Lecture title: Botulism

Synonyms : Shaker Foal Syndrome

Lecturer Affiliation: Department of Internal and preventive medicine

Summary:

Definition : **Botulism** is a progressive, flaccid paralysis resulting from *Clostridium botulinum* toxin production and absorption that can be rapidly fatal if not aggressively treated for the first signs of intoxication.

Botulism is a rapidly progressive neurologic disease with high mortality (100% in untreated animals).

Etiology

Clostridium botulinum (that produces an **exotoxin**)is an anaerobic, spore-forming bacterium, ubiquitous in soil..

Species affected: Cattle, sheep, horses, wild birds and poultry, and mink and ferrets. Dogs and pigs can also be affected, however, they seem to be more resistant to the disease.

Among livestock, **horses** appear to be the most susceptible to the effects of the botulinum toxin

Risk Factors

• Decaying vegetable matter in food and water.

• Soil contamination of feed (access to round bales)

• Animal carcass contamination (bird, fish, rodents, etc.) of feed, or standing water.

Outbreaks of botulism in livestock occur under a variety of conditions, but can in general be subdivided into two main categories:

- those associated with phosphorus deficiency and osteophagia in which the disease occurs sporadically over a relatively long period of time in animals

(mainly cattle, but also to a lesser extent sheep and goats) maintained under extensive ranching conditions, and

- secondly the form of cattle, sheep, goats and horses which are associated with the ingestion of toxic feed or water and which may result in the deaths of large numbers of animals over a short period of time.

Transmission

• Forage poisoning—consuming feed or forage containing **pre-formed toxins of** *C. botulinum*

• Wound contamination—allowing germination of spores and absorption of toxin from hypoxic areas

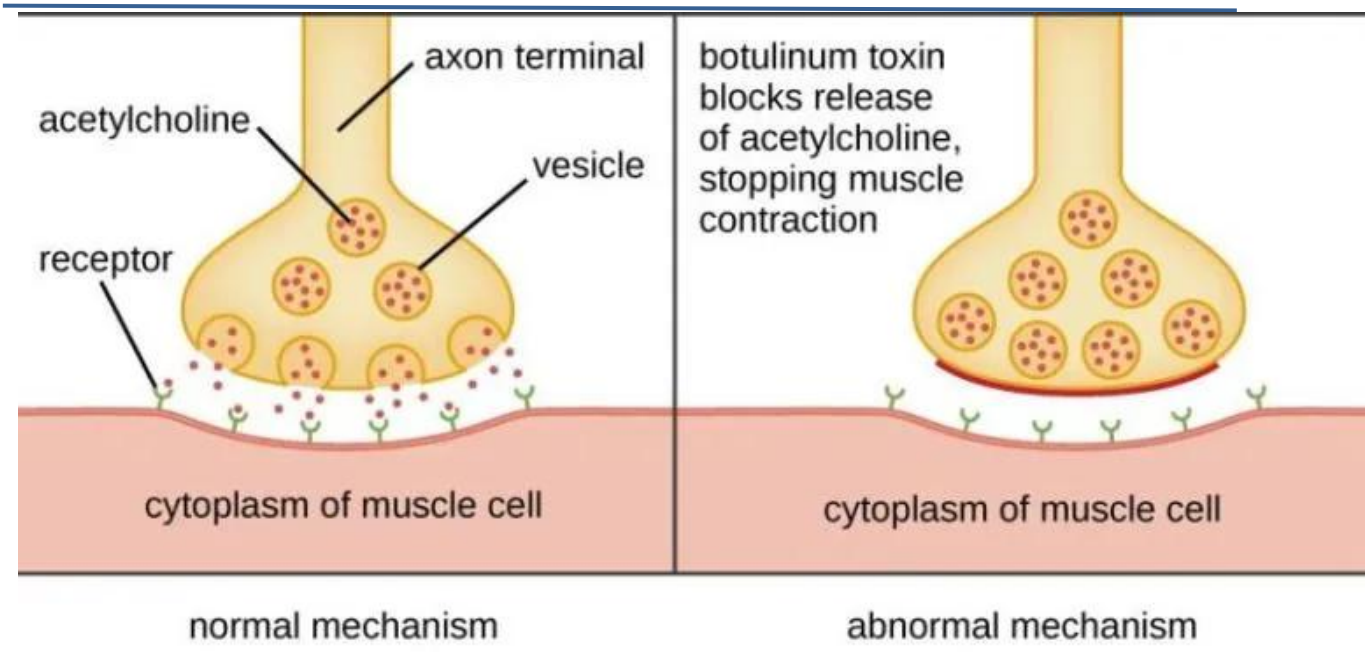
• Toxoinfectious ('Shaker foals')—consuming forage or feed containing the bacteria or spores by neonates suffering gastric ulceration may precipitate diseases. Necrotic/hypoxic foci allow germination of *C. Botulinum* spores and development of the vegetative bacterial form, enabling the production, liberation and local absorption of causative toxins.

