TETANUS

by

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DEFINITION

Tetanus is a highly fatal toxemia due to infection of injured or otherwise devitalized tissues by *Clostridium tetani.* It is characterized clinically by hyperesthesia, tetany and convulsions.

HISTORY

Tetanus has been recognized as a killer since its description by Hippocrates.[1] It was described by Aspyrtus in horses in the fourth century.[4] It was stated in 1896 that tetanus was due to a germ, *Tetanus bacillus,* entering the system through an abrasion of the skin or mucous membranes. In 1904 it was said to be caused by the bacillus of Nicolaier, and more recently by the *Clostridium tetani* organism.

Morphology

Clostridium tetani is a long slender rod, 0.5 microns in diameter by 2 to 5 microns in length, and long filamentous forms occur which later break up into shorter rods. The ends of the organism are rounded. It occurs singly or in short chains which are sluggishly motile by means of peritrichic flagella. The spores found in the organism are two or three times the diameter of the cell and are situated terminally, giving a "drumstick" appearance. The organism stains readily by the ordinary aniline dyes and is Gram-positive.[16] It is an obligate anerobe.

INCIDENCE AND OCCURRENCE

Tetanus occurs in all parts of the world and is most common on closely settled areas under intensive cultivation. It occurs in all farm animals mainly as individual, sporadic cases, although outbreaks are occasionally observed in young pigs and lambs. The mortality rate is usually about eighty per cent.[3]

It is more common in the extreme south than in the north; and is more liable to occur during the warm months than the cold.

It is seen in small animals, but it is reported that the dog, in proportion to weight, is 300 times as resistant to tetanus toxin as is the horse.[19]

Tetanus has been described as occuring in the turkey.[8]

Tetanus has been reported in horses and mules following puncture wounds of the limbs, castrations and umbilical infection (colts). There is a great deal of variation in susceptibility between animal species, the horse being the most susceptible and cattle the least. The organism exists normally in the intestinal tract of horses and is found in large quantities is equine feces.

It has been reported in pigs following castration and infection through the gums

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or umbilicus; in cattle following parturition, dehornings, the use of hypodermic needles and elastration procedures; in sheep following shearing wounds, docking, by self fecal contamination of a necrotic area due to docking[15] and from umbilical infections (lambs).

Tetanus has been reported in dogs and cats following ovariohysterectomy, harderian gland removal, and various injuries to the limbs and body.

PATHOGENESIS

The organism gains entrance through skin wounds caused by objects which carry it into the deeper tissues. Some cases of tetanus occur in which the atrium of infection is unknown. In the past, idiopathic causes were given quite a prominent position in the disease. As time progressed, however, it became accepted that some trauma was necessary to cause tetanus. In 1963 idiopathic tetanus was diagnosed in calves in England.[22] It was concluded that prior to the appearance of clinical signs, conditions in the digestive tract became favorable for the multiplication of *Clost. tetani* which resulted in sufficient production and absorption of toxins to be pathologically significant.[22]

After the spores are introduced into the anaerobic, traumatized area, a potent exotoxin is elaborated. There is controversy as to how the toxin gets to the target tissue. Originally it was thought to be spread by the blood stream. The incubation period required for the appearance of symptoms after injection of toxin, however, made toxemia doubtful.

The tetanus bacilli remain localized at the site of introduction and do not invade surrounding tissues. They start to proliferate and produce neurotoxin only if certain environmental conditions are attained, particularly a lowering of the local tissue oxygen tension. This may occur immediately after sufficient trauma has occurred, or may be delayed for several months until subsequent trauma to the site causes tissue damage. The original injury may have completely healed by this time giving rise to the idiopathic theory of cause.

The neurotoxin produced by the vegetative forms of *Clost. tetani* is absorbed through the motor end-plates of the muscles and apparently passes along the motor nerves to motor cells of the ventral horn of the spinal cord before general signs of the disease appear. A central and peripheral effect has been noted. The peripheral effect results from absorption of the toxin by the motor end-plates. The central effect arises from poisoning of the motor centers.

