 2 (AGES)	negative	n.t	n.t	n.t	negative
Case 1 = herd case 2 (Miprolab)		BoNT C/D	n.t	BoNT A/B	negative
Herd case 3 (AGES)	negative	n.t	negative	n.t	negative
Herd case 4 (AGES)	negative	negative	n.t	n.t	negative
Case 2 = herd case 4 (University)	n.t	n.t	n.t	n.t	*C. perfingens* (A)
Herd case 5 (AGES)	negative	negative	n.t	n.t	negative
Herd case 6 (AGES)	n.t	n.t	n.t	n.t	negative

n.t. = not tested

Figure 1. Case 1 showing paralysis of the tongue

completely anorectic. She was also totally unable to swallow and here tongue protruded out of her mouth. Due to welfare reasons, euthanasia was performed.

Botulism was suspected due to the clinical symptoms and the preliminary report.

The second cow ("case 2") which was referred to the clinic was pregnant six days prior to its calculated calving date. The clinical signs were similar to the first case. The medical treatment was the same as in case 1.

Despite the fluid therapy the animal became more distressed, started to breathe more frequently, and began to tuck its head into its flank. Due to the worsening condition, a Caesarean section was performed with the owner's permission and a live female calf was delivered. The cow was euthanized due to animal welfare reasons. The calf was released home in good general condition and was growing normally.

Post mortem findings

Case 1. The post mortem examination revealed a mild to moderate hyperaemia of parenchymatous organs, a severe alveolar pulmonary oedema with moderate emphysema in the cranial parts of the lung and small abomasal ulcers. The intestines were within the physiological ranges.

No macroscopic or histological pathological changes were found in the brain.

Case 2. In the second cow, dark bloody gut content with small blood coagula was found in the middle part of the jejunum during necropsy. Focal petechial haemorrhages were noted at the caecal mucous membrane. Eosinophilic inflammation was obvious at the serous liver membrane. Hyperaemia was seen in all parenchymal organs. All claws revealed signs of chronical laminitis. The brain revealed no sign of pathological changes.

Gut samples were taken for microbiological examination (Table 1) at the Institute for Bacteriology, Mycology and Hygiene at the University of Veterinary Medicine, Vienna.

Proof of *C. botulinum*, BoNT and antibodies

Samples from blood, ruminal content and faeces were taken from case 1 (herd case two) and tested by mouse inoculation testing and ELISA for BoNT A, B, C and D.

In faeces, BoNT C/D were found by mouse bioassay, and blood antibodies against BoNT A and B were verified by ELISA (Table 1).

DISCUSSION AND CONCLUSIONS

Botulism in cattle has seldom been described in Austria. The last case was reported by Glawischnig et al. (1999). The authors reported similar symptoms of a *C. botulinum* C2 outbreak with clinical signs such as weakness, stiff gait, recumbency, and decreased retraction strengths of the tongues. Furthermore, flaccid tail paralysis was described, which could not be observed in the presented case. In the beginning, the presumed diagnosis of botulism was made due to the clinical signs, epidemiology of the outbreak, non-specific haematology results and the *post mortem* findings. The clinical signs, including sudden or delayed recumbency, flaccid paralysis of the tongues, and high mortality were similar to those described by several authors (Galey et al. 2000; Cobb et al. 2002; Braun et al. 2005, 2006; Dirksen et al. 2006; Radostits et al. 2006; Sharpe et al. 2008) for carrion-associated botulism caused by BoNT C and D. Botulism caused by animal carcasses being ensiled and found in hay has been described before (Galey et al. 2000; Dirksen et al. 2006; Radostits et al. 2006) and was similar to our preliminary report. Toxins may persist in carrion for at least one year and contamination of feed may cause multiple cases of botulism (Radostits et al. 2006). Radostits et al. (2006) reported that clinical signs usually appear 3–17 days after infection and

the affected animals survive for 1–4 days. According to these findings, the outbreak started three days after the first feeding of the silage and the affected animals survived for 1–3 days in the presented case. Clinical cases developed signs within six days, which may indicate that the animals were exposed to the BoNT for a short time, e.g., a few days (Trueman et al., 1992). The short survival periods of 1–3 days could be caused by exposure to a rather higher dose of the BoNTs. The large quantities of food that a high yielding cow ingests, or alternatively, the heightened susceptibility of certain cows to BoNTs compared to other animals in the same herd, could be further explanations. The reasons for greater sensitivity might lie in differing individual immunity due to stress, lactation period, gravidity, or other reasons. In addition, the BoNT were possibly not equally cultivated in the possible source and thus distributed unequally. The farm had no technical mixing system and the feeding components were mixed manually and distributed in single components as the animals were kept in a tie-stall barn. Herd case 5, which survived the outbreak, showed diarrhoea. Diarrhoea has been described in connection with toxicosis due to BoNT B (Dirksen et al. 2006; Radostits et al. 2006).

The two referred cases showed the typical clinical signs as described before. As they had to be euthanized on welfare grounds they were further referred for necropsy.

