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Clinical Communications

TETANUS IN THE EMERGENCY DEPARTMENT: A CURRENT REVIEW

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□ **Abstract—Despite the availability of effective immunization to prevent tetanus, there are still up to one million cases per year worldwide. Although the majority of tetanus cases occur in third world countries, there are still significant numbers of cases occurring in countries such as the United States, where preventive immunization is easily accessible. The Emergency Physician has the opportunity to contribute to the decline of the incidence of tetanus through knowledge of those at greatest risk for inadequate immunization and through providing proper wound care and immunization prophylaxis. © 2001 Elsevier Science Inc.**

□ **Keywords—tetanus; immunization; wound care**

INTRODUCTION

Tetanus is a devastating disease of muscle spasm and autonomic instability with a high mortality. Despite being easily preventable with a highly effective vaccine, tetanus remains a significant source of morbidity and mortality worldwide. In the United States, the incidence of tetanus has significantly decreased since the 1940s when immunization programs were enacted; however, the incidence has remained relatively stable for the last 10 years (1). Tetanus prevention is addressed daily in the Emergency Department (ED) as part of wound care, but acute tetanus is rarely seen. This can lead to a false sense of familiarity with this disease and foster misconceptions about its management and prevention. This article reviews the presentations of acute tetanus, current treatment, and prophylaxis considerations in the ED.

EPIDEMIOLOGY AND ETIOLOGY

The true incidence of tetanus is not known, but is estimated to be between 500,000 to one million cases per year worldwide (2,3). The majority of cases occur in developing countries, with 50% of those cases occurring in neonates. In contrast, tetanus is uncommon in developed countries, with most cases occurring in older adults (1,4,5). In the United States, there were 124 cases of tetanus reported between 1995 and 1997, with a male to female ratio of 3:2. The incidence was 36–48 cases per year, or 0.15 cases per million annually (1). This rate has remained relatively stable since the late 1980s.

An acute injury precedes most cases of tetanus beyond the neonatal period (1) (Table 1), the most common of which are puncture wounds and lacerations. Recent surgery also accounts for several cases. Nonacute etiologies include chronic wounds, i.v. drug use, and complications of diabetes. Case reports have implicated other less obvious etiologies, including otitis media; intranasal foreign bodies; corneal abrasions; ulcers; foreign bodies; dental procedures; injections; abortions; childbirth; and burns (6–10). In 6–8% of cases, there is no obvious etiology (1,7,11).

Lack of immunization is the greatest risk factor for contracting tetanus. Among victims of tetanus whose immunization histories were known, 72% never completed a primary immunization series (1) (Figure 1). The groups identified as having the lowest rates of immunization in the United States include older adults, Hispanics, African Americans, those with poverty income lev-

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Table 1. Etiologies of Tetanus*

Source	Incidence (%)
Acute Injury	77
Puncture	49
Laceration	22
Abrasion	12
Recent Surgery	3
Animal bite	
Misc	
Non acute	23
Chronic wound	
Diabetes	
IVDU	

* Data from CDC (1).
IVDU, intravenous drug user.

Table 2. Classifying the Severity of Tetanus*

Severity	Incubation‡ (days)	Onset† (days)	Associated Findings
Mild	≥10	4–7	Local rigidity, mild trismus
Moderate	7–10	3–6	Severe trismus, dysphagia, spasms
Severe	<7	<3	Severe spasms, diffuse rigidity, autonomic dysfunction

* Adapted from Bleck (2).
‡ Time from injury to first symptom.
† Time from first symptom to generalized spasm.

els, and those without military service (12,13). Of these risk groups, older age is the most significant. Serologic surveys show that only 28–50% of those over 65–70 years old are adequately immunized, compared to more than 80% of those 6–39 years old (12,14). This lack of immunity results in a higher incidence of tetanus in older adults. Adults over 60 years old have an incidence of 0.33 per million, compared to 0.17 per million among 20–59 year olds and 0.026 per million among 5–19 year olds (1).

