A Review On Equine Tetanus

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Abstract: Tetanus or lockjaw is one of acute fatal disease of equine with a high mortality rate of 50-80%. It is caused by the gram-positive, spore forming, motile, anaerobic bacteria, *C.tetani*, which is characterized by increased muscle tone, prolapsed third eye lid, spasm of facial masticator muscles, erect ears, exaggerated response to external stimuli, dilation of nostrils, the tail became stiff and extended, convulsion and spastic paralysis. *C.tetani* grows in different media only in the absence of oxygen and can produce neurotoxins that block the release of neurotransmitters for inhibitory synapses to develop spastic paralysis. Horses are very susceptive spp of the animal to tetanus. Tetanus occurs either in individual horse or as an outbreak in foals following injection and foaling. The disease has to be diagnosed on the basis of clinical sign often with the history of puncture wound or other trauma contaminated with soil or feces and based on microscopic examination, culturing, serology, laboratory animal inoculation. No necropsy finding as well as definitive ante mortem test or post mortem lesions, but we may demonstrate from necrotized tissues. The treatment of equine tetanus includes eliminating of the infectious agent, providing muscular relaxations, neutralizing the unbound toxin, and other supportive treatments like maintaining hydration, nutritional status of the horse and ensuring good footing. Active and passive immunization should be taken as control and prevention measures. This paper reviews on the general features, diagnostic approaches, and treatment, prevention and control measures of tetanus in equines.
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1. Introduction

Tetanus was first described in Egypt over 3000 years ago and was prevalent throughout the ancient world. Despite the availability of passive immunization since 1893 and an effective active vaccination since 1923, tetanus remains a major health problem in the developing world and is still encountered in the developed world (DeSouza *et al*., 1992).

Tetanus, or lockjaw, is an often fatal disease caused by *C.tetani* (DeSouza *et al*., 1992). Equine tetanus is not a contagious disease but can be the result of the *C. tetani* toxins entering the horse's body via puncture wounds or penetrating nail wounds of the foot, perinatal umbilical infections (Hirsh *et al.,* 2004), open lacerations, surgical incisions or exposed tissues in unvaccinated horses (TAAEP, 2008). *C.tetani* is gram positive, spore forming, obligatory anaerobic bacteria, that forms terminal, bulging, spherical shape of spores (Andrews *et al.*, 2004), that have the appearance of a 'tennis-racket' or 'drumstick' (Acha and Szyfres, 2003). The spores are extremely resistant to environmental conditions (Andrews *et al*, 2004) in soil for many years (Lefevre *et al*., 2010). *C.tetani* is commonly present in the feces of animals and in the soil contaminated by the feces (Smith, 2002).

Tetanolysin and tetanospasmin toxins, which are produced by *C.tetani* can inhibit the production of the inhibitory neurotransmitter (Quinn *et al.,* 2002) and results in increased muscle tone, prolapse of third eye lid, exaggerated response to external stimuli (Radostits *et al.*, 2007**)**, dilation of nostrils and the tail became stiff and extended (Quinn *et al.,* 2002).

Equine tetanus is distributed worldwide causing acute potentially fatal disease (Acha and Szyfres, 2003), which affects many animal species including humans with varying degrees of susceptibility (Lefevre *et al*., 2010). All horses can be at risk for developing tetanus. The mortality rate can reach 50-80% (McAuliffe and Slovis, 2008). Equine tetanus has to be diagnosed based on the clinical signs and often with the history of puncture wounds contaminated with soil or feces (McAuliffe and Slovis), laboratorical tests (Lefevre *et al.*, 2010), microscopic examination with "drum stick" forms of *C.tetani* (Quinn and Markey, 2003), Culturing and inoculating the bacteria in laboratory animals (Quinn *et al.*, 2003).

Treatment of the disease also focus on elimination of the agent (Hirsh *et al.,* 2004),

neutralizing the unbounded toxin (Lefevre *et al.*, 2010), providing muscular relaxations (Smith, 2002; Quinn *et al.*, 2002) and Supportive treatments (Radostits *et al.,* 2007).

Treatment is based on the knowledge that the toxin gangliosides bond is irreversible (McAuliffe and Slovis, 2008). Horses should be placed in dark and quite environment with minimal stimulation (Quinn *et al.*, 2002).