Pathogenesis:

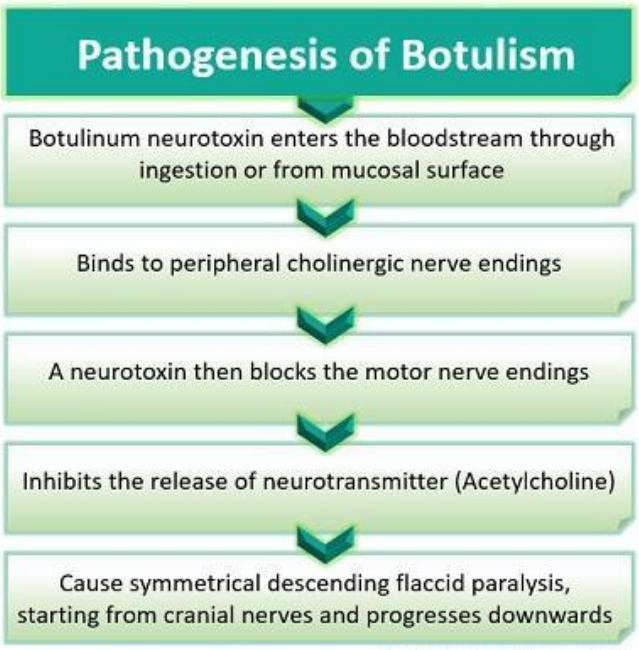
Botulinum toxins are absorbed from the intestinal tract or the wound and carried via the bloodstream to peripheral cholinergic nerve terminals including neuromuscular junctions, postganglionic parasympathetic nerve endings, and peripheral ganglia.

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 resultant blockade of the release of acetylcholine at the neuromuscular junction. Flaccid paralysis develops and the animal dies of respiratory paralysis.

In normal condition: Upon stimulation of peripheral and cranial nerves, acetylcholine is normally released from vesicles at the neural side of the motor endplate. Acetylcholine then binds to specific receptors on the muscle, inducing **contraction**.



Botulinum toxin acts by binding **to synaptic vesicles of cholinergic nerves**, thereby preventing the release of **acetylcholine (Ach)** at the peripheral nerve endings, including neuromuscular junctions. This results in a lack of stimulus to the muscle fibers, irreversible relaxation of the muscles**,** and **flaccid paralysis.**



Clinical Signs

The course of the disease can be very rapid, being 24 to 48 hours depending on the level of toxin or bacterial ingestion.

Wound contamination and toxoinfectious cases may have variable and prolonged periods from inoculation of the organism until toxin release precipitating clinical signs.

1- Peracute form: sudden death without any signs.
2- Subacute form

Clinical signs include:

• Severe muscle weakness
• Flaccid paralysis with normal mentation
• Inability to swallow (foals will reflux milk from the nostrils)
• Poor tail, tongue and eyelid tone
• Hypoventilation, respiratory arrest
• Paresis/inability to stand for extended periods • Limb paralysis
• Progression to muscular weakness and recumbency
• Drooling

Necropsy findings : None specific

Diagnosis:.

Diagnostic confirmation

1- clinical signs
2- Demonstration of toxin in intestinal contents, serum, or feed , or wounds.By Direct ELISA and mouse neutralization test.

Control:

1- Correction of dietary deficiencies.
2- Hygienic disposal of carcasses.
3- Vaccination.