



TETANUS – WHAT A SURGEON SHOULD KNOW ABOUT *CLOSTRIDIUM TETANI*

Tężec – co chirurg powinien wiedzieć o zakażeniu
Clostridium tetani



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Abstract

Tetanus is an infectious disease caused by the spore-forming bacterium *Clostridium tetani*, whose spores are commonly found in soil. Owing to the widespread implementation of vaccination programs, the incidence of tetanus has significantly declined; however, given the frequency of traumatic injuries and the environmental ubiquity of the pathogen, continued vigilance remains essential. The pathogenesis of the disease is primarily mediated by potent neurotoxins produced by the bacterium, particularly tetanospasmin. The hallmark clinical manifestations include generalized muscle spasms, which may progress to severe complications such as respiratory failure and death. Post-exposure management requires careful assessment of an individual's risk of developing tetanus in order to guide appropriate therapeutic strategies. In confirmed cases, treatment typically involves antimicrobial therapy, administration of tetanus-specific immunoglobulin, and supportive symptomatic care. The differential diagnosis should include conditions such as strychnine poisoning, viral encephalitis, oropharyngeal disorders, and rabies.

Streszczenie

Tężec jest chorobą zakaźną wywoływaną przez bakterię przetrwalnikującą *Clostridium tetani*, której przetrwalniki powszechnie występują w glebie. Dzięki szeroko zakrojonym programom szczepień ochronnych zapadalność na tężec znacznie się zmniejszyła, jednak ze względu na częste występowanie urazów oraz obecność drobnoustroju w środowisku, konieczne jest zachowanie czujności. Patogeneza choroby opiera się głównie na działaniu silnych neurotoksyn produkowanych przez bakterię, zwłaszcza tetanospazminy. Charakterystycznymi objawami klinicznymi są uogólnione skurcze mięśni, które mogą prowadzić do poważnych powikłań, takich jak niewydolność oddechowca, a nawet zgon. Postępowanie poekspozycyjne wymaga dokładnej oceny ryzyka rozwoju tężca, co umożliwi wdrożenie odpowiedniej strategii terapeutycznej. W przypadku potwierdzonego zakażenia leczenie zazwyczaj obejmuje antybiotykoterapię, podanie swoistej immunoglobuliny przeciw tężcowej oraz leczenie objawowe. W diagnostyce różnicowej należy uwzględnić zatrucie strychniną, wirusowe zapalenie mózgu, schorzenia jamy ustnej i gardła, a także wściekliznę.

Keywords: treatment; management; prophylaxis; *Clostridium tetani*

Słowa kluczowe: postępowanie; leczenie; profilaktyka; *Clostridium tetani*

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Introduction

Tetanus is an infectious disease caused by *Clostridium tetani*, a Gram-positive, anaerobic bacillus that naturally occurs in soil and in the gastrointestinal tract of mammals. The microorganism exists in both a vegetative form and as spores. *C. tetani* spores are highly resistant to adverse environmental conditions, allowing them to survive in soil and animal excreta for many years. Their main biological function is to ensure the survival of the strain in unfavorable conditions. Destruction of the spore form is possible through exposure to a temperature of 121°C for 15 minutes under a pressure of 1 atmosphere (autoclaving conditions).

Clostridium tetani produces two main toxins: tetanospasmin and tetanolysin. Tetanospasmin is a potent neurotoxin responsible for the characteristic neurological manifestations of tetanus. It acts by blocking the release of inhibitory neurotransmitters in the central nervous system, leading to uncontrolled muscle contractions. Tetanolysin, on the other hand, exhibits hemolytic properties, damaging the cell membranes of erythrocytes and potentially leading to hemolysis and secondary tissue necrosis [1, 2].

Epidemiology

Globally, tetanus remains a disease of epidemiological significance (particularly in its neonatal form in developing countries). According to the World Health Organization, 15,000 cases were reported in 2018 [3]. Before the introduction of routine vaccination in Poland in 1954, approximately 400 cases were reported annually. Between 2000 and 2017, the number of cases in Poland did not exceed 20 per year. In 2019, 17 cases were recorded, followed by two cases in 2020, and 13 in 2023, based on data from the National Institute of Public Health – National Institute of Hygiene in Poland. Notably, no cases of neonatal tetanus have been reported in Poland since 1984 [4].