Many cases of tetanus should be avoided by proper wound cleaning and dressing (Hirsh *et al.*, 2004), disinfection of the surgical and nail puncture wounds (Blood *et al*., 1983). If an unvaccinated animal has a wound, temporary prevention can be provided by an injection of TAT (Hadril, 2002). Previously immunized horses with tetanus prone resins should not be given antitoxin (Hirsh *et al.*, 2004).

Generally, the objectives of this paper are to review the general features of equine tetanus and to confer with the diagnosis, treatment, prevention and control measures briefly.

2. Equine Tetanus

Tetanus, or lockjaw, is an infectious and often fatal disease caused by the anaerobic bacteria *C. tetani* (DeSouza *et al*., 1992) that affects neuromuscular function of equines without inducing observable tissue damage (Quinn *et al.,* 2003). Once the toxins reach the central nervous system, they will inhibit the production of inhibitory neurotransmitters and the characteristic muscle spasms begin (Thomas and Smith, 2009).

Tetanus is not a contagious disease but can be the result of the *C. tetani* toxins entering the horse's body via puncture wounds, open lacerations, surgical incisions or exposed tissues in unvaccinated horses (TAAEP, 2008). The disease occurs in sporadic form, but epizootic peaks can be observed, especially in managemental practices involving saddling wounds and nail puncture wounds of hoof and where contaminated instruments have been used (Lefevre *et al*., 2010).

Tetanus in equine is very important because of its high case fatality and the very long convalescence in the survivors (Radostits *et al*,. 1994). The fatality rate of tetanus in horses varies widely between areas (Radostits *et al.,* 2007).

2.1. Etiology

C. tetani is a gram positive, spore forming, obligatory anaerobic rod shaped (Hirsh *et al.*, 2004), non-capsulated, fermentative, catalase negative, oxidase negative and require enriched media for growth (Quinn *et al.,* 2002). It is a motile bacillus, 2-2.5microns long by 0.3-0.5 microns in diameter (Acha and Szyfres, 2003).

It is a neurotoxic *Closteridial* species, which can cause 'sawhorse' appearance (Quinn *et al.,* 2003). Tetanus neurotoxin serotypes can be distinguished by their flagellar antigens. It is swarming and is hemolytic on blood agar due to the production of tetanolysin (Quinn *et al.,* 2002).

Table 1. Biochemical reactions of *C.tetani*

Clostridium spp	Egg yolk agar					Acid form						Additional characteristics
C.tetani	Lecithinase	Lipase	Hydrolysis of gelatin	Digestion of casein	Indoleproduction	Glucose	Lactose	Sucrose	Maltose	Urease	Milk digestion	Terminal, spherical endospores. round spores (Gupte *et al.,* 2002)
	-	-	+	-	V	-	-	-	-	-	+	

+=positive reaction -=negative reaction v=variable reaction. Source (Quinn *et al.* 1994).

C.tetani forms terminal, bulging, spherical shape (Andrews *et al.*, 2004), ovoid spores, that have the appearance of a "tennis racket" or drumstick (Acha and Szyfres, 2003). The spores are commonly present in the soil and can contaminate puncture wounds, crushing wounds, open lacerations, surgical incisions and the umbilici of foals. Spores can lie dormant in tissues after wound healing and produce toxins if the local oxygen level drops (Thomas and Smith, 2009). Upon gaining entrance in to the body, they produce a powerful neurotoxin that blocks neurotransmission, resulting in an opposed muscle contraction and spasm (Tetany) (Waldorf and Maryland, 2003).

The spores are extremely resistant to environmental conditions (Andrews *et al*, 2004) in soil for many years (Lefevre *et al*., 2010) and to many standard disinfection procedures,including steam heat at 100°c (212°f) for 20 minutes, but can be destroyed by heating at 115°c (239°f) for 20 minutes (Radostits *et al*., 2007) and also resist boiling up to 1.5 hours, but disinfect on by some halogen compounds (3% iodine) can be effective within several hours (Quinn *et al.,* 2002). A 'drumstick' or 'tennis-racket'appearance of *C.tetani* endospores (figure1 and 2*)*.

2.1.1. C. tetani toxins

C.tetani produces several toxins an oxygen labile neurotoxin (tetanolysin) and a plasmid-encoded, heat labile neurotoxin (tetanospasmin) (Murray *et al.,* 2002). The two toxins are antigenically uniform irrespective of serotype and antibodies induced by the

neurotoxin of any of the serotypes neutralize the neurotoxins produced by others (Quinn *et al.,* 2002). The action of the toxin is to inhibit the production of the inhibitory neurotransmitters GABA and Glycine at CNS interneuron.

