
Trismus: Or is it tetanus? A report of a case

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CUMBERLAND INFIRMARY

Tetanus is a life-threatening infection that is rare in the developed world. Trismus and dysphagia are the most common presenting symptoms, and thus oral and maxillofacial surgeons may be involved in the early stages of evaluation of the patient. Early diagnosis helps initiate prompt management and referral for intensive therapy. This article is a case report of severe generalized tetanus with a discussion of its diagnosis, pathophysiology, and management. (**Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:437-41**)

Tetanus is a life-threatening infection that is rarely encountered in the developed world.¹⁻³ The overall case-fatality rate among reported cases of tetanus in England and Wales between 1984 and 2000 was 29%.^{4,5} In the 19 years between 1985 and 2003, 145 cases have been reported in England and Wales, with 30 deaths (Table I). In Scotland only 7 cases have been reported between 1990 and 2003.⁶ There is a worldwide incidence of about 1 million cases yearly.⁷

Because of the rarity of the disease the clinician may be unfamiliar with the clinical presentation and unsuspecting of the diagnosis. However, given the severe and potentially fatal outcomes in untreated patients and the fact that there is a chance for full recovery if optimally managed, familiarity with the disease and its clinical features, pathogenesis, complications, and principles of management is important for every clinician.

There has been an increase in incidence in association with intravenous drug abuse. In the period of July to November 2003, 7 clinically diagnosed cases of tetanus were reported in intravenous drug abusers in England and Wales.⁸

Trismus can be the primary presenting symptom in 50%-75% of the cases^{9,10} and this may result in an initial presentation to an oral and maxillofacial surgeon. The following is a case report of a patient who was referred to our department with trismus as the presenting symptom.

CASE REPORT

The patient was a 58-year-old diabetic man, who was admitted under the care of the general surgeons for the amputation of his right second toe (Fig. 1). He was referred to

the maxillofacial surgeons on the fourth day postoperatively with a 24-hour history of dysphagia and trismus.

History revealed progressive difficulty in mouth opening and swallowing over the previous 24 hours with no related temporomandibular (TM) joint pain, toothache, or swelling. Past medical history included maturity-onset, insulin-dependant diabetes, Raynaud's disease, and peripheral vascular disease.

On examination he was afebrile. Maximal mouth opening was measured at 4 mm interincisally with minimal tenderness in the TM joints and the muscles of mastication; however, the masseters were in spasm bilaterally. The maxilla was edentulous but the lower teeth were in a reasonable state of health without any tenderness, pockets, mobility, or caries. The oral mucosa was unremarkable, as were the peritonsillar areas. The neck was nontender, nonswollen, and had a full range of movements. Swallowing was labored, but speech was normal. A general systemic examination was unremarkable, with no evidence of spasms elsewhere in the body. A panoramic radiograph showed normal TM joints and no obvious dental pathology.

Differential diagnosis included TM joint-related pathology, a subclinical orofacial infection, and tetanus.

A spatula test could not be performed owing to a lack of access to the posterior pharyngeal wall.

The postoperative antibiotic regime of intravenous (IV) flucloxacillin and metronidazole was continued and the patient was observed as an inpatient.

Six to eight hours later on day 4 postoperatively the trismus progressively worsened; the patient was increasingly restless but remained afebrile. Metoclopramide-induced dystonic reaction was suspected, and procyclidine 10 mg and diazepam were administered intramuscularly (IM). This stabilized the condition.

In the morning of day 5 postoperatively the patient demonstrated spasms of the sternocleidomastoid muscles with pain and limitation of neck movements. Temperature ranged between 37.5°C and 38°C. Cardiovascular respiratory and abdominal examinations were normal. Gross limb movements were normal. A diagnosis of tetanus was made and the patient was immediately transferred to the intensive care unit (ICU).

On admission to the ICU the patient was tachycardic (90-115 bpm), tachypnic (22-27/min) and febrile (38.1°C). Blood pressure fluctuated between 188/95 and 150/65 mm Hg and

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Table 1. Tetanus notifications, deaths, and vaccine uptake rates, England and Wales, 1985-2004

	Notifications	Deaths	Vaccine uptake rate
1985	12	3	85%
1986	11	3	85%
1987	9	2	87%
1988	12	5	87%
1989	16	2	89%
1990	9	1	92%
1991	8	3	93%
1992	6	1	95%
1993	8	3	95%
1994	3	0	95%
1995	6	2	96%
1996	8	0	96%
1997	7	2	96%
1998	7	1	96%
1999	3	1	95%
2000	2	1	95%
2001	6	0	94%
2002	4	0	94%
2003	8	0	94%
Totals	145	30	

Source: Notifications of Infectious Diseases (PHLS Communicable Disease Surveillance Centre), Office for National Statistics, Department of Health Statistics Division. Crown Copyright material is reproduced under Class License number C01W0000426 with the permission of Controller of HMSO and the Queen's Printer for Scotland.

oxygen saturations between 91% and 96%. Central venous pressure (CVP) ranged from +8 to +15, arterial blood gases (ABGs) were within normal limits, hemoglobin (Hb) was 11.9 g/dL, white cell count was 14.3K, and blood glucose was 5.9 mmol/L. The ECG showed a sinus rhythm, and a chest radiograph was normal. Urinary output was 60-90 mL/hour.