In recent years, reported cases of tetanus in Poland have predominantly occurred in individuals over 60 years of age, with a tendency to present outside the spring and summer seasons, which may be related to an increase in gardening activities. A common feature in many reported cases is the absence of information regarding the patient's vaccination status, which may indicate a low level of awareness about recommended booster vaccinations. Consequently, it appears necessary to strengthen educational efforts to raise public awareness about the importance of tetanus booster vaccinations in adults [5]. In the aforementioned patients – older adults over 60 years of age who have not received booster vaccinations over an extended period – tetanus tends to follow a particularly severe course, often leading to death.

In recent years, due to anti-vaccination movements, public trust in vaccines has been declining. The number of exemptions from mandatory vaccinations has increased dramatically. Over the past five years, the number of such exemptions in Poland nearly doubled, rising from 48.6 thousand in 2019 to 87.3 thousand in 2023 [6, 7].

Pathogenesis

Tetanus infection most commonly occurs through the entry of *Clostridium tetani* spores into the body following contamination of damaged skin with soil, dust, organic fertilizers, or contaminated objects. Particularly dangerous are situations involving tissue injury caused by non-sterile instruments, such as cutting the umbilical cord under non-aseptic conditions. In such cases, neonatal tetanus (*tetanus neonatorum*) may develop.

The incubation period typically ranges from 4 to 21 days, although in some cases it may be significantly longer – even over 50 days. *C. tetani* spores can remain dormant in the body, encapsulated by connective tissue, and become activated under favorable conditions – such as tissue necrosis, hypoxia, impaired blood supply, or the presence of an anaerobic environment.

This bacterium preferentially colonizes wounds contaminated with organic material and coexisting aerobic flora (e.g., pyogenic bacteria), which consume oxygen and thereby create conditions favorable for the growth of anaerobes. The risk of infection is particularly high in inadequately treated wounds – especially those older than 24 hours, containing necrotic tissue, or resulting from highly penetrating mechanisms such as puncture, laceration, gunshot, or crush injuries.

However, infection may also develop following seemingly minor injuries, such as puncture wounds caused by nails. Cases of tetanus have also been reported after procedures performed under non-sterile conditions, including abortions, tattooing, intravenous injections in individuals with substance use disorders, as well as in patients with chronic wounds, pressure ulcers, and other ulcers, as well as following dental extractions and in the course of middle ear infections.

Tetanospasmin, the primary neurotoxin produced by *C. tetani*, is extremely potent; in a non-immunized individual, the lethal dose is only 2.5×10^{-9} mg [1, 8–10].

After *Clostridium tetani* spores transition into the active vegetative form within a wound, the bacteria multiply intensively and synthesize toxins responsible for the clinical manifestations of tetanus [1]. Once produced at the site of infection, tetanospasmin spreads via the lymphatic vessels into the systemic circulation and subsequently shows affinity for structures of the nervous system. The toxin penetrates the anterior horns of the spinal cord and the motor nuclei of cranial nerves, where it binds irreversibly to neurons.

Tetanospasmin exerts its pathogenic effect by irreversibly inhibiting the release of inhibitory neurotransmitters, including glycine and gamma-aminobutyric acid (GABA) by blocking the synaptic mechanism responsible for their release. This results in a loss of physiological inhibition of motor neurons, leading to increased muscle tone and painful tonic-clonic contractions of striated skeletal muscles. Characteristic clinical symptoms include trismus (lockjaw), spasms of the facial, neck, and trunk muscles, and, less commonly, involvement of the limb muscles [11].

Prognosis

Untreated tetanus may lead to severe complications, including respiratory failure of either obstructive or central origin, acute myocardial infarction, paralytic ileus, and in approximately 90% of cases – death. The highest mortality rates are observed in two age groups: children with incomplete vaccination and consequently low levels of specific immunity, and elderly individuals (over 65 years of age), in whom significantly reduced concentrations of circulating anti-tetanus antibodies are found.

The clinical presentation – particularly the sequence in which increased muscle tone develops in different muscle groups – depends on the route through which tetanospasmin spreads within the nervous system. This process is determined by the location of the wound and the distance the neurotoxin must travel to reach the central nervous system and peripheral nerve endings.