Toxin binding is irreversible (McAuliffe and Slovis, 2008). Much of the progression and outcome of this disease depends on how much toxin makes it to the spinal cord (Thomas and Smith, 2009).

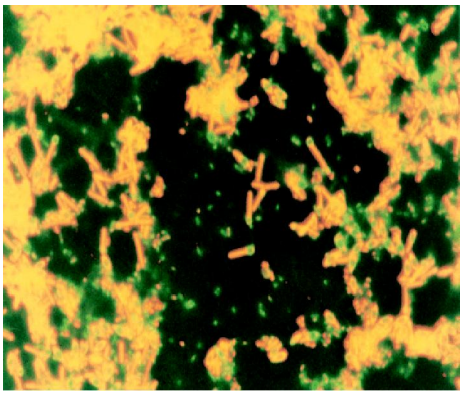

Figure 1. Acridine orange stains of characteristic *C.tetani* (De Souza *et al*., 1992).

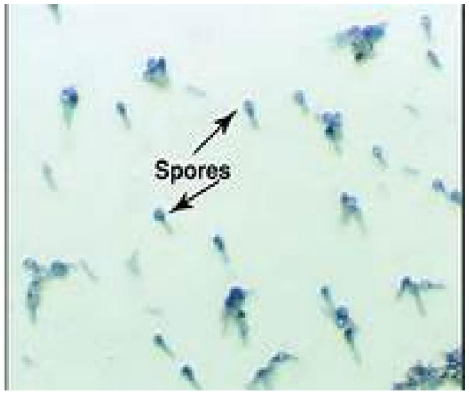

Figure 2. Sporing rods of *C.tetani* in gram stained smear of necrotic materials (Quinn *et al.,* 2002)

Tetanospasmin is produced by *C.tetani* growing under anaerobic conditions (Radostits *et al.*, 2007), during the stationary period of growth of the bacterium. The toxin is produced inside the cell in the form of peptide molecule which as a result of the action of the protease is transformed in to a more active molecule consisting of two linked sub units (Lefevre *et al.*, 2010).

Table 2. General feature of neurotoxin

Closteridial spp	Site of production	Genes which regulate production	Antigenic type	Mode of action	Clinical effect
c.tetani	In wound	In plasmid	One antigenic	Synaptic inhibition	Muscular spasm

Source (Quinn *et al*. 2002).

The toxin is a very potent toxin which, is released when the cell is lysed. It is estimated that less than 2.5ng/kg of body weight would be fatal for man and that 0.3-1ng/kg of body weight would be fatal for equines (Acha and Szyfres, 2003).

Tetanolysin is cholesterol binding cytolysin, which the cholesterol rafts in the eukaryotic cell membrane. Once bound it forms a pore resulting in the death of the cell (Hirsh *et al.*, 2004). It is inhibited by oxygen and serum cholesterol (Murray *et al.,* 2002). It is active against RBC of animals. Its pathogenic role in the production of tetanus may be as leucotoxin (Gupta, 2002). Its necrotizing activity can favor the multiplication of *C.tetani* within the tissues (Lefevre *et al.,* 2010). Non-spasmogenic is also the third toxin which is a peripherally active neurotoxin (Gupta, 2002).

2.1.2. Growth characteristics of the organism

C.tetani is an obligatory anaerobic organism that grows only in the absence of oxygen. The optimum temperature is 37^{o}c and PH 7.4. It grows fairly well in ordinary media (Abilo and Meseret, 2006). The organism cannot grow in normal tissue, but flourishes in deep penetrating wounds and occasionally in superficial wounds (McAuliffe and Slovis, 2008). Its differential reactions (carbohydrate fermentation, proteolysis, endole production) vary with the medium used (Quinn *et al.*, 2003).

Cultures for the growth of organisms include; cooked media *C.tetani* grows well with turbidity and gas formation on this media. It produces swarming growth in nutrient agar medium and foaming fine film over the medium. By increasing the concentration of agar in the medium after 2-4 days incubation colonies are irregularly round 2-5mm in diameter, transluscent greyish yellow with granular surface and ill defined edges (Gupta, 2002). The culture meat is not digested but turned black after prolonged incubation (Gupta, 2002). A zone of α heamolysis is produced in blood agar medium. It later on develops in to β hemolysis

due to production of hemolycin (tetanolysin) (Quinn *et al.,* 2002).