Even with optimal treatment, the mortality of tetanus is very high. The global fatality rate is estimated to be 30–50% (15,16). In the United States, the overall fatality rate between 1995–1997 was 11% (1). The age-related death rate was 7.5% of those under 60 years old and 18% of those over 60 years old (1). No one who completed a primary immunization series died (Figure 1). The fatality rate also varies with the clinical forms of tetanus.

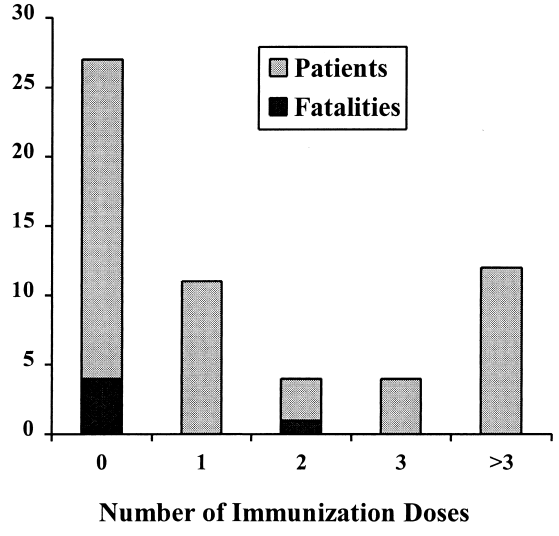

Figure 1. Vaccination history of tetanus patients. Data from CDC (1).

PATHOPHYSIOLOGY

Clostridium tetani is a spore-forming, Gram-positive bacillus. Although the bacillus is an obligate anaerobe, its spores remain viable at ambient oxygen concentrations. The spores are highly resistant to extremes in temperature and humidity and can survive indefinitely. Spores are ubiquitous in soil and in the feces of many animals and of humans (2,7). Carried into wounds along with soil, spores may not germinate immediately because of unfavorable tissue conditions. They may activate well after the wound has healed, which may account for the cases of tetanus that have no identifiable source. When conditions such as gross contamination or tissue injury favor anaerobic proliferation, the spores germinate into mature bacilli, which then form the toxins tetanolysin and tetanospasmin. Tetanolysin has an uncertain role in clinical tetanus; it may contribute to an anaerobic environment by damaging viable tissue (2).

Tetanospasmin is primarily responsible for the clinical manifestations of tetanus. It enters peripheral nerves and travels via the axonal retrograde transport system to the central nervous system (CNS). Tetanospasmin then enters presynaptic neurons and disables neurotransmitter release, most importantly, the inhibitory neurotransmitters gama-aminobutyuric acid (GABA) and glycine. This results in a disinhibition of end-organ neurons, such as motor neurons and those of the autonomic nervous system. This accounts for the muscle spasms characteristic of tetanus and for the autonomic instability seen with severe tetanus. The rapidity of incubation and onset correlates with the severity of disease (Table 2). Recovery involves synthesis of new presynaptic components and their transport to the distal axon (2). This accounts for the typical 2–3 week period before clinical improvement begins.

CLINICAL PRESENTATIONS OF TETANUS

There are four clinical forms of tetanus representing the extent and location of neurons involved: generalized, local, cephalic, and neonatal. In the United States and other developed countries, generalized tetanus is the most common form, occurring in 80% of cases (1). The initial symptom is trismus—"lockjaw"—secondary to masseter muscle spasm in 50–75% of cases (17). *Risus sardonicus*, the "ironical smile of tetanus," can occur because of facial muscle contraction. Nuchal rigidity and dysphagia also may be initial complaints. As the disease spreads, generalized muscle spasms occur, either spontaneously or to minor stimuli such as touch or noise. Opisthotonus, a tonic contraction very similar to decorticate posturing, is classically described with tetanus. Severe spasms can result in vertebral fractures, long bone fractures, and detachment of tendons from their insertions (9,10). Unfortunately for the patient, mental status is not affected, and spasms are experienced with severe pain (9).

