

Tetanus: Uncommon, But Still a Threat

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Abstract

Tetanus spores are ubiquitous. Clinical infection with tetanus leads to four separate syndromes. A case presentation involving tetanus is described with a review of the four presentations, along with diagnostic and therapeutic recommendations.

Case report

A 37-year-old female complaining of severe and worsening pain in the left arm over the last eight hours presented for care. She was unable to flex the elbow and progressive swelling extended from the elbow to the hand. She also complained of pain in the jaw with inability to open her mouth and dysphagia for the last four hours. Approximately a day and a half prior to presentation she had sustained a puncture wound to the left forearm from a rusty nail. The nail penetrated her arm a very small distance (“less than a centimeter”). At the time of the incident she washed the area with soap, water, and peroxide and took some over the counter analgesics. Past medical history was unremarkable except for chronic obstructive pulmonary disease controlled with albuterol and montelukast. She was allergic to penicillin having experienced rash. She was married, unemployed, a smoker, and a social drinker, but denied intravenous drug abuse. She had not completed her vaccination schedule as a child because of religious reasons and the only documented tetanus immunization was a single injection after a car accident in 1994.

On physical examination she was an obese white female in no acute distress with a blood pressure of 130/85, heart rate 88, respiratory rate 20, temperature 97.8, and an O₂ sat of 96 percent on room air. She had trismus which limited the jaw opening to 2cm. There was intense pain to palpation at the jaw lines and over the temporomaxillary joints bilaterally. The left upper extremity was swollen from the hand to the elbow. There was a puncture wound in the ventral forearm with mild surrounding erythema which was very tender to touch. The range of motion of the entire extremity was decreased secondary to pain. The rest of the examination was normal, including the neurological examination. A radiograph of the forearm did not reveal any retained foreign bodies, nor was there any gas formation found in the subcutaneous tissue. Laboratory investigation included a complete

blood count, chemistry function testing, and urinalysis, all of which were normal. With a presumptive diagnosis of tetanus, the patient was admitted and given IV metronidazol 500 mg and diazepam five mg every six hours. After one day, symptoms of pain and trismus were significantly better and the patient was discharged on oral metronidazol 500 mg four times a day for a total of 14 days of therapy plus diazepam five mg three to four times a day as needed for muscle spasms and hydrocodone/acetaminophen 5/500 mg as needed for pain. She was also instructed to follow up with her primary care physician to complete the tetanus immunizations in two months.

Epidemiology

Widespread immunization programs and the introduction of the tetanus vaccines in the 1940's have made tetanus an uncommon disease in the United States with an incidence of approximately 50 cases reported per year, most of which occur among immigrants.¹ Worldwide, primarily in developing countries, there are over one million cases reported yearly.² Once common during wartime, most current cases are related to penetrating wounds, IV drug use, tattooing, body piercing and animal bites, although 20 percent of patients have no history of any injury. Other affected groups include the neonatal population with exposure occurring soon after delivery as the umbilical cord may be cut with a contaminated instrument, and the elderly in which the prevalence of protective antibodies is less than 50 percent.³

Pathophysiology

Produced by *Clostridium tetani* (*C. tetani*), a spore-forming gram negative bacilli, the tetanus exotoxin is one of the most toxic biologically active substances second only to botulinum toxin.^{2,4} The spores are resistant to moisture, chemical disinfectants and temperature changes. After penetration into tissue, the spores germinate into mature bacilli, which multiply and produce two types of toxins, tetanolysin and tetanospasmin.⁴ In an adequately immunized individual, the toxins are neutralized without further effects. But in the partially immunized or unprotected person, tetanolysin facilitates bacterial multiplication by damaging tissues while tetanospasmin is transported via blood stream to the presynaptic terminals,

ascending in retrograde direction through the axons to the spinal cord and central nerve system.^{2,4,5} Once located in these areas, the toxin exerts its action by blocking neurotransmitter release, especially gamma-aminobutyric acid (GABA) and glycine. The diagnosis of tetanus is based on clinical history and examination. Laboratory tests, wound cultures and antibody titers are unreliable, inconsistent and take time to provide results.⁵ A good history of adequate immunization practically, but not entirely, rules out the diagnosis. The clinical presentation of tetanus and the onset of symptoms vary according to the amount of toxin produced and transported to the CNS. There are four clinical tetanus syndromes: localized, generalized, cephalic and neonatal.