2.2. Epidemiology

2.2.1. Geographic occurrence

Tetanus occurs worldwide, but is more frequently encountered in warmer climates with soil rich in organic matter as in Africa, south Asia and America (Lefevre *et al.,* 2010). The disease occurs more frequently in tropics than in temperate or cold climates (Acha and Szyfres, 2003). Tetanus is relatively rare in the United States, because of high incidence of immunity brought by vaccination (Murray *et al.*, 2002).

2.2.2. Host occurrence

Tetanus causes acute potentially fatal intoxication which affects many species including humans (Radostits *et al.,* 2007). Horses and humans are highly susceptible, ruminants are moderately susceptible, carnivores and pigs are less susceptible than equines and ruminants while cats, birds and cold blooded animals seem to be particularly resistant (Quinn *et al.,* 2002). Foals are more sensitive than adults, probably due to the progressive development of natural immunity in older equines (Lefevre *et al.*, 2010).

Table 3. Report on occurrence of equine tetanus in Ethiopia (2001-2004).

Year	Place/site	Spp
2001	Diredawa	Equine
	Sebeta	Donkey
2002	Around Addis Ababa	Equine
	Hosaena	Equine
	Diredawa	Horse
2003	Diredawa	Horse
	Hawasa	Equine
	Addis Ababa	Horse
	Bahier Dar	Horse
	Addis Ababa	Donkey
2004	Nazereth	Horse
	Around Modjo	Horse

Sources: (Naod, 2005).

2.2.3. Source of Infection

C.tetani is commonly present in the feces of animals and in the soil contaminated by the feces (Smith, 2002). Normally the organism has two main habitants namely the soil and intestinal tracts of horses and humans (Andrews *et al.,* 2004). Surveys in different areas of the world show that it is present in 30-42% of soil samples. The survival periods of the organism in the soil vary from soil to soil (Radostits *et al.,* 2007).

C.tetani can be found in dirt that had no documented contact with equine. This indicates that the agent should be considered a primary soil contaminant (smith, 2002). Spores can survive adverse weather condition in the absence of direct sun light for months to years and can be found readily in dust and debris (Greene, 2006).

Areas where the exposure risk is high are referred to as ‘’telluric foci of *C.tetani* (Acha and szyfres, 2003).Contaminated soil with equines’ feces commonly contains *C.tetani* spores. Therefore, humans working around horses and horse farms should seek immediate medical attention when injured, especially with penetrating wounds such as those caused by nails (Waldorf and Maryland, 2003).

Wound contamination typically leads to infection, as a well-cleaned wound is not likely to result in tetanus and rather it is usually a wound that contains foreign matter such as soil (Thomas and Smith, 2009).

2.2.4. Mode of transmissions

Occurrence of tetanus is linked to the introduction of *C.tetani* into traumatized tissue, penetrating nail wounds of the foot, saddling wounds, barnyard surgery, ear tagging, injections, shearing wounds, postpartum uterine infections, perinatal umbilical infections (Hirsh *et al.,* 2004). Foal tetanus occurs when there is infection in the umbilical cord with insanitary conditions at parturition in mares (Radostits *et al.,* 1994; Scanlan, 1988). Nail puncture wounds of the hoof and traumatic skin wounds are most often the origins of spore introduction and infection in equines (Lefevre *et al.,* 2010).

The toxin subsequently crosses the intestinal wall and is transported via the blood stream to the motor neuron junctions (Lefevre *et al.*, 2010). Out breaks of idiopathic tetanus occur associated with the grazing of rough, fibrous feed and toxin is produced in wounds in the mouth or intestine or in ingested preformed toxin in the feed (Radostits *et al.,* 2007).

2.3. Pathogenesis

The tetanus bacilli remain localized at their site of introduction and do not invade surrounding tissues. They proliferate in anaerobic condition and produce toxins (Radostits *et al.*, 2007). The toxin can be distributed via the lymph and blood stream to reach first the motor neuron trunks in the muscles of the head and neck before expanding to other muscles in the lower body causing’’ descending tetanus’’ (Lefevre *et al.,* 2010). But in others toxin reach in to local peripheral motor neuron trunk at neuromuscular junction and then join CNS which is called as “ascending tetanus’’ (Lefevre *et al.,* 2010).