In the acute phase, death results from acute respiratory failure caused by diaphragmatic paralysis or laryngeal spasms (2,18). With intensive medical intervention, including the use of paralysis and mechanical ventilation, such deaths can be averted. With patients surviving beyond the acute phase, autonomic instability becomes the major cause of death, with a fatality rate of 11–28% (16,19). Autonomic instability occurs several days after the onset of generalized spasms and manifests most importantly as labile hypertension, tachycardia, and pyrexia. Dysrhythmias and myocardial infarction are the most common fatal events (16). The exact mechanism of this syndrome is unclear, but likely involves disinhibition of the sympathetic nervous system. Case reports have documented elevated levels of catecholamines in patients with autonomic instability; levels fall as patients are successfully treated (20–23).

Local tetanus presents as persistent muscle rigidity close to the site of injury. The rigidity may linger for weeks to months and often resolves without sequelae (2). A caveat is that what appears to be localized tetanus may instead be the first symptom of generalized tetanus. The incidence is 13%, and the fatality rate is about 1% (1,24).

Cephalic tetanus is an uncommon variant of localized tetanus that involves the cranial nerves. It has an incidence of 6% (1). Cephalic tetanus uniquely results in nerve palsies as well as muscle spasms. The seventh cranial nerve is most often involved, followed by the 6th, 3rd, 4th, and 12th in decreasing order of frequency (25). Cephalic tetanus also presents with trismus, but in 42% of cases cranial nerve deficits precede the onset of trismus (25). In such cases, cephalic tetanus is easily misdiagnosed. With its predilection for the 7th cranial nerve, it commonly mimics Bell's Palsy. Head trauma and otitis media are commonly cited etiologies (24,26). About two thirds of the patients progress to generalized tetanus, and the overall mortality is 15–30% (25).

Neonatal tetanus is generalized tetanus that occurs in the newborn around the first week of life. Symptoms begin with nonspecific irritability and poor feeding and rapidly progress to generalized spasms. The portal of entry is the freshly cut umbilical cord. The risk of contracting neonatal tetanus is directly related to the cleanliness of delivery conditions and to maternal immunization because passive transfer of maternal immunoglobulins is protective (27). Mortality is very high, 50–100%, due to the high load of toxin per body weight in neonates (3,9,28). Neonatal tetanus largely occurs in developing countries, with an estimated 80% of cases concentrated in 12 developing countries in Africa and Asia (29). Programs to immunize pregnant women in these high incidence areas have significantly decreased the incidence and fatality rates of neonatal tetanus. The global fatality rate has decreased from 6.5 per 1000 live births (787,000 deaths) in 1988 to 2.1 per 1000 live births (277,376 deaths) in 1993 (3,30). In the United States and other developed countries, neonatal tetanus is very rare due to cleaner delivery conditions and a high rate of immunization among women of childbearing years. There were three reports of neonatal tetanus in the United States between 1991–1998, all involving inadequately immunized mothers (1,31).

DIAGNOSIS

The diagnosis of tetanus must be made on clinical grounds alone. There are no laboratory tests that can diagnose or rule-out tetanus. A "protective" serum antitoxin antibody level, commonly accepted as 0.01 U/mL (in vivo) or 0.15 U/mL (in vitro), makes the diagnosis of tetanus very unlikely, but not impossible. There are case reports of tetanus in immunocompetent individuals with "protective" antibody levels (32–34). Unfortunately, antitoxin antibody levels are not likely to be available at the time when management decisions must be made. Fortunately, the presentation of tetanus is so characteristic that a presumptive diagnosis can be made in most cases.

The differential diagnoses are few. Trismus can be caused by peritonsillar and odontogenic abscesses. These can be excluded by history and examination. Dystonic reactions can also present as trismus; a positive response to benztropine or diphenhydramine will quickly differentiate it from tetanus. Strychnine poisoning can closely resemble generalized tetanus. Strychnine disables glycine release like tetanospasmin, but does not affect GABA release. There were 40 cases (no deaths) of

strychnine poisoning in 1997, an incidence very similar to tetanus (35). In most cases, a timely strychnine level will not be available to the Emergency Physician, nevertheless, the test should be ordered for inpatient follow-up. Hypocalcemia causing tetany is another mimic, which can be easily excluded with laboratory testing. Other entities that cause diffuse muscle spasms, such as seizures, toxidromes, and encephalopathies, are accompanied by changes in mental status. Processes that affect muscles locally, such as myopathies or neuropathies, tend to cause weakness rather than spasm and rigidity. In addition, neuropathies are associated with sensory deficits, which is not a feature of tetanus. Cephalic tetanus without trismus can be easily mistaken for Bell's Palsy, CNS tumor, or stroke; with the inevitable appearance of trismus and muscle spasm, the diagnosis becomes clear. Similarly, neonatal tetanus initially presents much like a host of other disorders, including infectious, toxic, and metabolic etiologies. However, once generalized spasms begin, the diagnosis is obvious.