The severity of the clinical course is strongly correlated with the amount of toxin produced by a given strain of *Clostridium tetani*. The most severe forms of the disease are observed in patients with deep and extensive wounds, in women during the postpartum period, and in individuals who use intravenous drugs [11].

Recovery from tetanus does not confer immunity. Immunity is acquired through vaccination and is age-dependent; in individuals over 60 years of age, it may be as low as 30% [9, 10]. The major risk factors for tetanus infection are presented in Table 1.

Clinical symptoms

In adults, tetanus most often develops after minor skin injuries, such as abrasions or small cuts, while in children the most common route of pathogen entry is chronic otitis media. The initial phase of the disease is the prodromal phase, preceding the onset of full-blown tetanus. During this phase, nonspecific symptoms may occur, such as anxiety, general malaise, increased muscle tension, excessive sweating, headache, insomnia, as well as pain and paresthesias surrounding the wound.

In the advanced stage of the disease, particularly in more severe cases, the dominant clinical symptom is generalized tonic spasms of the skeletal muscles, which may be accompanied by respiratory arrest and cyanosis, while full consciousness is preserved, resembling the clinical picture of botulinum toxin poisoning. One of the pathognomonic symptoms of tetanus is the tonic spasm of the jaw muscles, leading to an inability to open the mouth, known as trismus (lockjaw), which sometimes results in the characteristic “snout-like” positioning of the lips.

Persistent increased tension of the facial mimic muscles gives the face an immobile, mask-like expression, sometimes creating the appearance of an ironic smile, referred to as risus sardonicus (sardonic smile) [12–14].

In severe, fully developed cases of tetanus, repeated episodes of generalized tonic spasms of the skeletal muscles are observed, lasting from a few seconds up to several minutes. Their frequency can exceed a dozen episodes per hour. These spasms, resulting from uncontrolled excitation of motor neurons, lead to a significant increase in muscle metabolism, which in turn causes hyperthermia and symptoms related to the autonomic nervous system, such as tachycardia, hypertension, excessive salivation, and sweating. The spasms primarily affect the muscles of the neck, back, trunk, abdomen, and lower limbs, producing the characteristic arched posture and stiffening of the body known as opisthotonus. Involvement of the upper limbs is rare and usually does not dominate the clinical picture of the disease [12–14].

Depending on the severity of symptoms, the clinical course of tetanus can be classified into the following degrees of severity – mild form: trismus (lockjaw), sardonic smile, occasional and mild muscle spasms, possibly slight stiffness; moderate form: dysphagia, marked stiffness, more frequent periodic muscle spasms, increased respiratory rate (>30 breaths/min); severe form: respiratory failure, generalized muscle spasms, tachycardia, blood pressure fluctuations, heart rate >120/min, increased respiratory rate (>40 breaths/min), episodes of apnea; very severe form (sometimes mentioned): all symptoms of the severe form, plus pronounced autonomic nervous system

Table 1. Tetanus – risk factors for disease development

Risk factors for tetanus development	
<ul style="list-style-type: none"> • Injury while working with soil • Obesity (poorer response to vaccination) • Intravenous drug use • Lack of up-to-date tetanus vaccination 	
High risk of tetanus infection	Low risk of tetanus infection
Crush wounds	Well-vascularized, small superficial wounds occurring in a home environment, without contain necrotic tissue
Deep puncture wounds	
Gunshot wounds	
Wounds containing foreign bodies	
Wounds heavily contaminated with soil, feces, or saliva	
Wounds not treated within 24 hours	
Wounds with associated shock	
Burns or frostbite	

Table 2. Clinical forms of tetanus [1, 8, 9, 12, 14]

Forms of tetanus	Course
Generalized	Most common form, occurring in about 80% of cases. Spasms appear in a descending pattern, starting with increased tension of the masseter muscles, difficulty chewing, progressing to trismus and sardonic smile (increased tension of the orbicularis oris muscle), followed by stiffness of the neck, difficulty swallowing, and stiffness of the chest muscles.
Localized	Rare form of tetanus, characterized by muscle stiffness localized around the wound (site of infection). In cases of partial immunity to the tetanus toxin, it may resolve spontaneously. Most commonly, it represents a prodromal phase of generalized tetanus.
Neonatal	Generalized form occurring in newborns of non-immunized mothers. Common in developing countries due to infection of the umbilical stump. Presents with poor feeding, facial grimacing, and severe spastic contractions that can be triggered by touch.
Cephalic	Rare form of tetanus, sometimes described as a specific type of localized tetanus, resulting from head injury. Presents as cranial nerve palsy (most commonly affecting the facial nerve, cranial nerve VII) and often weakness of the facial muscles (due to damage to the lower motor neuron).

disturbances, including severe hypertension, tachycardia or hypotension, and bradycardia [13, 14].