The toxin is absorbed by the motor nerve endings and passes up the axis cylinders to the anterior horn cells—which explains why contractions are confined first to the affected limb or part. Irritation of the motor nerves to the skeletal muscles causes the characteristic tonic muscular spasms. The exact means by which the toxin exerts its effects on nervous tissue is not known. Structural lesions are not produced, but there is central potentiation of normal sensory stimuli so that a state of constant muscular spasticity is produced and normally innocuous stimuli cause exaggerated responses. Death occurs by asphyxiation due to fixation of the muscles of respiration.[1, 7]

CLINICAL SIGNS AND DIAGNOSIS

The incubation period varies between one and three weeks with occasional cases occurring as long as several months. In the ovine, cases appear three to ten days after shearing or docking. The clinical picture is similar in all animal species.

A general increase in muscle stiffness is first observed and is accompanied by muscle tremor. There is localized stiffness usually involving the muscles of the jaw, the hindlimbs, and rarely, the muscles in the region of the infection. There is trismus, prolapse of the third eyelid and an unsteady, straddling gait with the tail held out stiffly, especially when backing or turning. The prolapse of the third eyelid is one of the earliest signs and can be exaggerated by sharp lifting of the muzzle or tapping the face below the eye.

After 24 hours the signs become generalized and pronounced, with hyperesthesia and tonic spasms. There is an anxious and alert expression. It is possible to elicit a spasm or convulsion merely by clapping the hands. 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 through the nose.

Constipation is usual and the urine is retained due to an inability to assume the normal position for urination.

The temperature and pulse rate are within the normal range in the early stages but may rise later. Bloat is an early sign in younger cattle but is usually not severe and is accompanied by strong, frequent rumen contractions.

As the disease progresses, muscular tetany increases, and the animal develops a 'saw-horse' posture. Consciousness apparently is not affected; sweating, disturbance of circulation and respiration, increased heart action, rapid breathing and congestion of the mucous membranes are common. There is great difficulty in walking, and the animal is inclined to fall with the limbs still in a state of tetany. Uneven muscular contractions may cause the development of a curve in the spine and deviation of the tail to one side.

The convulsions are at first only stimulated by sound or touch but soon occur spontaneously.

The temperature may rise as high as 108° F to 110° F toward the end of a fatal attack. 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. Mortality is eighty per cent.

The course of the disease varies both between and within species. The duration of a fatal illness in horses and cattle is usually five to ten days, but sheep usually die about the third or fourth day. Although tetanus is usually fatal a long incubation period is usually associated with a mild syndrome, a long course and a favable prognosis. Mild cases which recover usually do so slowly; the stiffness disappears gradually over a period of weeks or even months. A convalescent period of three to six weeks is required in horses.

Fully developed tetanus is so distinctive clinically that it is seldom confused with other diseases. In its early stages however, there may be some confusion with other conditions such as strychnine poisoning, eclampsia, acute laminitis or cerebrospinal meningitis to mention a few possibilities.

Strychnine poisoning is uncommon in farm animals, usually affects a number at one time or results from overdosing—history would be helpful. Tetany between convulsive episodes is not so marked.

Hypocalcemic tetany (eclampsia) of mares is confined to lactating mares and responds to treatment with calcium salts.

With acute laminitis there is not tetany nor prolapse of the third eyelid.

Cerebrospinal meningitis causes rigidity, particularly of the neck, and hyperesthesia, but the general effect is one of depression and immobility rather than excitement and hypersensitivity to external stimuli.

Lactation tetany of cattle and whole milk tetany of calves are accompanied by convulsions, but these are more severe than those seen in tetanus and prolapse of the third eyelid and bloat are absent.

Enzootic muscular dystrophy may be mistaken for tetanus because of the marked stiffness but there is an absence of tetany.

Other more marked nervous symptoms make differentation from enterotoxemia easy.

TREATMENT

There has been much work to find an effective cure for tetanus. It was stated in 1959 that regardless of method of treatment adopted, good nursing is essential for a successful outcome. This has particular application to the horse.[20]

Four severe cases of tetanus in humans were treated by complete paralysis of the patient with curare and positive pressure respiration. Three completely recovered.[10] Succinylcholine has been used successfully in a similar manner. Antibiotics are used in a supporting role.[23]

Treatment of tetanus in the horse, once the disease is fully established, has usually not been successful. Keeping the animal in a darkened, quiet stall, plus the use of sedatives, has been the most successful rationale of treatment until the middle 1950's.