As an aid in clinical diagnosis, Apte and Karnad described a bedside "spatula test" for tetanus. A spatula is carefully inserted into the pharynx. If the patient gags and tries to expel the spatula, the test is negative for tetanus; if the patient bites the spatula because of reflex masseter spasm, the test is positive for tetanus. They reported 94% sensitivity and 100% specificity (11). The authors found the test most useful in the early phase of tetanus, before the onset of generalized spasms, when the diagnosis may not be obvious. The authors admitted the limitation that their results were achieved in an area with a high incidence of tetanus (India). In areas such as the United States where tetanus is less common and trismus has more common etiologies, the "spatula test" is not likely to achieve the same performance. Nevertheless, it is a potentially useful adjunct to diagnosis in the early stages of the disease. Obviously, it should be used with caution in patients who may not be able to protect the airway.

TREATMENT OF ACUTE TETANUS

Treatment involves neutralizing tetanospasmin, removing the source of the toxin, and providing supportive care for muscle spasms, respiration, and autonomic instability. Human tetanus immunoglobulin (HTIG) is given to neutralize circulating tetanospasmin. It cannot inactivate toxin already within neurons. Its half-life is 25 days; only a single dose is necessary (36). The optimal dose is not well defined. In a 1976 retrospective study, Blake et al. found that a dose of 500 IU intramuscular was as effective as the 3,000–10,000 IU doses that were commonly used (37). The smaller dose has the advantage of requiring fewer injections to deliver. This is important because HTIG is supplied in 250 IU doses, and injections are powerful stimuli for spasms. Authorities now consider 500 IU the preferred dose (2,15). The adult and pediatric doses are the same. The burden of tetanospasmin within the patient determines the amount of HTIG needed, not the patient's weight.

Intrathecal administration of immunoglobulin is theoretically attractive. By delivering immunoglobulin to the CNS, one hopes to bind the toxin as it crosses from postsynaptic to presynaptic neuron. Clinical trials have yielded conflicting results. A meta-analysis by Abrutyn and Berlin concluded that there was insufficient evidence to recommend intrathecal therapy (38). They noted that many of the studies were of poor design and cautioned against forming final conclusions regarding this therapy. They made special mention of a study by Vakil et al., the only randomized and blinded trial, that showed no benefit to intrathecal immunoglobulin (39). To date, there are too few well-designed studies to definitively support or refute intrathecal therapy. It should be noted that in the United States, HTIG is not approved for intrathecal use.

To prevent on-going production of toxin, antibiotics are given to eliminate *Clostridium tetani*. Metronidazole is the drug of choice (2,15,40). It is superior to penicillin, the former drug of choice, in terms of recovery time and mortality (40). Metronidazole penetrates vascularly compromised wounds and abscesses better than does penicillin. Furthermore, penicillin has GABA-antagonist activity, which may potentiate the effects of tetanospasmin. In addition to administering antibiotics, dirty wounds, abscesses, or devitalized tissue must be cleaned, drained, or excised to decrease the bacterial load.

Benzodiazepines are the drug of choice for muscle spasms because of their GABA-agonist and sedative properties. Very large doses may be necessary; there are case reports of 3,400 mg of diazepam and 1,440 mg of midazolam given over 24 h (22,41). When benzodiazepines have failed, other treatments that have been used include dantrolene, an agent that acts at the sarcoplasmic reticulum, and intrathecal baclofen, which is also a GABA-agonist (28,42–45). Topically applied mephenesine, a centrally acting muscle relaxant, was used in one case to control facial contractions (46). For severe cases, paralytics may be needed. Vecuronium is an ideal agent for immediate and long-term control because of its minimal cardiovascular effects (47). Pancuronium exerts cardiovascular effects that may confuse the management of autonomic instability.