Tetanospasmin toxin acts by blocking the release of neurotransmitter (example, GABA, Glycine) for inhibitory synapses thus causing excitatory synaptic activity to be un regulated (spastic paralysis) (Songer and Post, 2005). The neurotoxin binds irreversibly to

ganglioside receptors on motor neuron terminals (Murray *et al.,* 2002) and is transported to the cell body and its dendritic processes in the CNS in toxin containing vesicles by retrograde intra-axonal flow (Quinn *et al.,* 2003).

Bound toxin is not neutralized by antitoxin (Quinn *et al.,* 1994). So recovery depends on the production of new axons (Murray *et al.,* 2002). Toxin is transferred trans-synoptically to its site of action in the terminals of inhibitory neuron (Songer and post, 2005). In ascending and descending tetanus cases the effect of the toxin on the CNS is to prevent inhibition of muscular contraction, resulting in continuous tetanic contraction of muscle groups (Pugh, 2005).

Toxin can hydrolyze the synaptobrevins, protein components of the vesicles containing neurotransmitters (Quinn *et al.*, 2002). A heavy chain of toxin is responsible for receptor binding and internalization of the toxin (Quinn *et al.*, 2003). The resistant to clostridial toxins might be attributed to difference in the membrane binding, intracellular trafficking of the toxins or in the susceptibility of the synaptic target to proteolytic attack (Holst, 2000). After the neurotoxins inhibit the production of inhibitory neurotransmitters, spasm of the muscle will be resulted (Thomas and Smith, 2009) and death is caused by paralysis of the breathing muscles, asphyxiation and heart failure (Lefevre *et al.*, 2010).

2.4. Clinical Signs

The incubation period of tetanus is variable and depends on localization of the portal entry of bacteria, the number of bacteria introduced and the quality of toxin produced at the point of inoculation (Lefevre *et al.,* 2010). It is usually between 5 and 10 days but may extend to three weeks (Quinn *et al.,* 2002), approximately 8 days (range 3 to 21 days) (Waldorf and Maryland, 2003), 1-3 weeks (Mcauliffe and Slovis, 2008). Wounds on or near to the head are usually associated with a shorter IP and an increased tendency to generalized tetanus (Quinn *et al.,* 2002).

The disease is characterized by increased muscle tone and especially prolapse of third eye lid; spasm of facial masticator muscles, erect ears and pointed backwards (Jana and Ghosh, 2011), exaggerated response to external stimuli (Radostits *et al.*, 2007**)**, dilation of nostrils and the tail became stiff and extended (Quinn *et al.,* 2002). Spasms of the muscles of the neck and head and the stiffness of the legs causes an extended posture of the animal often described as "a saw horse stance" (Lefevre *et al.,* 2010). The muscles of the lips are pulled back like the horse is smiling, showing his teeth (Thomas and Smith, 2009). Equines show a frightened expression and become stiffer. They stand with their legs spread out and cannot chew so strands of saliva may hang from the mouth, Stiff gait and choppy stride, reluctance to move, inability to open the mouth to eat or drink, eyes wide open, ears erect and pointed backwards.

The name "lockjaw'' or " trismus*"* describes the muscular contraction that rigidly closes or locks the jaws together (Jana and Ghosh, 2011). Constipation is usual and the urine is retained partly as a result of inability to assume the normal position to urination. While the rectal temperature and pulse rate are within the normal range in the early stage, but may rise later when muscular tone and activity are further increased (Radostits *et al.*, 1994). Finally sweating may be profuse and the temperature rises often 42^{o}c (107^{o}f) (Radostits *et al.*, 2007**).**

Most cases die in 3-10 days (Hadrill, 2002). As the disease progresses the animal shows difficulty in rising, moving, breathing and in later stage falling to the ground, the skeletal muscle become hard and contracted, convulsion may occur and death is caused by paralysis of the breathing muscles, asphyxiation and heart failure (Lefevre *et al.*, 2010).

Horses which recover from tetanus are not necessarily immune because the amount of toxin which can induce clinical disease is usually below the threshold required to stimulate the production of neutralizing antibodies (Quinn *et al.*, 2003).

