

DISEASES CAUSED BY CLOSTRIDIUM SP.-PATHOGENESIS, TREATMENT , PREVENTION AND CONTROL OF TETANUS

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DISEASES CAUSED BY CLOSTRIDIUM SPP

Tetanus - <i>Clostridium tetani</i>	Enterotoxemia caused by <i>Cl. perfringens</i> type D (pulpy kidney, overeating disease)
Bacillary hemoglobinuria	
Botulism- <i>Cl. botulinum</i>	Braxy (bradsot)
Enterotoxemia caused by <i>Cl. perfringens</i> type A	Focalsymmetrical encephalomalacia
Blackleg <i>Cl.chauvoei</i>	Infectious necrotic hepatitis (black disease)
Enterotoxemia caused by <i>Cl. perfringens</i> type B,C andE	Enterocolitis associated with <i>Clostridium difficile</i>
Malignant edema (gas gangrene)	

PATHOGENESIS

The tetanus bacilli remain **localized** at their site of introduction and do not invade surrounding tissues. They proliferate and produce tetanolysin and tetanospasmin only if certain environmental conditions are attained, particularly lowering of the local tissue oxygen tension. Tetanolysin promotes local tissue necrosis. 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. The original injury may be inapparent by them.

Tetanospasmin diffuses to the systemic circulation, is bound to motor end-plates and travels up peripheral nerve trunks via retrograde intra axonal transport. The exact mechanisms by which the toxin exerts its effects on nervous tissue are not known (2) but it blocks the spontaneous and nerve impulse evoked release of neurotransmitter resulting in the disinhibition of gamma motor neurons. No structural lesions are produced but there is central potentiation of normal sensory

stimuli so that a state of constant muscular spasticity is produced normally innocuous stimuli cause exaggerated responses. Death occurs by asphyxiation due to fixation of the muscles of respiration.

CLINICAL FINDINGS

The **incubation period** varies between 3 days and 4 weeks with occasional cases occurring as long as several months after the infection is introduced. In sheep and lambs cases appear 3-10 days after shearing or docking.

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 lambs causing an untidy, straddling gait and the tail is held out stiffly especially when backing or turning. Retraction of the eye and prolapse of the third eyelid across the cornea followed by a slow retraction – is one of the earliest and consistent signs (with the exception in sheep) and can be exaggerated by sharp lifting of the muzzle or tapping the face below the eye. 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.

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 are taken, attempts at swallowing are followed by regurgitation from the nose. Constipation is usual and the urine is retained due in part to inability to assume the normal position of 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. 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. Uneven muscular contractions may cause the development of a curve in the spine and deviation of the tail to one side. 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. Once down it is almost impossible to get a large animal to its feet again. Titanic convulsions begin in which the tetany is still further exaggerated. Opisthotonos is marked the hind limb are stuck out stiffly behind and the forelegs forward. Sweating may be profuse and the temperature rises, often to 42 degree C (107°F). The convulsions are at first only stimulated by sound or touch but soon occur spontaneously. In fetal cases there is often a transient period of improvement for several hours before final severe titanic spasm during which respiration is arrested.

The course of the disease and the prognosis vary both between and within species. The duration of a fatal illness in horses and cattle is usually 5-10 days but sheep usually die on about the third or fourth day. A long incubation period is usually associated with a mild syndrome, a long course and a favourable prognosis. Mild cases which recover usually do so slowly, the stiffness disappearing gradually over a period of weeks or even months. The prognosis is poor when signs rapidly progress. Animals vaccinated in the past year have a better prognosis, as do horses that have received parenteral penicillin and tetanus antitoxin and in which the wounds was aggressively cleaned when fresh.

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, 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

The main principles in the treatment of tetanus are to :

- Eliminate the causative bacteria
- Neutralize residual toxin
- Control muscle spasms until the toxin is eliminated or destroyed
- Maintain hydration and nutrition
- Provide supportive treatment

There are no structural changes in the nervous system, and the management of cases of tetanus depends largely on keeping the animal alive through the critical stages.

