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CASE REPORTS

Natural *Clostridium botulinum* Type C Toxicosis in a Group of Cats

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Clinical signs of botulism were observed in a group of eight cats, four of which died, after being fed pelican carrion. *Clostridium botulinum* type C was isolated from one cat. The microorganism and its toxin were found in the pelican. This is apparently the first report of natural botulism in cats.

CASE REPORTS

A dead adult white pelican (*Pelecanus onocrotalus*) was found beneath high-tension electricity lines near some fishponds and was taken for a study of the skeleton. On the next day necropsy revealed only generalized muscle hyperemia (thought to be consistent with death by electrocution). Muscle tissue (3 to 4 kg) of the pelican was offered to a group of eight "backyard" cats, which eagerly ingested all the meat.

On the following day (day 3), all the cats were mildly depressed and anorexic. One cat was recumbent and showed a flaccid paralysis, initially evident in the hind limbs, and dyspnea. No other clinical manifestations were seen. On day 4, another cat showed similar signs, progressing to quadriplegia, and three more were affected to a lesser degree. On day 5 the two very sick cats died, and by then all the other six cats presented mild to severe signs. On the night of day 5, two more cats died. On day 6, three cats were much improved clinically.

The remaining sick cat, a 5-month-old intact male domestic shorthair, was presented to the Hebrew University Veterinary Teaching Hospital. Physical examination revealed hypothermia (35.8°C), tachycardia (200 beats/min), and mild dehydration (6%). The cat was quadriparetic, with some voluntary movement of the front limbs, but not in the hind limbs. The withdrawal reflexes were absent in the hind limbs but were present, although very weak, in the front limbs. The patellar and sciatic reflexes were weak, and the cranial tibial and gastrocnemius reflexes were absent in both hind limbs. The tricep and bicep reflexes were very weak in both front limbs. Superficial and deep pain sensation was present in all limbs. The hopping and wheelbarrow responses could not be evaluated, as the cat could not stand. Cranial reflexes were normal. A complete blood count was performed and revealed only a mild mature neutrophilic leucocytosis (18×10^9 neutrophils/liter; reference interval [RI], 2.5×10^9 to 12.5×10^9 neutrophils/liter), with no toxic morphological changes. Serum biochemistry analysis revealed a mildly increased albumin concentration (40 g/liter; RI, 24 to 37 g/liter), decreased urea concentration (10.57 mmol/liter; RI, 15.35 to 22.85 mmol/liter), and increase in serum creatine kinase activity (732 U/liter; RI, 27 to 235 U/liter) and lactate dehydrogenase activity (431 U/liter; RI, 50 to 280 U/liter). Glucose concentration was normal (5.77 mmol/liter; RI, 3.88 to 6.66 mmol/liter). Urinalysis, performed by cystocentesis, was normal. Immunofluorescence antibody test (IFA) for *Toxoplasma gondii* was negative. Serum tested by a botulism bioassay was negative (see below). The cat had a good appetite and ate well and was treated with intravenous lactated Ringer's solution (10 ml/min) and ampicillin (75 mg every 8 h), with mild warming. During the first 24 h of hospitalization the cat did not urinate voluntarily and had a large overflow atonic urinary bladder, which was emptied by mild manual pressure. The cat improved gradually and regained the hind limb withdrawal reflexes 10 h from admission. Voluntary movements improved, and the cat was observed crawling 2 h later, by which time the body temperature normalized. Twenty-four hours after admission the frontal-limb reflexes were normal and the cat had full voluntary movements in the frontal limbs, although there was still some paraparesis. Withdrawal reflexes and the extensor thrust were normal in both hind limbs. Proprioception was normal in all limbs, and so was the hopping response. The first signs of urination and defecation were observed 38 h from admission. Twelve hours later the cat could stand normally but was still mildly paraparetic, and there was further improvement of all reflexes of both hind limbs. The cat was discharged 56 h after admission, with mild paraparesis, and subsequently continued to completely recover.

Two cats belonging to the same group but absent when the muscle was fed remained healthy throughout the event, thus linking morbidity with muscle consumption.

The last two cats to die were necropsied at the Kimron Veterinary Institute, but no abnormalities were found from gross pathology. Rabies was excluded by examining brain tissue by immunofluorescence. Blood from the hospitalized cat and stomach content and tissues from the two freshly dead cats were examined for likely toxicological causes. The presence of insecticide cholinesterase inhibitors (in stomach contents) was ruled out by gas chromatography/mass spectrometry (GC/MS).

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Residues of ionophore coccidiostats (in stomach contents) were ruled out by thin-layer chromatography, and lead and other elements (in kidney and liver) were ruled out by inductively coupled argon plasma-atomic emission spectroscopy. Acetylcholinesterase levels in blood determined spectrophotometrically were normal. A GC/MS screen of stomach contents revealed no abnormal chemical contents.

Brain, liver, spleen, and lung samples were inoculated onto nutrient agar, blood agar, and McConkey agar plates and incubated for 48 h at 37°C. Intestinal contents were enriched for salmonella isolation in tetrathionate broth at 37°C for 24 h and then inoculated onto McConkey and brilliant green agar plates. The plates were examined after further incubation for 24 h at 37°C. No pathogenic bacteria were found in the two cats.