2.5. Diagnosis

Due to the extreme sensitivity of equids to tetanus, the disease has to be diagnosed on the basis of clinical sign (Pugh, 2005) often with the history of puncture wound contaminated with soil or feces (McAuliffe and Slovis, 2008). The diagnosis is a clinical one, relatively easy to make in areas where tetanus is often seen, but often delayed in the developed world where cases are seen infrequently (DeSouza *et al.,* 1992). Sometimes isolation of *C.tetani* from infected equines is often very difficult, because the organism is not invasive and the amount of toxin needed to produce clinical tetanus is so small that there may be no apparent infected wound site to sample (Scanlan, 1988).

2.5.1. Clinical and laboratorial diagnosis

History of a wound and the absence of active immunization may help to orient clinical diagnosis (Lefevre *et al.*, 2010). It is not always possible to isolate the etiologic agent from a wound (Acha and Szyfres, 2003).

Gram stained smears may demonstrate gram-positive rods and dark staining spherical endospores, the morphology with a "drum stick" or "tennis racket" appearance (Quinn *et al.*, 2002). Diagnosis by culturing the organism is more dependable. An excised bit of tissue from necrotic depth of wound is inoculated into cooked meat broth, blood agar and lactose egg yolk medium (Gupte, 2002). It is characterized by "swarming colonies" and sporulating

bacterial cells with a "drumstick" or "tennis- racket'' appearance (i.e. bacilli with round terminal spores) (Bartelt, 2000; Hirsh and Zee, 1999).

Laboratory animal inoculation is also the other way of diagnostic method. Mouse is a suitable laboratory animal for demonstration of toxigenecity. Serum from affected equines may be used to demonstrate circulating neurotoxin, using mouse inoculation (Quinn *et al.,* 2002). From 2 to 4 days of old cooked meat culture (0.2ml) is inoculated into the root of tail of a mouse (Quinn *et al.*, 2003; Hirsh and Zee, 1999). A second mouse which has received tetanus antitoxin (1000IU an hour earlier serve as control. Symptoms appear in test animal in 12 to 24 hours with stiffness of tail. Rigidity develops in the inoculated side of the leg, opposite to leg, trunk, fore limb in this order. The animals die within 2 days (Gupta, 2002; Bisping and Amtseberg, 1988).

2.5.2. Necropsy findings and clinical pathology

There are no structural changes in the nervous system (Radostitis *et al.,* 2007) as well as no gross and histological findings (Saxena *et al.*, 1998). There are no specific abnormalities in blood or CSF and no ante mortem test of value in confirming the diagnosis. Blood level of tetanus toxin are usually too low to be detected (Radostitis *et al.,* 2007).

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 (Radostitis *et al.,* 1994).

2.5.3. Differential diagnosis

Because of the distinct clinical sign classical tetanus is seldom confused with other diseases (Andrews *et al.*, 2003).

Botulism affects the nervous system of the body and cause paralysis like tetanus but it causes flaccid paralysis. Hypocalcaemic tetany (eclampsia) also resembles tetanus, but it is confined to lactating mares and responds to treatment with calcium salt (Radostits *et al.*, 1994). Strychnine poisoning could affect the function of nervous system. But it lasts for only a few hours (Andrews *et al.*, 2003). Acute lamnitis resembles tetanus; but there is no tetany or prolapsed of third eye lid (Blood *et al.,* 1983). Cerebrospinal meningitis cause rigidity particularly of the neck and hyperesthesia to touch but the general effects are one of the depression and immobility rather than excitement and hypersensitivity to sound and movement (Radostits *et al.,* 1994). Postmortem investigation and clinical chemistry help to rule out the diseases (Lefevre *et al.*, 2010).

2.6. Treatment

The treatment is primarily symptomatic and supportive (McAuliffe and Slovis, 2008). Therapy aims at neutralizing of toxins, suppression of toxin production, life support and symptomatic relief to the patient (Quinn *et al.*, 2002) and control of muscle spasm (McAuliffe an d Slovis, 2008). Treatment is based on the knowledge that the toxin gangliosides bond is irreversible (McAuliffe and Slovis, 2008).

