BOTULISM

ETIOLOGY

The causative organism *Clostridium botulinum*, a spore-forming anaerobe, produces neurotoxins during vegetative growth. Spores can survive in the environment for over 30 years.

Under favorable conditions of warmth and moisture the spores germinate and vegetative cells multiply rapidly. elaborating a stable and highly lethal toxin which, when ingested, or absorbed from tissues, causes the disease. The toxin is also capable of surviving for long periods, particularly in bones or if protected from leaching. Seven antigenic ally distinct toxin types (A-G), some with subtypes, have been identified. Farm animal disease is produced primarily by types B, C and D. .

**EPIDEMIOLOGY**

**Occurrence**

The disease occur in most countries.

The source of exposure to toxin and the risk for disease differ between regions because of differences in food storage, feeding, and management practices.

**Source of infection**

1-Most incidents of botulism are associated with the ingestion of preformed toxin(forage botulism) .

2-Toxin in feeds may result from the primary growth of *C. botulinum* in the feed or from the contamination of the feed with toxincontaining carrion (carrion-associated botulism) .

3- Less common sources are(wound botulism) or growth and toxin production in the alimentary tract (toxicoinfectious botulism) .

**Forage botulism**

Forage botulism occurs when pH, moisture, and anaerobic conditions in the feedstuff allow the vegetative growth of C*. botulinum* and the production of toxin.

* Big bale silage is a particular risk. The type of forage ensiled in big bales often has insufficient water-soluble carbohydrate for adequate lactic acid fermentation to achieve a stable low pH, and the higher dry matter content can also lead to a higher pH.3 Clostridial multiplication is inhibited below pH 4.5.
* Proliferation of the organism can occur in decaying vegetable material.
* Poultry manure and ensiled poultry litter have caused outbreaks of botulism when used as cattle feed, bedding , or in cattle and sheep are grazing in pastures that have been fertilized with poultry manure or poultry litter, It is probable that the source of toxin in poultry litter is from poultry carcasses.
* Direct carrion ingestion can occur where cattle subsist on a phosphorusdeficient diet and manifest osteophagia, with subsequent ingestion of carrion.
* In sheep, pica is more usually associated with a dietary deficiency of protein or net energy.
* Occasional outbreaks occur that are due to drinking of water contaminated by carcasses of dead animals.
* Wound botulism is rare but is recorded in horses following castration, umbilical hernias treated with clamps.

**Risk factors**

**Animal risk factors**

Botulism is most common in birds, particularly the domestic chicken and wild waterfowl. Cattle, sheep, and horses are susceptible but pigs, dogs, and cats appear to be resistant. The horse appears to be particularly susceptible to type B toxin. Cattle and sheep are usually affected by types C and D.

**Environment risk factors**

Botulism in range animals has a seasonal distribution. Outbreaks are most likely to occur during drought periods when feed is sparse, phosphorus intake is low and carrion is plentiful. Silage-associated botulism is also seasonal with the feeding of silage.

**Zoonotic implications**

The meat and milk from cattle that have botulism should not be used for human consumption.

**PATHOGENESIS**

The toxins of C. botulinum are neurotoxins and produce functional paralysis without the development of histological lesions. Botulinum toxins are absorbed from the intestinal tract or the wound and carried via the bloodstream to peripheral cholinergic nerve terminals including neuromuscular junctions, postganglionic parasympathetic nerve endings, and peripheral ganglia. The heavy chain of the toxin is responsible for binding to the receptors and translocation into the cell and the light chain of the toxin for resultant blockade of the release of acetylcholine at the neuromuscular junction. Flaccid paralysis develops and the animal dies of respiratory paralysis.

CLINICAL FINDINGS

**Cattle and horses**

Signs usually appear 3-17 days after the animals gain access to the toxic material,

**Peracute**

1-cases die without prior signs of illness,

2-The disease is not accompanied by fever

3-progressive symmetric muscular paralysis affecting particularly the limb muscles and the muscles of the jaw, tongue, and throat.

4-Muscle weakness and paralysis commence in the hindquarters and progress to the forequarters, head, and neck.

muscle tremor and fasciculation, often sufficient to make the whole limb tremble. Colic may be an initial sign in horses.

**sub acute.**

1-Restlessness, incoordination, stumbling, knuckling, and ataxia are followed by inability to rise or to lift the head.

2-Mydriasis and ptosis occur early in the clinical course

3-Skin sensation

4-In some cases the tongue becomes paralyzed and hangs from the mouth, unable to chew or swallow and it drools saliva.

5- Affected animals lie in sternal recumbency with the head on the ground or turned into the f1ank, similar to the posture of a cow with parturient paresis.

6-Ruminal movements are depressed. Defecation and urination are usually unaffected

7-Paralysis of the chest muscles results in a terminal abdominal-type respiration.

8-Sensation and consciousness are retained until the end.

**Sheep**

1- in Sheep do not show the typical flaccid paralysis until the final stages of the disease.

2- stiffness while walking, and in coordination and some excitability in the early stages.

3-The head may be held on one side or bobbed up and down while walking (limber neck) .

4-Lateral switching of the tail, salivation, and serous nasal discharge .

5-In the terminal stages there is abdominal respiration, limb paralysis, and rapid death.

**Diagnosis**

1-History and management , presence of hypophosphatemia and protein deficiency

2-Muscle enzyme activities

3-Detection of preformed toxin in serum, intestinal tract contents, or feed.

4- Demonstration of spores of C. botulinum in the feed or gastrointestinal contents.

5- Detection of antibody in recovering or clinically normal at-risk animals by ELISA test.

**Differential diagnosis**

 **Ruminants**

Clinically and at necropsy the disease resembles parturient paresis in cattle and hypocalcemia in sheep but the conditions under which the diseases occur are quite different.

• Tick paralysis

• Paralytic rabies

• Poisoning by Phalaris aquatica

• Organophosphate/carbamate poisoning

• Louping ill in sheep

**Horses**

• Equine protozoal myelitis

• Equine encephalomyelitis

• Hepatic encephalopathy

• Paralytic rabies

**NECROPSY FINDINGS**

There are no specific changes detectable at necropsy, although the presence of suspicious feedstuffs in the forestomachs or stomach may be suggestive ( nonspecific subendocardial and subepicardial hemorrhages and congestion of the intestines).

**Treatment**

1-high quality of intensive care fluid therapy, enteral or parenteral feeding, nasal insufflation with oxygen and mechanical ventilation if required.

2-Specific or polyvalent antiserum, if administered early in the course at a dose of 30 000 IU for a foal and 70 000 IU for adult horses.

3-Muzzling may be required to prevent aspiration pneumonia and frequent turning to prevent muscle necrosis and decubital ulcers.

4-Bladder catheterization may be required in horses that do

not urinate

5-Mineral oil is used to prevent constipation.

6- antimicrobial drugs are used to treat secondary complications such as aspiration pneumonia. Therapy should avoid the use of drugs that deplete the neuromuscular junction of acetylcholine, such as neostigmine, and those, such as procaine penicillin, tetracyclines, and aminoglycosides, that potentiate neuromuscular weakness.

**CONTROL**

1- correction of dietary deficiencies by supplementation with phosphorus or protein.

2- Hygienic disposal of carcasses is advisable to prevent further pasture contamination.

3- Vaccination.