Local tetanus begins with pain, rigidity and muscle spasms of the affected body part. This may be the only complaint in a patient with a small amount of toxin production or with some antibodies able to neutralize circulating toxin.⁵ If the toxin is not neutralized, it continues its ascending way into the CNS leading to the **generalized** form of the disease. The “Lockjaw,” which refers to pain and stiffness of the masseter muscles, is an early sign and the most common complaint of this stage. This is due to the fact that nerves with short axons are affected first, as typified by the trigeminal nerve.⁶ As the toxin spreads into the spinal cord, more and more muscles are affected with simultaneous spasms and tonic contractions of agonist and antagonist muscle groups, resulting in the characteristic posturing of flexed arms, extended legs, clenched fists and arched back known as “opisthotonos.” These generalized contractions can be strong enough to cause laryngeal obstruction, respiratory muscle paralysis and even fractures. The autonomic dysfunction of this stage of the disease also represents a significant therapeutic challenge and contributes to the high morbidity and mortality. It is characterized by high catecholamine release, as demonstrated by profuse sweating, tachycardia, hyperthermia, hypertension, and urinary retention. **Cephalic** tetanus is quite rare; it occurs after head injuries, most commonly lacerations, penetrating trauma and rarely otitis.⁶ The symptoms result from cranial nerve dysfunction, most commonly the seventh. Cephalic tetanus can remain localized or progress into the generalized form which has a poor prognosis. Finally, **neonatal** tetanus, which represents less than one percent of cases, is seen in developing countries where non-immunized women deliver their babies at home and the umbilical cord is cut with unclean scissors (or a knife) or where the wound is not kept clean. The first symptoms develop within 2-4 days of birth and the infant presents with difficulty feeding and excessive crying. On examination diffuse muscle spasms, a heightened startle reflex and risus sardonicus are classic signs of this disease variation.²

Differential Diagnosis

The differential diagnosis of tetanus is comprised of a variety of entities that can deceive the inexperienced clinician.

Localized dysinhibition may produce a paralysis, which can mimic stroke. But following detailed examination, it should become evident that weakness is not present and the immobility is caused by simultaneous contraction of agonist and antagonist muscles. Dystonic reactions to dopamine blockade (e.g., phenothiazines), meningitis, rabies, peripheral neuropathies, alveolar and peritonsillar abscesses, and psychiatric disorders can also be confused with tetanus.

Treatment

To improve the outcome of patients suffering tetanus, a high level of suspicion in the early stages is paramount. Once the toxin is bound and internalized in the peripheral nerve endings, neutralization by antibodies is not possible. However, early and rapid administration of antitoxin can neutralize unbound toxin. Systemic administration of penicillin (10-20 millions units IV daily in divided doses every 4-6 hours) or metronidazol (500 mg orally every 6 hours; not FDA approved for this use) are both effective against *C. tetani*.⁶ If wound debridement (not the same as early cleansing by the patient) is to take place, some authorities recommend waiting several hours after administration of antitoxin to allow neutralization of toxin before disturbing the target tissue.

Prognosis and extended care

Recovery from a severe case of tetanus can take weeks as nerve synapses regenerate and multiple complications can occur during the course of treatment. Some can be early (such as painful muscle spasms) or more delayed (infection, autonomic dysfunction, pulmonary embolism, pressure sores, malnutrition and paralytic ileus). The prolonged muscle spasms of tetanus can cause vertebral fractures and rhabdomyolysis. For most cases, repeated doses of benzodiazepines (diazepam 5-10 mg IV or lorazepam 2 mg IV) are enough to cause relaxation and mild sedation, whereas for severe cases, neuromuscular blockage and intubation may be required.⁵ Early hydration and frequent assessment of electrolytes and creatinine can help to detect deterioration in renal function. Typically, in the absence of hypoxic encephalopathy, the cognitive function of these patients remains well preserved with the nervous system becoming hypersensitive leading to painful spasms requiring sedation. If prolonged recovery is expected, experts recommend early tracheotomy and parenteral nutrition to improve outcomes. With advances in intensive care modalities, mortality rates from mild and moderated tetanus have decreased, but for severe cases and in delayed care, mortality may continue to be as high as 80 percent.² Poor prognostic features include neonatal and elderly cases, cases with rapid onset of symptoms, generalized tetanus and poor response to therapy.

The single most important intervention in reducing morbidity and mortality from tetanus remains prevention. The

standard immunization schedule includes a primary series of three doses (often combined with diphtheria or diphtheria and pertussis) followed by a booster every ten years. Pregnant women and women in childbearing age should also be immunized to reduce late pregnancy and infant cases. Patients with clean wounds who have had a basic series of three injections need an additional Td booster only if it has been more than ten years since the previous immunization, or if their immunization history is unknown. In these cases no immune globulin is needed unless signs or symptoms of clinical tetanus appear. For patients who have contaminated wounds, who have not completed their primary series or who have not received a booster within the last five years, tetanus toxoid should be administered as well as tetanus immune globulin (250-500 international units IM) in separate sites.

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