The serum of one surviving sick cat, the stomach contents of two dead cats that died shortly before the examination, and the leg muscle tissue from the pelican were examined for the presence of preformed botulinic toxin. Serum (0.5 ml) was injected intraperitoneally (i.p.) into two 4-week-old mice. Stomach contents were suspended in phosphate buffer, pH 6, and centrifuged at 10,000 × *g* for 10 min. One part of the supernatant was heated to 80°C for 20 min, as a negative control. Finally 1 ml each of the test and inactivated supernatant was injected i.p. into two 4-week-old mice. Pelican muscle tissue was ground with sterile glass beads and then processed in the same fashion as the stomach contents. In addition, pelican muscle and the cat stomach contents were examined for the presence of toxigenic *Clostridium botulinum* bacteria. Samples were inoculated into fortified egg medium vials (13) and heated to 80°C for 20 min, sealed with paraffin oil, and incubated at 33°C for 5 days. The suspensions were centrifuged, and the supernatants (0.5 ml) were injected i.p. into two 4-week-old mice.

The neutralization test was performed when both mice died within 96 h of inoculation. The supernatant to be examined was diluted 5:1 with B, C, or D antitoxins (Centers for Disease Control and Prevention) and incubated for 30 min at 37°C. Five groups of two mice each were inoculated with 0.5 ml of one of the following: the native untreated supernatant, supernatant with B antitoxin, supernatant with C antitoxin, supernatant with D antitoxin, or the native supernatant heated at 80°C for 20 min. Survival of the group injected with one of the neutralized samples (in this case type C) and the heated samples established the final identification of the toxin.

Toxigenic *C. botulinum* type C bacteria were found in the stomach of one cat and in the pelican muscle. Preformed toxin was found in the pelican's muscle. Although the exact quantification of *C. botulinum* type C toxin present in the pelican muscle was not determined, the supernatant had to be diluted 1:1,000 before it could be neutralized by antitoxin (it is our experience that no dilution is usually necessary to achieve toxin neutralization), indicating an extremely high toxin content. It is possible that encountering the high-voltage lines was a consequence of the pelican's disorientation caused by mild, initial botulism.

Discussion. Six types of *C. botulinum* toxin, marked A to F (a seventh type, G, is produced by *Clostridium argentinense*), have been described, and a certain level of host specificity has been attributed to each. Thus, types A, B, and E are associated primarily with human intoxications (the last linked to the marine environment); botulism associated with types B and D is more prevalent in herbivores (horses and cattle), and type C is more prevalent in birds and is found rarely in carnivores (7). The most common animal species affected in Israel is the bovine (toxins D, C, and, more rarely, B) (16).

The origin of the microorganism and/or its toxin is primarily decaying animal tissue. The occurrence of botulism in carnivores is very rare. Several reports of type C botulism in dogs (14), including one following carrion consumption (5), and lions (6) have been published. To the best of our knowledge, however, there are no reports of natural botulism in cats, although these animals have been used as experimental models (4). Interestingly, the cats were found to be much more susceptible to parenteral exposure to botulism toxin than to oral exposure (9). Attempts to elucidate the mechanism upon which this difference in susceptibility is based using toxin neutralization by natural antibodies, tissues, and digestive enzymes were unsuccessful (9).

The presence of *C. botulinum* spores in the feces of clinically healthy animals is a controversial topic in the veterinary literature. Some authors believe that such spores are ubiquitous (15) and can be found in the intestine as a transient component of the enteric microflora (10), thus significantly reducing their diagnostic significance. Our experience, supported by findings of other authors (11, 12), is, however, that the amount of free toxin is frequently too low to be detected and that the presence of spores in feces of healthy animals is rare, thus making cultural methods suitable to support the clinical diagnosis of botulism (14).

The neurological signs in the hospitalized cat were compatible with an ascending lower motor neuron disease with no other involvement of the central nervous system, and the differential diagnoses included acute polyradiculoneuritis, tick paralysis, and acute myasthenia gravis (MG) (1, 2, 3, 5, 8). Tick paralysis could be ruled out as the cause because ticks have never been reported in Israel, and MG was ruled out on the basis of the cat's improvement with no specific therapy against this disease. Acute polyradiculoneuritis, mostly reported in dogs, is in many cases an idiopathic condition, although systemic illness or postvaccination reaction can induce the disease in cats (3, 8). The anamnesis, however, ruled out these conditions. Another supportive piece of evidence for botulism in this cat is the ascending pattern of neurological signs, which was also described in cases of canine botulism (1, 2, 5), with the order of the subsequent improvement from the front limbs to the hind limbs. Electrodiagnostic procedures were not performed in this case, but these usually assist only in the diagnosis and are not definitive. In most cases of botulism the diagnosis is primarily based on the history and suggestive clinical signs (8). The relative resistance of cats to oral botulinic intoxication is underlined by the quick recovery of the hospitalized animal, 3 days, in contrast to mildly affected canine cases that were reported to recover in 3 to 4 weeks (2).

Thus, although toxin was not detected in the afflicted cats, the epidemiological and clinical characteristic of the described case, the absence of other plausible etiological agents, the presence of a high concentration of *C. botulinum* type C toxin

in the putative source of intoxication, and the results of the laboratory examination constitute, in our opinion, strong evidence that the cats suffered from botulism.

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