BOTULISM

B05.01 OIE ☐ BALAI ☐ EU AHL ☐

BACTERIA *Clostridium botulinum* ZOONOSIS ☐

SUSCEPTIBLE ANIMAL GROUPS	TRANSMISSION	CLINICAL SIGNS	SEVERITY	TREATMENT	PREVENTION AND CONTROL
Mammals and birds (mainly ruminants, equids, primates, mustelids, waterfowl)	Spore or toxin ingestion Wound contamination	Progressive flaccid paralysis	Fatal	Supportive care Anti-toxin administration	Good husbandry practices Toxoid vaccination not available in EU

FACT SHEET COMPILED BY

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DISEASE AGENT

Clostridium botulinum is a gram-positive, spore-forming, obligatory anaerobic rod belonging to the noninvasive clostridia. It produces seven protein neurotoxins (A to G) that have a similar pharmacologic action but differ in potency, antigenic properties, and distribution. Any of the neurotoxins can cause a neuroparalytic intoxication, although type C is the most frequent in animals notably in non-human primates, wild ducks, pheasants, chickens, mink, cattle and horse. Type D can be encountered in cattle (causing "lame disease", linked to phosphorus-deficient ranges, where grazing animals feed on carcasses and bones that often contain botulinum toxin), horses and sporadically in dogs. Types A and E have occasionally caused botulism in minks. Type B toxin is generally associated with the "shaking foal" syndrome. Type F botulism only occurs in people but type A, B, and E toxins also are important in human botulism.

SUSCEPTIBLE ANIMAL GROUPS

Many avian and mammalian hosts, including ruminants, equids, primates (*Papio hamadryas*, *Hylobates lar*, Callitrichidae), mustelids, game birds, fowl (particularly ducks) and other birds like loons, mergansers, geese and gulls. Fish are also sensitive. Swine are reported to be resistant, whereas carnivores are rarely infected. Incidence of the infection, which is not precisely known, is relatively low in cattle and horses, more frequent in chickens, and high in wild waterfowl.

ZOONOTIC POTENTIAL

None: there is no direct transmission to humans. However, people can be indirectly infected by toxin ingestion, spore ingestion (important in human infant botulism) and wound contamination by spores from the environment.

DISTRIBUTION

Worldwide.

TRANSMISSION

Reservoirs of *Clostridium botulinum* are soil and aquatic sediments. Contaminated animals and plant materials can act as a vehicle of toxi-infection. Transmission occurs when spores or toxins are ingested (contaminated feed), or by wound contamination.

INCUBATION PERIOD

Varies with level of exposure: 12 hours to 10 days in cattle and horses.

CLINICAL SIGNS

Progressive flaccid paralysis progressing cranially. Death occurs in 6 to 72 hours after the onset of clinical signs, as a result of respiratory or cardiac arrest. Motor paralysis can cause drowning of waterfowl. Other clinical signs are also caused by muscle paralysis and include disturbed vision, difficulty in chewing and swallowing, and generalized progressive weakness.

Ruminants: drooling, inability to urinate, dysphagia, protrusion of the tongue, sternal then lateral recumbency. Equids: dysphagia, protrusion of the tongue. Primates: sudden death or lethargy with mild ataxia of the hindquarters progressing to severe ataxia, drowsiness, flaccid paralysis, respiratory distress, collapse and death. Mustelidae: sudden death, dyspnea, flaccid paralysis. Birds: typical flaccid paralysis of the neck, but also of the legs and wings (inability to fly). In waterfowl, neck paralysis can lead to drowning.

PATHOLOGY AND POST MORTEM FINDINGS

No characteristic lesions. Pulmonary edema and congestion can be noticed, as well as excessive pericardial fluid containing free-floating fibrin. Congestion of some intestinal loops can also be observed.

DIAGNOSIS

A presumptive diagnosis is often made based on clinical signs and exclusion of other causes of motor paralysis. Demonstration of toxin in feedstuffs, fresh stomach contents or vomitus supports the diagnosis. Confirmation is based on toxin identification in serum, intestinal content or tissue, before death or from a fresh carcass. Isolation of the organism, especially from intestinal contents, and postmortem demonstration of toxin are not definitive.

Toxin is extracted from suspected material. Samples are homogenized in an appropriate buffer, centrifuged, and injected intraperitoneally to susceptible laboratory animals (mice). Clinical signs and death depend on the amount of toxin injected and generally occur in 3 hours to 3 days. *Clostridium botulinum* can be detected in enrichment cultures from suspected samples by toxin production and PCR analysis targeted on neurotoxin genes. Isolation of *Clostridium botulinum* on agar plates and further characterization require strict anaerobic conditions and are performed in specialized laboratories.

SAMPLES REQUIRED FOR LABORATORY ANALYSIS

Serum, intestinal content or tissue samples.

TREATMENT

In case of suspected recent toxin ingestion, evacuation of the stomach and purging are helpful. Clinical cases can be managed with supportive care and administration of specific anti-toxin. However anti-toxin treatment response and prognosis vary considerably with the type of toxin and the species of the host (often successful for mink and ducks). Some affected birds may also recover without treatment.

Antibiotics should be used in case of suspected aspiration pneumonia but are not reported to be effective against toxi-infectious or wound botulism. *Clostridium* spp. are generally resistant to aminoglycosides, and penicillins (ampicillin *per os*) should be preferred to attempt an antimicrobial therapy. Guanidine hydrochloride (11 mg/kg body weight) and aminopyridine stimulate acetylcholine release and have some therapeutic efficacy against type A botulism, whereas germine intensifies neural impulses. Nonetheless, the use of these molecules has been reported only a few times and not extended enough to determine their value.

PREVENTION

Good husbandry practices (keeping vermin populations low, buying feed from reputable sources). Primates should not be kept in enclosures previously used for ungulates. Removal of affected waterfowl to dry land. Quick disposal of carcasses. Removal of decaying grass or spoiled silage from the diet.

Immunization with toxoid vaccines (not available in Europe). Types A, B, C and D vaccines were developed for mink, types C and D toxoids were successfully used in cattle in South Africa and Australia while type B is available for horses in North America, and the type C mink vaccine was successfully used in waterfowl.

CONTROL

Chlorhydric acid, formaldehyde, glutaraldehyde, calcium hypochloride, sodium hypochloride, ethylene oxyde, and

hydrogene peroxyde have a sporicide activity depending on their concentration and duration of action. Poultry litter should be treated (although not always effective) by sodium bisulfate at 1kg/200 square meters.

LEGISLATIVE REQUIREMENTS

Not notifiable under OIE 2019, BALAI (Council Directive 92/65/ECC) or AHL (Regulation EU 2016/429). Might be notifiable according to national law of each country.

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