Succinylcholine can be used in the initial phase of the disease, but not later because of the risk of hyperkalemia. During the course of tetanus and other denervating diseases, up-regulation of muscle acetylcholine receptors occurs. Using succinylcholine in this "supersensitive"

state causes an outpouring of potassium from hyperstimulated muscles (48). This effect does not occur for 4 days and peaks at 14 days after the onset of disease (49). Early reports of fatal hyperkalemia caused by succinylcholine involved its use several days into the course of disease (50). Succinylcholine may also cause hyperkalemia after recovery from tetanus because acetylcholine receptor up-regulation may persist for months.

When paralytics are used, mechanical ventilation is needed. Orotracheal intubation can be performed initially, but the presence of an endotracheal tube may precipitate or exacerbate laryngeal spasms in lightly paralyzed patients. To avoid this and to facilitate long-term ventilatory support, tracheostomy is often performed early.

Treatment of autonomic instability has been problematic and is the subject of on-going research. No therapeutic regimen has proven to be universally effective. Alpha- and beta-blockers are used, but have disadvantages. Unopposed alpha blockade can result in reflex worsening of tachycardia; unopposed beta blockade can result in worsening hypertension and an increased risk of sudden death (19,51). Labetalol is recommended as a balanced agent, but case reviews have shown poor results with this agent, possibly because of its predominate beta effects (2,19,20,52).

Agents that modulate sympathetic output also have been tried. Clonidine has yielded variable success (53,54). Magnesium, which blunts catecholamine release, also has given mixed results (40,53,55). One case report suggested that magnesium is ineffective alone, but efficacious when given with sedation (41). Morphine and fentanyl centrally decrease sympathetic outflow and generally produce good control of hypertension and tachycardia (19,22). They have the additional benefit of providing sedation. Fentanyl may be superior to morphine because it does not depress the myocardium nor does it induce histamine release. Finally, there are case reports of epidural anesthesia successfully controlling autonomic instability (23,56). This is believed to work by blocking preganglionic sympathetic neurons.

Experimental therapy includes the use of corticosteroids. Promising results have been achieved, but more work needs to be done to define the target patient, optimal dosing, and expected outcomes (57,58). The mechanism by which steroids might offer benefit is unknown. At this time, there are insufficient data to include steroids as standard therapy.

Supportive care includes placing the patient in a quiet, dark environment, minimizing patient manipulation, and treatment for complications, most significantly rhabdomyolysis. It is important that survivors also receive a tetanus immunization series. The amount of tetanospasmin produced in clinical tetanus is small and partially sequestered in neurons; consequently, an immune response does not occur. Nonimmunized survivors of tetanus have become victims a second time (59,60).

PREVENTION

Tetanus is preventable with proper use of tetanus toxoid and HTIG. Tetanus toxoid is an inactivated form of tetanospasmin. It is available in four forms, variably combined with diphtheria and pertussis vaccine: DTP, DT, Td, and single-antigen tetanus toxoid (TT). The combination vaccines are generally recommended because concurrent immunization is typically appropriate. Tetanus vaccination in the previously unimmunized confers protective antibody levels in 81–95% of people after two doses and in 100% of people after three doses (61). Protective antibodies develop rapidly after a booster dose of tetanus toxoid once primary immunization has been completed (27).

Common adverse reactions to tetanus toxoid include erythema, swelling, and tenderness at the injection site. These are minor and of no long-term consequence. Occasionally, a persistent nodule or, rarely, a sterile abscess forms at the injection site. Nonspecific systemic effects such as fever, malaise, and anorexia can also occur. These are more common with DTP than the other vaccine combinations because of the pertussis component. Reactions tend to occur more often and more severely if boosters are given more frequently than the recommended schedule. Patients who give a history of "allergy" to tetanus vaccine are most likely referring to a local or nonspecific systemic reaction. These are not contraindications to receiving tetanus toxoid. Other false contraindications include mild, acute illness; fever; and family history of an adverse reaction to vaccination. Anaphylactic reactions, neuropathies, and encephalopathies are rare and constitute the only true contraindications for giving toxoid. Patients who give a history of anaphylaxis should be referred for skin testing because they may no longer be reactive and can receive future vaccinations (27).