2.6.1. Elimination of the infectious agent

Penicillin remains the standard therapy for tetanus in most parts of the world. Parenteral administration of penicillin in large dose is aimed at stopping toxin production as well as illumination of the organism (Hirsh *et al.,* 2004). Penicillin G 20,000-40,000 IU/kg of 12 hours or 2 times daily should be administered IV or IM (Quinn *et al.,* 2002; Smith, 2002). Penicillin does not readily cross the blood-brain barrier, but in high cumulative doses it can cause CNS hyper excitability. In tetanus this side effect of penicillin could synergize with the action of the toxin in blocking transmitter release at GABA neurons (Blood *et al.,* 1983). Metronidazole is a safe alternative, and may now be considered as the first line therapy, that is aimed at stopping toxin production (Hirsh *et al.,* 2004).

The infection is best illuminated additionally by surgical debridement and cleaning of the infected areas but only after antitoxin has been administered because debridement and irrigation with hydrogen per oxide and local penicillin may facilitate the absorption (release in to circulation) of toxin (Radostits *et al.*, 2007). Flushing with hydrogen peroxide produces aerobic condition which helps to inhibit bacterial replication at the site of injury (Lefevre *et al.,* 2010).

General anesthesia is usually required to debridement wounds and removes necrotic tissues and visible foreign materials. If wound site is located and irrigated well the recovery is always good (Greene, 2006).

2.6.2. Neutralizing the unbounded toxin

The immediate concern when treating tetanus is administering of adequate dose of antitoxin to neutralize circulating toxin that has not bound to CNS (Lefevre *et al.*, 2010). The timing and route of administration of antitoxin are important in determining the effectiveness of detoxification. Whereas elimination of bound toxin in affected animal is gradual; the recovery is usually slow and progressive (Radostits *et al.*, 2007).

TAT has little value after the onset of clinical sign (Radostits *et al.*, 1993). The dose that is given should be based on the history of the case and influenced by delay in treatment several hours after injury, lack of aggressive debridement several hours after injury and no history of vaccination (McAuliffe and Slovis, 2008). Injecting 10,000 to 300,000 IU of TAT for horses by intrathecal administration (Hirsh *et al,* 2004), IV or subarachnoid space is effective to

neutralize the toxin (Quinn *et al.,* 2004). Local injection of some of antitoxin around the wound is also advisable (Radostits *et al*., 2007).

2.6.3. Providing muscular relaxation

Drugs are administered that may reduce the muscular spasm (Smith, 2002). This includes; chlorpromazine (0.4-0.8mg/kg of body weight IV for 3 to 4 times daily) and acetylpromazine (0.05mg/kg of body weight) IV 2 times daily for 8-10 days administration until several signs subside are widely used (DeSouza *et al.,* 1992). Mephenesin (10-20mg/kg IV given 3 times daily) and Quaifenesin are also interfering with the interconnected neurons of the spinal cord that participate in reflex muscle activities, but do not have a high therapeutic efficacy for tetanus. A combination of Diazepam (0.01-0.4mg/kg IV 2-8 times daily) and Zylazine (0.5-1.0 mg/kg IV) effectively reduces muscular spasm in large animals by enhancing GABA, but prolonged administration of this drug to horses is expensive because of short duration in plasma and CNS (Lefevre *et al.,* 2010).

Packing or plugging the ears with cotton to minimize auditory stimulations also can help to reduce muscle spasm (Smith, 2002; Quinn *et al.*, 2002).

2.6.4. Supportive treatments

Maintaining hydration and nutritional status; tetanic equines frequently have difficulty in prehension and swallowing solid feeds but they can usually eat blended foods or fluids by sucking through clenched teeth (Greene, 2006). The food and water should be placed over the ground in an elevated feed bunk or huge net to allow easier access (Smith, 2002). Artificial feeding via nasogastric tube or intravenously after the hypersthetic phase (Hirsh *et al,* 2004) or in dysphagic horse, are also good way of supportive treatment, but frequent passage of nasogastric tube should be avoided to reduce stimuli (McAuliffe and Solvis, 2008).

Ensure good footing; horses that cannot stand should be supported in a sling, provided they do not become frantic while suspended (Smith, 2002). Affected equines should be kept in well bedded quarters with non slip floor and plenty of room to avoid injury if convulsions occur (Radostits *et al.,* 2007). Soft comfortable bedding should be provided (Greene, 2006).

Horses should be placed in dark and quite environment with minimal stimulation (Quinn *et al.*, 2002). Horses which are in lateral recumbency have little chance of recovery and euthanasia should be considered (McAuliffe and Solvis, 2008).

