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Lecture title: **Tetanus**

Lecturer Affiliation: **College of Veterinary Medicine**

Summary:

ETIOLOGY:

1- Tetanus (Locked jaw) is caused by *Clostridium tetani*, a gram positive, spore-forming obligate anaerobe bacillus.
2- It is a ubiquitous organism and a commensal of the gastrointestinal tract of domestic animals and humans.
3- The organism forms highly resistant spores that can persist in soil for many years. The spores survive many standard disinfection procedures, including steam heat at 100°C for 20 minutes but can be destroyed by heating at 115°C for 20 minutes.
4- After a period of anaerobic incubation spores germinate to their vegetative form, which starts replicating and producing a complex of exotoxins causing the clinic signs characteristic for this condition. The toxins produced are tetanolysin, tetanospasmin, and neurotoxin or non-spasmolytic toxin.

EPIDEMIOLOGY:

- **Occurrence**:

Tetanus occurs in all parts of the world and is most common in closely settled areas under intensive cultivation. It occurs in all farm animals, mainly as individual, sporadic cases, although outbreaks are occasionally observed in young cattle, young pigs, and lambs following wound management procedures.

- **Case Fatality Rate:**

In young ruminants the case–fatality rate is over 80%, but the recovery rate is high in adult cattle. In horses it varies widely between areas. In some areas almost all animals die acutely, and in others the mortality rate is consistently about 50%.

- **Source of Infection:**

C. tetani organisms are commonly present in the feces of animals, especially horses, and in the soil contaminated by these feces. Surveys in different areas of the world show it is present in 30% to 42% of soil samples. The survival period of the organism in soil varies widely from soil to soil.

- **Transmission:**

1- The portal of entry is usually through deep puncture wounds, but the spores may lie dormant in the tissues for some time and produce clinical illness only when tissue conditions favor their proliferation.
2- Puncture wounds of the hooves are common sites of entry in horses. Introduction to the genital tract at the time of parturition is the usual portal of entry in cattle. A high incidence of tetanus may occur in lambs following castration, shearing, docking, vaccinations, or injections of pharmaceuticals, especially anthelmintic.
3- Neonatal tetanus occurs when there is infection in the umbilical cord associated with unsanitary conditions at parturition.
4- Cases of tetanus in ruminants after thermic dehorning and ear-tagging have been reported.
5- Outbreaks of "idiopathic tetanus" occur occasionally in young cattle without a wound being apparent, usually in association with the grazing of rough, fibrous feed, and it is probable that toxin is produced in wounds in the mouth or gastrointestinal tract or is ingested preformed in the feed. Proliferation in the rumen may also result in toxin production.

- **Animal Risk Factors:**

  1- The neurotoxin of *C. tetani* is exceedingly potent, but there is considerable variation in susceptibility between animal species, and horses are the most susceptible and cattle the least susceptible.
  2- The variation in prevalence of the disease in the different species is partly caused by this variation in susceptibility but is also because exposure and wound management practices are more likely to occur in some species than in others.

PATHOGENESIS:

1- The tetanus spores remain localized at their site of introduction and do not invade surrounding tissues.
2- Spores germinate to their vegetative form to proliferate and produce tetanolysin, tetanospasmin, and neurotoxin only if certain environmental conditions are attained, particularly a lowering of the local tissue oxygen tension.
3- Toxin production may occur immediately after introduction if the accompanying trauma has been sufficiently severe, or if foreign material has also been introduced to the wound, or may be delayed for several months until subsequent trauma to the site causes tissue damage.
4- Of the three mentioned exotoxins, tetanospasmin is the most relevant for the pathophysiology of the condition. Although tetanolysin was found to promote local tissue necrosis, its role in the pathogenesis of tetanus remains doubtful.
5- Tetanospasmin diffuses to the systemic circulation, is bound to motor end plates, and travels up peripheral nerve trunks via retrograde intra axonal transport to the CNS.
6- The exact mechanisms by which the toxin exerts its effects on nervous tissue are not known, but it blocks the release of neurotransmitters such as GABA and glycine. No structural lesions are produced.
7- Death occurs by asphyxiation caused by fixation of the muscles of respiration.

CLINICAL FINDINGS:

1- The incubation period varies between 3 days and 4 weeks, with occasional cases occurring as long as several months after the infection is introduced.
2- In sheep and lambs cases appear 3 to 10 days after shearing, docking, or castration.
3- Clinical findings are similar in all animal species. Initially, there is an increase in muscle stiffness, accompanied by muscle tremor. There is trismus with restriction of jaw movements; prolapse of the third eyelid; stiffness of the hind limbs causing an unsteady, straddling gait; and the tail is held out stiffly, especially when backing or turning. Retraction of the eye and prolapse of the

third eyelid, is one of the earliest and consistent signs (with the exception of sheep).