Chlorpromazine hydrochloride (approximately 0.2 mg./lb.) was administered intravenously twice daily, to a horse with tetanus. Enough relaxation followed each injection to allow the animal to eat and drink. Previously, heavy sedation with chloral hydrate or other hypnotics had been used as a part of the treatment of tetanus. However, these drugs have the disadvantage of lowering the body temperature and interfering with the animal's ability to eat and drink. Passing a stomach tube would induce spasms. This can be overcome with the use of chlorpromazine.

The portal of entry of the organism and a method of controlling its multiplication and production of toxin has to be considered along with the somewhat symptomatic treatment that has been discussed above.

Necrotic tissue should be removed surgically, if practical, and drainage established in the wound area. Local injection of some of the antitoxin around the wound area is advised. Parenteral administration of penicillin or a broad spectrum antibiotic in maximum doses is indicated. If the disease is seen in its early stage, tetanus antitoxin may be administered at a level of 100,000 to 200,000 units or more.

Affected animals should be kept as quiet as possible and should be provided with dark, well-bedded quarters with plenty of room to avoid injury if convulsions occur. The uses of barbiturates, curare and general anesthetics seem to lead to more expense than would be practical.

PROPHYLAXIS

This is the most important area in the control of this disease, and one that the layman should be as aware of as the veterinarian.

Many cases of tetanus could be avoided by proper skin and instrument disinfection at castrating, docking and shearing time. Lambs are usually given 100 to 150 units after docking when the incidence of tetanus of the premises is high. Ewes satisfactorily immunized can transmit to their lambs specific antibodies via colostrum in amounts assuring their full protection against infection early in life.

Doses of 1,500 to 3,000 units of antitoxin are injected IM in horses, the dose varying with the extent and duration of the injury. Horses contracting tetanus when antitoxin is administered promptly are unheard of. The antitoxin will provide complete protection for a period of several weeks.

In 1932 Glenney *et al,* recommended tetanus toxoid for the protection of horses. In 1940 Gibbons advised tetanus toxoid immunization of all colts suffering from navel-ill. He also advocated yearly vaccination of horses with the tetanus toxoid.

Further tests were run to determine the validity of immunization of foals through colostrum. Active immunization was obtained for several months in all cases except one in which the foal was denied the colostrum for 36 hours due to a history of hemolytic disease in earlier foals.

Active immunization of the mares late in pregnancy was the preferred method of prophylaxis, and would be a good method to be used as the initial stage in providing lifelong protection against tetanus. Lemetayer, *et al,* in the proceedings of the International Veterinary Congress in Stockholm in 1953, showed that foals five or six weeks old can be successfully vaccinated using the alum precipitated vaccine.

Active immunization is accomplished through the use of tetanus toxoid. Two doses should be given at six-week intervals followed by booster injections annually. If wound infections occur in vaccinated animals, it is wise to give a dose of tetanus antitoxin. Antitoxin does not interfere with the production of antibodies by toxoid so that both can be administered at one time, the antitoxin providing short term passive immunity until an active immune status is attained.

SUMMARY

Tetanus is a disease caused by the *Clostridium tetani* organism. It is seen in all species of animals, being most prevalent in the horse, and it is seen in all areas, warmer climates and highly cultivated areas predominating.

A neurotoxin produced under anaerobic conditions, provided by trauma or devitalized tissues, is responsible for the common

symptoms of trismus, a prolapsed third eyelid, tetanic convulsions, a saw-horse posture, hyperesthesia and death due to respiratory collapse.

Treatment of tetanus is directed toward prevention of further absorption of the toxin by injecting antitoxin, giving appropriate attention to the wound, and administering antibiotics. Production of muscle relaxation and control of spasms by various drugs, the best of which is chlorpromazine, is important. Supportive measures should include all the details of good nursing. Semi-solid or liquid foods, glucose, saline and electrolytes are useful supportive measures, also.

Prevention should be accomplished by a combination of sanitary practices and active immunization with tetanus toxoid. Tetanus antitoxin should be administered in the event of any traumatic occurence that could lead to tetanus, and booster shots should be given annually to best prevent the occurence of this disease.

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