HTIG is derived from human plasma. Consisting of IgG, it has a half-life of 25 days (36). A single dose will provide long-lasting protection. It is available in standard 250 IU doses and is approved only for intramuscular use. Intradermal injection will cause local irritation caused by the concentration of the product and does not represent an allergy to HTIG. Because of this reaction, infiltration of HTIG at the wound site is not recommended. Intravenous injection can cause hypotension. Adverse reactions to properly administered HTIG are rare and consist largely of discomfort at the injection site and slight temperature elevation (27). A history of a severe adverse

Table 3. Wound Characteristics*

Nontetanus Prone	Tetanus Prone
<6 hours old	>6 hours old
<1 cm deep	>1 cm deep
Clean	Contaminated
Linear	Stellate
Neurovascularly intact	Denervated, ischemic
Not infected	Infected

* Adapted from Edlich et al. (66).

reaction likely represents previous use of equine antitoxin, which has a 10% incidence of serum sickness and 1/100,000 incidence of fatal anaphylaxis (36). HTIG is widely available in the United States, and so equine antitoxin is rarely used. In contrast, equine antitoxin is used extensively in developing countries because of greater availability and affordability.

In the setting of an acute injury, the Centers for Disease Control and Prevention (CDC) recommendations for tetanus prophylaxis depend on the wound characteristics and the patient's immunization history (Tables 3 and 4). Many wounds encountered in the ED can be considered non-tetanus-prone: recent, linear with sharp edges, well vascularized, and not obviously contaminated or infected. All other wounds are considered tetanus-prone, particularly those resulting from blunt trauma and bites, and those that are grossly contaminated or infected. The CDC recommends HTIG 250 IU only for patients with tetanus-prone wounds and who have never completed a primary immunization series. HTIG should be given at a site separate from the tetanus toxoid to avoid interaction between the two.

If the patient has not completed a primary immunization series, a tetanus booster is required, and the patient will need follow-up to complete the series. If the patient has had primary immunization, a booster is given if the last dose was more than 5 years previous in a tetanus-prone wound or more than 10 years previous in a non-tetanus-prone wound. Patients with a contraindication to tetanus toxoid must be managed with administration of HTIG alone (27).

Table 4. Tetanus Prophylaxis in the Acute Wound*

		Clean Wounds	Tetanus-prone wounds	
Primary Immunization		Td†	Td†	TIG
Not Complete		Yes	Yes	Yes
Completed, Last Booster:	<5 yrs	No	No	No
	>5 yrs	No	Yes	No
	>10 yrs	Yes	Yes	No

* Adapted from Immunization Practices Advisory Committee (27).
† DTP or DT for children <7 years old.

Table 5. Simplified Tetanus Prophylaxis in the Acute Wound

Primary Immunization		Td*	TIG
Not Complete		Yes	Yes
Completed, Last Booster:	<5 yrs	No	No
	>5 yrs	Yes	No

* DTP or DT for children <7 years old.

Some authors advocate a "simplified" approach to prophylaxis and suggest that all wounds be considered tetanus-prone (2,62). This view has validity because surgical wounds, theoretically the cleanest wounds possible, have been the source of tetanus (1). This approach would eliminate the sometimes imprecise task of characterizing the tetanus potential of wounds and simplify the use of HTIG and tetanus toxoid. HTIG would be indicated for anyone who has not completed a primary immunization series, and tetanus toxoid would be indicated if the last booster was more than 5 years previous (Table 5).

Recommendations for primary immunization depend on the age of the patient. If the patient is younger than 7 years old, DTP should be given at 2, 4, 6, and 15 months of age, with a booster at 4 to 6 years of age. DT can be used if there is a contraindication to pertussis vaccine. If the patient is over 7 years old, immunization can be accomplished by three injections of Td. The first two doses should be given at least 4 weeks apart with the third dose 6 months after the second dose.

IMMUNIZATION IN HIGH-RISK GROUPS

Patients from certain high-risk groups often present to the ED for care and require special attention, as standard prophylactic care may not provide sufficient protection from tetanus. These groups include the elderly, HIV-infected individuals, and i.v. drug users (IVDU).