4- Additional signs include an anxious and alert expression contributed to by an erect carriage of the ears, retraction of the eyelids and dilation of the nostrils, and hyperesthesia with exaggerated responses to normal stimuli.

5- The animal may continue to eat and drink in the early stages but mastication is soon prevented by tetany of the masseter muscles and saliva may drool from the mouth. If food or water is taken, attempts at swallowing are followed by regurgitation from the nose.

6- Constipation is usual and the urine is retained, partly as a result of the inability to assume the normal position for urination. The rectal temperature and pulse rate are within the normal range in the early stages but may rise later when muscular tone and activity are further increased.

7- In cattle, particularly young animals, bloat is an early sign but is not usually severe and is accompanied by strong, frequent rumen contractions As the disease progresses, muscular tetany increases and the animal adopts a sawhorse posture.

8- There is great difficulty in walking and the animal is inclined to fall, especially when startled. Falling occurs with the limbs still in a state of tetany and the animal can cause itself severe injury.

9- Tetanic convulsions begin in which the tetany is still further exaggerated. Opisthotonus is marked, the hindlimbs are stuck out stiffly behind and the forelegs forward. Sweating may be profuse and the temperature rises, often to 42°C. The convulsions are at first only stimulated by sound or touch but soon occur spontaneously.

10- In fatal cases there is often a transient period of improvement for several hours before a final, severe tetanic spasm during which respiration is arrested. The course of the disease and the prognosis vary both between and within species.

11- The duration of a fatal illness in horses and cattle is usually 5 to 10 days, but sheep usually die on about the third or fourth day.

CLINICAL PATHOLOGY:

1- There are no specific abnormalities in blood or CSF and no antemortem test confirming the diagnosis.
2- Blood levels of tetanus toxin are usually too low to be detected. Gram stain of wound aspirates is considered of limited value because sporulated as well as vegetative forms of *C. tetani* resemble other anaerobic bacteria.
3- Culturing the pathogen is difficult because of the low number of organisms normally present and the strict anaerobic conditions required for culture.
4- Culture in combination with PCR has been used for identification of *C. tetani*. A bioassay consisting of injecting infectious material into the tail base of mice and observing for onset of characteristic clinical signs is possible.

NECROPSY FINDINGS:

1. There are no gross or histologic findings by which a diagnosis can be confirmed.
2. Culture of the organism is difficult but should be attempted.
3. If minimal autolysis has occurred by the time of necropsy, the identification of large gram-positive rods with terminal spores (“tennis-racket morphology”) in smears prepared from the wound site or spleen is supportive of a diagnosis of tetanus.

DIFFERENTIAL DIAGNOSIS:

Fully developed tetanus is so distinctive clinically that it is seldom confused with other diseases. The muscular spasms, the prolapse of the third eyelid, and a recent history of accidental injury or surgery are characteristic findings. However, in its early stages or mild forms, tetanus may be confused with other diseases.

All species:
• Strychnine poisoning
• Meningitis

Horses:
• Hypocalcemic tetany (eclampsia)
• Acute laminitis
• Hyperkalemic periodic paralysis
• Myositis, particularly after injection in the cervical region.

Ruminants:

• Hypomagnesemia (cows, sheep and calves)
• White muscle disease
• Polioencephalomalacia
• Enterotoxemia

TREATMENT:

1- Eliminate the causative bacteria, the parenteral administration of penicillin in large doses (44,000 IU/kg), preferably by intravenous administration.
2- Neutralize residual toxin. The objective of administering tetanus antitoxin is to neutralize circulating toxin outside the CNS.
3- Control muscle spasms until the toxin is eliminated or destroyed.
4- Relaxation of the muscle tetany can be attempted with various drugs. Chlorpromazine (0.4–0.8 mg/kg BW intravenously, or 1.0 mg/kg BW intramuscularly, three or four times daily).
5- Maintain hydration and nutrition.

CONTROL:
1- Many cases of tetanus could be avoided by proper skin and instrument disinfection at castrating, docking, and shearing time. These operations should be performed in clean surroundings; in the case of lambs docked in the field, temporary pens are preferred over permanent yards for catching and penning.

2- Passive Immunity:

Short-term prophylaxis can be achieved by the injection of 1500 IU of tetanus antitoxin. The immunity is transient, persisting for only 10 to 14 days.

3- Active Immunity:

Available vaccines are formalin-inactivated adjuvanted toxoids; they induce long-lasting immunity. Primary vaccination requires two doses 3 to 6 weeks apart. Protective titers are obtained within 14 days of the second injection and last for at least a year and up to 5 years.