Depending on the clinical course, tetanus can present in four: generalized, localized, neonatal, and cephalic, which are presented in Table 2 [1, 8, 9, 12, 14].

Post-exposure prophylaxis – management

The procedure for managing suspected tetanus cases has been developed and adopted by the Epidemiological Committee of the Sanitary-Epidemiological Council under the Chief Sanitary Inspector. Management of wounds at risk of tetanus infection includes:

I. Wound care (cleaning and surgical management) – prevents the multiplication of *Clostridium tetani* in the wound, thereby inhibiting the production of tetanus neurotoxin.

II. Administration of antibiotics – to inhibit the growth of *C. tetani*. The most effective antibiotic is metronidazole, administered intravenously at a dose of 500 mg every 6 hours or 1,000 mg every 12 hours for 7–10 days. Older textbooks also note the effectiveness of penicillin, which is active in vitro against *C. tetani*, but as an antagonist of GABAergic transmission it may worsen prognosis. In case of metronidazole allergy, doxycycline (100 mg every 12 hours) is the drug of choice, or alternatively a macrolide or clindamycin, for 7–10 days. However, antibacterial therapy plays a relatively minor role.

III. Active immunization – involves administration of tetanus toxoid vaccine, either monovalent (tetanus only) or polyvalent (combined with pertussis and diphtheria). The primary vaccination in children consists of 4 DTP doses over two years, which provides immunity for approximately 5 years. Booster vaccinations are given to maintain basic immunity at ages 6, 14, and 19 years.

IV. Active-passive immunization – administration of human tetanus immunoglobulin (HTIG) in addition to the vaccine. Prophylactically, a dose of 250 IU is recommended. If more than 24 hours have passed since injury or if the wound is complicated, a dose of 500 IU should be administered. Higher doses are also recommended for obese individuals. For therapeutic purposes, the dose should be 3,000–6,000 IU, given as a single intramuscular injection

(without allergy testing; dosage may be modified according to manufacturer recommendations), combined with appropriate supportive treatment. In exceptional cases where HTIG is unavailable, equine antitoxin may be administered intramuscularly at 40,000–100,000 IU (after allergy testing). The antitoxin neutralizes free neurotoxin that has not yet bound to receptors, which shortens disease duration and alleviates symptoms [14, 15].

It is important to remember that:

- a single dose of vaccine given to an unvaccinated person does not provide immunity;
- the level and duration of immunity depend on the number of vaccinations received;
- having had tetanus does not confer immunity and does not protect against reinfection;
- routine use of antibiotics is not recommended in tetanus prophylaxis; however, wound observation and appropriate antibiotic therapy should be prescribed if signs of infection occur;
- tetanus prophylaxis should always be undertaken [14–16].

Table 3 may help facilitate decision-making regarding management.

In surgical practice, physicians should know when to apply post-exposure prophylaxis in patients presenting with wounds. The risk of developing tetanus is low in fresh, minimally contaminated wounds without necrotic tissue. The risk is high in heavily contaminated wounds, puncture wounds, lacerations, crush injuries, gunshot wounds, wounds treated after more than 24 hours, or wounds with necrotic tissue present within the injury [15, 17].

Differential diagnosis

Tetanus should be differentiated from other causes of increased muscle tone and spasms. In distinguishing tetanus from strychnine poisoning, normal muscle tone during the period without spasms supports strychnine poisoning, which is not seen in tetanus. When differentiating from viral encephalitis, which can also present with increased muscle tone and seizures, tetanus is indicated by complete preservation of consciousness [1]. Unlike encephalitis or meningitis, headache is not a feature in tetanus [13].

