The etiological agent for tetanus is *Clostridium tetani* (2).

Tetanus is contracted when *C. tetani* enters an opening in the skin, such as through a wound or cut. It is not passed from an infected individual to another (4). The bacteria reside in soil, dust, and manure (2).

**General Characteristics**

*C. tetani* is an anaerobic, spore-forming, Gram-positive bacillus that produces a neurotoxin called tetanospasmin (6). Before maturing, *C. tetani* are sensitive to high-heat and oxygen. However, the spores produced by mature bacteria are resilient. These spores can survive temperatures of 250°F for 10-15 minutes and are resistant to antiseptics (4). After entering the body through wounds or punctures, spores germinate in low-oxygen conditions and result in the development of the disease (3).

**Virulence Mechanisms**

The neurotoxin tetanospasmin binds to the presynaptic membrane of the neuromuscular junction and reaches the central nervous system by retrograde axon transport along peripheral nerves (6). Tetanospasmin is a highly potent metalloprotease that blocks neurotransmitter release via zinc-dependent cleavage of the protein components that function in neuroexocytosis. As few as 4-10 molecules of toxin are sufficient to block neurotransmitter release due to the molecule’s high neurospecificity and its zinc-endopeptidase activity (5). Once the tetanus toxin interferes with the normal function of neurotransmitters, such as blocking inhibitor impulses, unopposed muscle contractions and spasms ensue (2).

**Signs and Symptoms**

The incubation period for developing tetanus varies between 3-21 days, depending on how far the injury site is from the central nervous system. Shorter incubation periods usually result from heavily contaminated wounds and have worse prognosis than longer incubation periods (3). There are three clinical presentations of tetanus, which include localized tetanus, cephalic tetanus, and generalized tetanus. Generalized tetanus occurs in over 80% of reported tetanus cases (7). The first symptom to typically appear is a spasm or stiffness of jaw muscles, referred to as lockjaw. Later, symptoms include pain or spasms of muscles (neck, back, extremities), seizure-like activity, and fever (4). Due to facial muscle spasms, *risus sardonicus* may develop which resembles a sneering expression in affected patients. More severe cases, develop laryngeal
spasms which leads to airway obstruction and can be fatal if spasm does not pass or is relieved by tracheal intubation (6). The more rare clinical presentations of tetanus differ in that localized tetanus only exhibits muscle spasms at the infected site of injury; and cephalic tetanus is associated with ear infections (otitis media) or head lesions, and resembles cranial nerve palsies (7).

Control/Treatment

There are no available laboratory tests to confirm a tetanus infection, rather the disease is diagnosed based on the provider’s physical exam and inquiry about the patient’s medical history. Tests may be used to rule out other diseases that present with similar symptoms, such as meningitis or rabies (8). Clinically, the organism is rarely recovered from the site of infection (2).

There is no cure for tetanus, and the disease is treated with supportive treatment and management of complications. Notably, the best treatment for tetanus is disease prevention through immunization (3). Once an individual is infected, hospitalization is recommended for disease management. Muscle relaxants and sedatives can be used to control seizures and painful spasms. Possible drugs include diazepam, favored because of its rapid action, or a strong neuromuscular blocking agent, such as d-Turbocurarine or pancuronium, in more severe cases (6). In the event of respiratory complications, endotracheal intubation can be used to establish an adequate airway and in severe cases a tracheostomy, or machine assisted respiration is necessary (2). To reduce the amount of circulating toxin, a tetanus antitoxin (human tetanus immune globulin) can be administered to neutralize any toxin that has not yet reached the central nervous system. However, it does not treat or neutralize toxin that has already been incorporated in the nervous system (6). The neurotoxin responsible for tetanus does not kill the affected neuron, instead it causes the paralysis of the synapse. A new motor neuron grows around the compromised synapse within a few weeks (5). In total, a patient is usually hospitalized between 5-8 weeks (6).

Patients who fully recover from tetanus disease are not protected against future infections because such a miniscule amount of toxin is responsible for the disease. Therefore, complete immunization is recommended (4). In the United States, routine immunizations begin in infancy with a conjugated vaccine called DTap that protects against diphtheria, pertussis, and tetanus. Then, subsequent booster doses of Td should be given every ten years, or after a tetanus exposure if it has been more than 5 years since the last booster. Pregnant females should receive Tdap during each pregnancy, regardless of time between vaccines. Adults aged 19 and above should replace one dose of Td with Tdap to continue immunity against pertussis (8).

Historical/Current Information
The etiology of tetanus was first discovered in 1884 by Carle and Rattone (1). The first inactive form of tetanospasmin (tetanus toxoid) was developed in 1924, primarily for use in the armed services after the high prevalence of tetanus related deaths in World War I (4). Tetanus did not become a nationally reportable disease until 1947, with about 500-600 cases reported per year. Then between 2000-2007, only an average of 31 cases were reported (3). Since the start of nationally reporting the disease, reported cases have declined 95% and tetanus fatalities have declined more than 99% (2). According to the WHO, tetanus disease remains prevalent in parts of the world where immunizations are not incorporated into routine development. The majority of reported cases are birth-associated, specifically with unhygienic deliveries, abortions, or postnatal cord care. Neonatal tetanus typically occurs when non sterile instruments are used to cut the umbilical cord, or nonsterile dressings are used for wound care (7).

Works Cited