Elimination of the organism is usually attempted by the parenteral administration of penicillin in large doses, preferably by intravenous administration. If the infection site is found, the wound should be aggressively cleaned and debrided but only after antitoxin has been administered, because debridement, irrigation with hydrogen peroxide, and the local application of penicillin may facilitate the absorption of the toxin.

Tetanus antitoxin is administered but is of little value once signs have appeared. After the experimental administration of toxin, antitoxin is of limited value at 10 hours and ineffective by 48 hours. The recommended dose is controversial but for optimal results horses should receive 30000 units 12 hourly for 3 injections. Local injection of some of the antitoxin around the wound is advised. There have been a number of attempts to justify the treatment of early cases of equine tetanus by the intrathecal injection of antitoxin but there is limited evidence of therapeutic value and the procedure has risk.

Relaxation of the muscle tetany can be attempted with various drugs, Chlorpromazine (0.4 -0.8 mg/kg body weight intravenously, 1.0 mg/kg body weight intramuscularly, t.i.d. to q.i.d.) and acetyl promazine (0.05 mg/kg body weight b.i.d.) administered until severe signs subside, are widely used . A combination of diazepam (0.01 – 0.4 mg/kg) and xylazine (0.5 -1.0 mg/kg, IV or IM) may be effective in horses refractory to phenothiazine tranquilizers.

Hydration can be maintained by intravenous or stomach-tube feeding during the critical stages when the animal cannot eat or drink. The use of an indwelling tube should be considered because of the disturbance caused each time the stomach tube is passed. Feed and water containers should be elevated and the feed should be soft and moist.

Additional supportive treatment includes slinging of horses during the recovery period when hyperesthesia is diminishing. Affected animals should be kept as quiet as possible and provided with dark, well-bedded quarters with non slip flooring and plenty of room to avoid injury if convulsions occur. Administration of enemas and catheterization may relieve the animals discomfort. This level of nursing, plus penicillin, ataractic drug, and antitoxin for an average of 14 days can deliver something like a 50% recovery by an average of 27 days, but the cost is

high. Horses that fall frequently sustain bone fractures and may need to be destroyed.

CONTROL

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

Passive Immunity

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

Tetanus antitoxin

Tetanus antitoxin should be given to any horse with a penetrating wound or deep laceration, and the wound should also be cleaned aggressively. Tetanus toxoid can be administered at the same time as tetanus antitoxin, provided they are injected at different sites and using different syringes. Animals which suffer injury are usually given an injection of antitoxin and one of toxoid to insure complete protection.

Tetanus antitoxin is often routinely given to mares following foaling and to newborn foals. In some areas the risk for tetanus in young foals is high and repeated doses of antitoxin at weekly intervals may be required for protection. On farms where the incidence of tetanus in lambs is high, antitoxin is usually given at the time of docking or castration; 200 IU has been shown to be effective. The risk for tetanus in calves is lower than in lambs and tetanus antitoxin is not commonly given at the time of castration.

There is a risk for serum hepatitis in horses that have been given tetanus antitoxin and a policy of routine active immunization of the mare to provide the mare with active immunity and the foal with passive colostral immunity is preferred to one that relies on antitoxin. Provided

foals get an adequate supply of colostrums they are protected during the first 10 weeks of life by active vaccination of the mare during the last weeks of pregnancy. Prevention of tetanus in new born lambs is also best effected by vaccination of the ewe in late pregnancy.

Active Immunity

Available vaccines are formalin inactivated adjuvanted toxoids; they induce long lasting immunity. Primary vaccination requires 2 doses 3-6 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.

Foals should receive primary vaccination at 3-4 months of age. Whereas immunity lasts longer than one year it is common to revaccinate horses yearly with a single booster injection. Pregnant mares should receive a booster injection 4-6 weeks before foaling to provide adequate colostrum immunity to the foal.

Ewes are immunized with a similar schedule except that the primary doses are usually given at a managementally convenient time when the flock is yarded. A booster pre lambing booster vaccination a given yearly. Commonly commercial vaccine for sheep also contain antigens for other clostridial diseases for which sheep have high risk.

Vaccination of cattle is usually not considered unless an outbreak of the disease has occurred in the immediate past and further cases maybe anticipated.