Table 3. Tetanus post-injury prophylaxis

Vaccination history	Risk of developing tetanus	
	Low risk	High risk
Unvaccinated person Uncertain vaccination history Incomplete vaccination	Unassociated tetanus vaccination according to the basic schedule (3 doses)	Unassociated tetanus vaccine according to the basic schedule + specific immunoglobulin (toxoid)
Last vaccine dose administered > 10 years ago	One booster dose	One booster dose + specific immunoglobulin (toxoid)
Last vaccine dose administered 5–10 years ago	One booster dose	One booster dose
Last vaccine dose administered < 5 years ago	Immunoprophylaxis not required	Immunoprophylaxis not required; in very high-risk cases, administration of one booster dose should be considered

Trismus is not unique to tetanus and requires exclusion of peritonsillar abscess, cellulitis of the floor of the mouth, odontogenic periostitis, and serum sickness, in which jaw joint pain with trismus may sometimes occur. Swallowing disorders may be of central origin, so this cause should be considered in the differential diagnosis, as along with rabies [1].

Tetanus should also be differentiated from tetany, overdose of psychoactive substances, or acute dystonic reaction, which may result from the use of haloperidol or promethazine. In acute dystonic reactions, neck muscle stiffness occurs with accompanying head twisting to the side, a feature not observed in tetanus [18].

Tetanus is diagnosed clinically, as there are no laboratory tests routinely available in practice to confirm the disease. A useful diagnostic tool is the spatula test: touching the posterior pharyngeal wall with a spatula normally triggers a gag reflex, whereas in tetanus it elicits a masseter muscle spasm, causing the patient to bite down on the spatula [1, 13]. This test has 100% specificity and 94% sensitivity [1].

Cerebrospinal fluid in tetanus usually shows no abnormalities [10].

Tetanus treatment

In suspected symptomatic tetanus, rapid action is essential. Whenever possible, it is important to ask the patient about their vaccination status, take a detailed history, attempt to identify the entry point of infection, place the patient in a dark and quiet room (preferably in the Intensive Care Unit), and draw blood for biochemical and toxicological tests (including tests for strychnine, neuroleptic drugs, phenothiazine derivatives, narcotics, and other tests guided by clinical suspicion) [14].

To quickly neutralize circulating toxin, it is crucial to administer human tetanus immunoglobulin (HTIG) at 3,000–6,000 IU intramuscularly as soon as possible, without allergy testing, or if unavailable, equine antitoxin at 40,000–100,000 IU intramuscularly and/or intravenously after an allergy test. Dosage should be adjusted according to the manufacturer's recommendations [14]. Surgical removal of necrotic tissue and thorough wound debridement to eliminate *Clostridium tetani* spores from the wound is also very important [14].

To eliminate the bacteria, antibiotic therapy should be initiated:

- Metronidazole IV, 500 mg every 6 hours or 1,000 mg every 12 hours for 7–10 days. In case of intolerance or allergy to metronidazole:
- Doxycycline (100 mg every 12 hours). For intravenous administration, proper preparation of the solution is essential: the doxycycline solution should be protected from light; the contents of the ampule should be diluted with sterile water for injection to 10 ml, then the stock solution should be further diluted in 100 to 1,000 ml of 0.9% sodium chloride or 5% glucose solution (yielding a concentration of 0.1 mg to 1 mg doxycycline per 1 ml). The solution should be prepared immediately before use and administered as an infusion over 1–4 hours;
- Alternatively, a macrolide or clindamycin may be considered (antibiotic treatment for 7–10 days) [12, 19].

It is crucial to maintain control over muscle spasms and excessive sympathetic nervous system activity. Frequent, strong spasms triggered by external stimuli – such as touch, pain, bright light, or sounds – can cause apnea and rhabdomyolysis [13]. To achieve sedation, reduce muscle tension, and prevent spasms, intravenous benzodiazepines may be administered (e.g., diazepam 10–40 mg every 1–8 hours or midazolam 5–15 mg/hour, depending on need). Based on available literature, midazolam may be the preferred choice; however, current reports are based on case series and individual clinical cases in which combinations of different drugs were used [1, 14, 20]. During benzodiazepine therapy, clinicians must monitor for excessive sedation, respiratory depression, or even coma. Antispasmodic treatment for tetanus should be continued for a prolonged period. After treatment ends, it is important to gradually reduce doses to avoid withdrawal syndrome [14].

Equally important is ensuring airway patency and proper ventilation. If airway obstruction persists, intubation and mechanical ventilation should be performed [14]. Early elective tracheostomy is recommended in moderate or severe cases to prevent aspiration and stridor [13].