2.7. Prevention and Control

Vaccination is effective than treatment for tetanus, because treatment is probably of little value unless administered in the very early stages of the disease (Lefevre *et al.*, 2010).

Many cases of tetanus should be avoided by proper wound cleaning and dressing (Hirsh *et al.*, 2004), disinfection of the surgical, castrating, shearing, nail puncture wound or similar instruments when under taking any husbandry or surgical procedures and reduces the risk of contamination (Blood *et al.,* 1983). Cold sterilization of infected instruments used for surgical procedure should not be used (Greene, 2006).

If unvaccinated equines have a wound, temporary prevention can be provided by an injection of TAT (Hadril, 2002). The recommended dosage of tetanus antitoxin is 1500-3000 IU subcutaneous or IM for adult horses and foals from unvaccinated days should receive 1500 IU of TAT at birth (Blood *et al.,* 1983). Previously immunized horses with tetanus prone resins should not be given antitoxin, but actively unimmunized horses are given antitoxin after injury or surgery (Hirsh *et al.*, 2004).

In addition to TAT tetanus toxoid should also be administered subcutaneously to promote an active immune response or as a protective humurol immunity which is not induced by natural disease (McAuliffe and Solvis, 2008). The vaccines are prepared from formalin killed cells adsorbed on to ammonium hydroxide producing toxoid (Andrews *et al.*, 2003) or inactivated vaccines with djuvant toxoid and they induce long lasting immunity (Lefevre *et al.,* 2010).

The intramuscular injection of toxoid produces less local inflammation and an increased immune response. Reactions to absorbed toxoid in horses, which take the form of several local swelling, can be avoided by using a product containing minimal amount of aluminum hydroxide (Blood *et al.*, 1983).

In high risk flocks where equines are exposed to traumatic management (nail puncture of hoof, saddling and other trauma or surgical procedures, antitoxin and toxoid may be administered simultaneously to the equines. Antitoxin does not interfere with the production of antibodies by toxoid so that both can be administered at one time, but the two materials must not be mixed in one syringe, but in separate syringes on opposite side of the neck (Lefevre *et al.*, 2010). Antitoxin provides short term passive immunity or immediate protection which lasts for 2-3 weeks until an active immune status is attained (Lefevre *et al.*, 2010).

In some areas the incidence of tetanus in foals is high and repeated doses of antitoxin at weekly intervals is not always completely effective. Provided foals get an adequate supply of colostrums, they can be passively immunized during the first 10 weeks of life by active vaccination of the mare during the last weeks of pregnancy and the foals vaccinated at 3 months of age (Lefevre *et al.,* 2010).

Primary vaccination requires two doses of 3-6 weeks apart. A second dose course in the first year of life followed by annual booster is recommended. Additionally, booster dose may be recommended to individual animals (Pugh, 2005). Protective titers are obtained within 14 days of the second injection and lasts for at least a year up to 5 years (Radostitis *et al.*, 2007). One injection gives immunity in 10-14 days lasting for a year and revaccination in 12 month gives solid immunity for life (Blood *et al.,* 1983). A more vigorous program of two vaccines 6-8 weeks apart followed by annual booster vaccination is preferred. A transient phase of reduction of antibody titer occurs after this booster injection in horses and may be more susceptible at this time (Blood *et al.,* 1983).

3. Conclusion And Recommendations

Tetanus is highly important disease in developing countries with a high susceptibility of horses and human beings. Its worldwide distribution, highly resistant spore forming nature of the bacteria, bounded nature of the toxin and lack of practiced vaccination program makes it difficult to prevent and control equine tetanus. Equines play a great role in the source of economy especially for rural economy by performing major parts of agricultural activity. Hence the treatment, prevention and control of the disease are an important issue for the development of the country. Based on the above points the following recommendations are forwarded.

- Clinics and hospitals of equine should be established with full facilities, because tetanus requires long course of treatment and hospitalization.
- Equines should be vaccinated for tetanus annually or as recommended by veterinarian.
- Proper immunization and wound management should be conducted for tetanus case.
- Veterinarians should create awareness to the owners of equine about the side effect of the disease, good shoeing of the horse and use of comfortable saddles.
- All humans working with horses should ensure that their vaccination status for tetanus must be completed as recommended by Physicians.

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