The main *post mortem* findings in both cases were hyperaemia of the parenchymatous organs. Both animals showed a moderate decreased general condition and circulatory disorders. The hyperaemia might have been due to this or might have resulted from the euthanasia. In case 1, a severe alveolar pulmonary oedema with moderate emphysema in the cranial parts of the lungs and small abomasal ulcers were present. Pulmonary oedemas are very often caused by euthanasia and abomasal ulcers are frequently found in stressed animals (Ok et al. 2001; Dirksen et al. 2006). The gross abnormal findings in case 2 were the bloody gut content with small blood coagula in the middle part of the jejunum and petechial haemorrhages at the caecal mucous membrane. Similar findings were recorded by Cobb et al. (2002) and could be due to clostridial enterotoxaemia caused by *Clostridium perfringens* (*C. perfringens*) type A, which could be demonstrated in bacterial culture. Clostridial enterotoxaemia caused by *C. perfringens* type A has been described (Weiss and Popischil 2007; Lebrun et al. 2010). No specific pathological findings were found at the necropsies, which were in accordance with previously published reports (Dirksen et al. 2006; Radostits et al. 2006; Dahme et al. 2007). Macroscopic and histological examination of the brains revealed no pathological abnormalities (Dirksen et al. 2006; Radostits et al. 2006; Dahme et al. 2007). The differential diagnoses for botulism include mineral imbalances (hypocalcaemia, hypophosphatemia), spinal meningitis, cerebrocortical necrosis, abscess of the brain base, listerial encephalitis, tick paralysis, paralytic rabies, poisoning by *Phalaris*, organophosphates, or carbamates (Dirksen et al. 2006; Radostits et al. 2006)). The mineral imbalances could be ruled out by the haematology results. Other diseases such as listerial encephalitis, tick paralysis, paralytic rabies and poisoning by *Phalaris*, could be eliminated due to its unlikely occurrence and the characteristic signs. Moreover, spinal meningitis, cerebrocortical necrosis, abscess of the brain base, and listerial encephalitis were rather unlikely due to the fact that clinical signs occur over a prolonged period of time with slightly different symptoms and mostly affecting individual cows. Organophosphates or carbamates intoxication are associated with miosis, diarrhoea, tonic-clonic convulsive seizures, and bradycardia (Dirksen et al. 2006), which were not seen in the concerned animals. The haematological examination revealed no specific changes, which has been described for botulism (Dirksen et al. 2006; Radostits et al. 2006; Weiss and Popischil 2007). The leucocytosis may be physiologically induced by stress or pathologically in combination with infectious diseases or in the context of endogenous intoxications (e.g., intestinal toxicosis) (Kraft and Durr 2005). Case 1 showed increased AST values but GLDH and GGT values were in range. AST is mainly found in liver and muscles and, therefore, compared to GLDH and GGT, is not a liver-specific enzyme. The value might have been increased in the context of the recumbency. To our knowledge, this is the first time that a live calf has been delivered by a cow suffering from acute botulism. The calf was in good condition and showed a physiological development. This is interesting, as Bohnel et al. (Bohnel and Gessler, 2005) mentioned fertility problems in connection with visceral botulism. Stillbirth and abortion of calves have also been described. BoNTs are described to reduce the blood supply and muscular action of the uterus (Morris et al. 2001). The calf was probably not affected by possible uterine undersupply due to the short time

of two days in which its dam showed clinical signs. Furthermore, the calf was almost fully developed, which made it more resilient. The clinical findings of case 1 together with the report of cat carcasses pointed to suspected carrion-associated botulism.

The ELISA testing showed further antibodies against BoNT A and B in the blood of case 1. *Clostridia* are part of the physiological intestinal flora in humans and animals and can often be found in faeces (Selbitz 2007). Carrion-associated botulism is mostly caused by BoNT C or D but also described by BoNT B (Dirksen et al. 2006; Radostits et al. 2006). The so-called forage-associated botulism is mostly in connection with the ingestion of BoNT B (Rings 2004; Hogg et al. 2008). Botulism is difficult to verify by analytical tests for various reasons (Hogg et al., 1990; Galey et al., 2000; Rings, 2004; Sharpe et al. 2008). The "gold standard" for definite confirmation of botulism is the mouse bioassay (Radostits et al. 2006; Hogg et al. 2008).

The presence of BoNTs was proven in our case; it should still be kept in mind that cases with typical clinical signs but with no detection of BoNTs might be suspected cases of botulism as have been recently described in Ireland (Sharpe et al. 2008). In these cases, the clinical signs and the ruling out of other causes hint at the possibility of botulism (Hogg et al. 1990). Furthermore, there are implications for human food safety in botulism cases as the presence of BoNT B could be demonstrated in bovine milk (Bohnel et al. 2005). In addition, other modes of contamination in the dairy chain are possible and standard milk pasteurisation treatment does not eliminate spores from the milk originating from a dairy farm affected by botulism (Lindstrom et al., 2010).

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