In cases of very severe muscle spasms persisting despite sedation and/or mechanical ventilation disturbances caused by muscle contractions, intrathecal baclofen (administered at a dose of 1,000 µg every 24 hours or 40–200 µg as a single dose, followed by continuous infusion) can be considered. Alternatively, neuromuscular block-

Table 4. Tetanus treatment

Tetanus treatment scheme		
Symptomatic treatment	Control of muscle spasms, securing the airway, maintaining proper blood pressure	Sedatives, muscle relaxants, mechanical ventilation, beta-blockers, morphine, magnesium sulfate
Specific treatment	Antibiotic therapy	Metronidazole, tetracycline
	Specific immunoglobulin	
Surgical treatment	Removal of microorganisms through proper wound cleansing	

ade using agents such as pancuronium or vecuronium may be employed, as these have been shown to be effective in the treatment of severe tetanus [9, 14]. When using neuromuscular blockade, it is important to maintain intravenous benzodiazepine therapy at the same doses to prevent autonomic nervous system hyperreactivity. The duration of neuromuscular blockade itself should be kept as short as possible [13].

It should also be remembered that the effect of muscle relaxants is influenced by factors such as acid-base balance disturbances (acidosis increases the effect of non-depolarizing skeletal muscle relaxants, while alkalosis decreases it; the opposite effect occurs with depolarizing muscle relaxants), and liver or kidney dysfunction. Therefore, in cases of renal failure or hepatic dysfunction, atracurium or cisatracurium appear to be the preferred drugs. However, in renal failure, clinicians should be aware of the possible accumulation of laudanosine, a metabolite with potential epileptogenic and hypotensive effects [21, 22].

For severe symptoms related to the sympathetic nervous system (tachycardia, excessive secretions, blood pressure spikes, excessive sweating, urinary retention), the following medications may be used:

- Magnesium sulfate i.v. (40 mg/kg over 30 minutes, followed by 2 g/h continuous infusion); literature shows it has a beneficial effect on controlling muscle spasms and sympathetic instability, reducing the need for ventilatory support and decreasing mortality; during magnesium sulfate treatment, patellar reflex should be periodically assessed; if absent, the dose should be reduced.
- Labetalol i.v. (0.25–1 mg/min)
- Morphine i.v. (0.5–1 mg/kg body weight per hour as continuous infusion) [1, 11, 14, 20].

If these measures are ineffective, epidural blockade may be considered [13, 14]. In cases where labetalol alone is insufficient to lower blood pressure, clonidine has been reported to improve outcomes [20].

For bradycardia, cardiac pacing should be applied; in hypotension, administration of crystalloids is recommended. During treatment, enteral nutrition via feeding tube should also be ensured – preferably with a high-calorie diet – alongside thrombosis prophylaxis and pressure sore prevention [1, 14].

For severe or moderate tetanus cases, treatment should take place in the Intensive Care Unit [14].

After resolution of muscle spasms, rehabilitation should begin, including physiotherapy and psychotherapy. Additionally, tetanus vaccination should be planned and started. Complete primary vaccination is recommended for unvaccinated patients, while previously vaccinated individuals should receive two doses spaced more than four weeks apart. The vaccine should be administered at a site different from where HTIG was given [14]. The therapeutic approach is summarized in Table 4.

Summary and conclusions

Tetanus is an acute infectious disease characterized by tonic spasms of the skeletal muscles. The spasm of the masseter muscles is referred to as trismus. Every patient with a wound requires tetanus prophylaxis, which can be either nonspecific or specific. Nonspecific prophylaxis involves cleaning and surgical debridement of the wound. Specific prophylaxis includes active-passive immunization, guided by an assessment of the patient's risk of developing tetanus. Despite widespread vaccination in many countries, tetanus remains present worldwide. There is a continuing need for further public education to raise awareness about the importance of booster tetanus vaccinations in adults. This disease leads to serious complications that can be life-threatening, so treatment requires both specific immunoglobulin therapy and supportive care, including intensive therapy. The availability of a highly immunogenic vaccine and effective, safe immunoglobulin allows for prevention even after exposure to tetanus spores. The most important factor is proper patient assessment for appropriate management. Key factors include the patient's tetanus vaccination history, immune status, and the nature of the wound.

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