Because of a declining immune system, the elderly do not respond as well to vaccination as do younger adults and children. The formation of tetanus antibodies after vaccination is not as quick, does not peak as high or last as long as in younger individuals (61). In a similar fashion, the immunogenic response to vaccination in HIV-infected individuals is blunted, and antitoxin antibody levels fall more quickly over time than in HIV-negative individuals (63). As expected, the response to vaccination deteriorates as HIV progresses.

The elderly, HIV-infected, and otherwise immuno-

compromised patient may not have "protective" levels of tetanus antibodies at the time of injury, and vaccination alone may not lead to a quick or adequate enough formation of antibodies to protect the patient. If prophylactic treatment is indicated, a more liberal use of HTIG in these patients, regardless of primary immunization, may be warranted to ensure protection against tetanus. More frequent dosing of tetanus toxoid (i.e., every 5 years) may help to sustain adequate antibody levels in the "protective" range. There are no official guidelines endorsing this approach. However, in considering the risks and benefits to a given patient, it should be kept in mind that HTIG and tetanus toxoid are very safe, whereas the morbidity and mortality associated with acute tetanus is considerable.

IVDUs are a burgeoning high-risk group for tetanus. They accounted for 18% of all cases of tetanus between 1995 and 1997, whereas drug abusers accounted for only 2.1–4.5% of cases between 1982 and 1994 (1). Factors that place IVDU at risk include low rates of immunization; contaminated drugs; repeated injection wounds under dirty conditions; and formation of skin abscesses and chronic ulcers, which provide ideal conditions for tetanus development (1). Social and behavioral factors often lead to delayed and sporadic medical care, which compound these risk factors. Intravenous drug users may present for complaints unrelated to acute wounds; however, their drug use should be considered a risk factor that requires attention for tetanus prophylaxis. Both the CDC and the American College of Emergency Physicians recommend using each clinical encounter as an opportunity to ensure adequate immunization, even if the presenting complaint is not wound-related (1,64). These recommendations can be applied to all patients at risk for tetanus, especially high-risk groups such as the elderly and the immunocompromised.

IMMUNIZATION DURING PREGNANCY

In treating the pregnant woman, there are three goals: protecting the mother from acute tetanus, preventing neonatal tetanus, and avoiding treatment harmful to the fetus. Fortunately, these goals are not contradictory. Td is considered safe for the fetus. When possible, immunizations are avoided in the first trimester to avoid teratogenic effects. However, there is no evidence that tetanus or diphtheria toxoid is teratogenic (27). A case-control study by Silveira et al. showed no difference in rates of congenital anomalies between babies born to mothers who received tetanus vaccine in the first trimester and those who did not (65). Td is contraindicated during pregnancy only if it is contraindicated for the woman herself.

The safety of HTIG in pregnancy is not as well documented as Td. Immune globulins, being native human proteins, would not be expected to have an adverse effect on the fetus; on the other hand, failure to prevent acute tetanus in the mother would. If the situation is appropriate, HTIG should be used. Certainly, if a pregnant woman has acute tetanus, HTIG should not be withheld.

CONCLUSION

Tetanus is uncommon in the United States. Thanks to laws mandating pediatric immunization, tetanus is a rare disease in children. The patients most likely to present with acute tetanus in the United States are older adults and IVDU. Lack of routine medical care and vaccination contribute to low levels of immunity. Tetanus is diagnosed clinically, through recognition of its characteristically inducible muscle spasms. Treatment includes aggressive use of muscle relaxants, benzodiazepines and paralytics, metronidazole, tetanus toxoid, and HTIG. Even with state-of-the-art treatment, tetanus is associated with significant mortality. Prevention is far more successful. A conservative approach to prophylaxis is best. Tetanus boosters and HTIG should be used if there is any doubt about adequate immunization. Pregnancy should not prevent proper prophylaxis. Although the causative bacteria, *C. tetani*, is ubiquitous in nature and can never be eliminated, acute tetanus can be made vanishingly rare in the United States with meticulous surveillance, immunization, and wound care.

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