A decision was made to secure the airway via a percutaneous tracheostomy and to insert a nasogastric (NG) tube and a urinary catheter. A continuous infusion of magnesium sulphate ($MgSO_4$) was commenced at the rate of 1 mg/hour following a loading dose of 40 mg given over 30 minutes. The dose was titrated against the presence of the patellar reflex. Sedation was achieved with 8 mg of IV midazolam followed by 1 mg as necessary, and with boluses of diazepam 2 mg 4 times daily via the NG tube. Morphine sulphate was infused at 8 mg/hour and 30 mg of codeine was given via the NG tube. Five thousand IU of human tetanus immunoglobulin (HTIG) was given IV, and 0.5 mL of tetanus toxoid was given IM. The patient was also on IV flucloxacillin, metronidazole, and insulin (sliding scale) as well as 20 mg subcutaneous enoxaparin.

The patient was self-ventilated on 40% warmed and humidified oxygen at 9 L/min; feeding was commenced via the NG tube. The foot wound was cleaned and dressed. The patient was unable to speak but communicated well.

Day 6-10 postoperatively saw a deterioration of the clinical picture; spasms worsened and there was increased sympathetic overactivity (SOA). Spasms became more generalized, affecting the limbs, back, and abdomen. The patient became increasingly restless and hypersensitive to light, noise, and

touch; these symptoms were managed with increasing doses of $MgSO_4$, morphine, midazolam, and diazepam.

Increased mucopurulent secretions from the tracheostomy site required chest physiotherapy and frequent suction. Arterial blood gases deteriorated (pO_2 11.2 Kpa; pCO_2 7.2 Kpa) and SpO_2 dropped (82%-86%) necessitating assisted ventilation. BP, heart rate, and temperature continued to fluctuate. Hemoglobin dropped to 8.8 g/dL and a unit of blood was transfused.

Sputum cultures showed coliforms and *Candida*, and blood and wound cultures identified methicillin-sensitive *Staphylococcus epidermidis* and coliforms. Amoxicillin-clavulanic and fluconazole were added to the IV antibiotic regime and flucloxacillin was discontinued.

By the end of the first week in ICU, the spasms started to decrease in severity and the patient was hemodynamically stable. A repeat chest radiograph was normal. Midazolam was stopped, but the remaining drugs were continued. The second and third weeks in ICU were complicated by urinary tract infection, constipation, and anemia requiring 5 units of red cell concentrate. Only light sedation was required and antibiotics were discontinued after a 12-day course, but $MgSO_4$ and morphine were continued. The wound was regularly dressed; it was clean and started to heal.

By the beginning of the fourth week in ICU, a speech and swallowing assessment allowed initiation of a soft diet, and a speaking valve was inserted. $MgSO_4$, morphine, and diazepam were discontinued. By the end of the week, the tracheostomy tube, the NG tube, and the urinary catheter were removed uneventfully; the patient was mobilizing with the help of a Zimmer frame; he was discharged from ICU to the ward for further recovery.

During the next 10 days on the ward mobility gradually improved to predisease status. He was finally discharged with outpatient follow-up appointments.

DISCUSSION

The etiology and differential diagnosis of trismus can be divided into intra-articular (ankylosis, arthritis synovitis, and disk pathology) and extra-articular, which can be further subdivided as follows:

- Infection/odontogenic—pulpal, periodontal, pericoronaral
- Nonodontogenic—peritonsillar/brain/parotid abscess, tetanus, meningitis
- Trauma—fracture mandible/zygomatic arch, foreign bodies
- Iatrogenic—postextraction, local anesthetic injection
- TMD—trauma, myofascial muscle spasm, internal derangement
- Tumors and oral care—tumors of epipharyngeal and parotid region, TMJ, submucous fibrosis
- Drugs—phenothiazine, succinyl choline, tricyclic antidepressant, metaclopramide, halothane
- Radiotherapy—postradiation fibrosis, osteoradionecrosis



Fig. 1. "Risus sardonicus," which developed 24 hours after presenting with trismus.

Congenital—hypertrophy of coronoid, trismus-pseudocamptodactyly syndrome
Miscellaneous—hysteria, lupus erythematosus¹¹

Trismus is often associated with infections involving the masticator space. Needle tract infection or foreign body reaction involving the pterygomandibular space following administration of mandibular nerve block is an uncommon but recognized complication of local anesthesia.¹²

Tetanus is an infectious disease that results from wound contamination with *Clostridium tetani*, an anaerobic, gram-positive, motile, spore-forming rod that is ubiquitous in nature. Spores may survive for years in some environments. Vegetative cells, however, are easily inactivated and are susceptible to several antibiotics, such as metronidazole and penicillin.¹³

Contamination of wounds with spores of *C tetani* is probably frequent. Germination and toxin production, however, take place only in wounds with low oxidation-reduction potential, such as those with devitalized tissue, foreign bodies, or active infection.¹³ The organism produces 2 exotoxins: hemolysin and tetanospasmin. Tetanospasmin is the neurotoxin responsible for



Fig. 2. Postsurgical wound on right foot.

the clinical manifestations of the disease. The toxin spreads hematogenously to the peripheral nerves and travels in a retrograde fashion along the nerve fibers to reach the central nervous system where it blocks the release of γ -aminobutyric acid (GABA) from presynaptic inhibitory neurons.¹⁴ This loss of inhibitory impulses results in the cardinal clinical manifestations of reflex irritability and autonomic hyperactivity.

Tetanus is an often severe and fatal disease that is difficult to diagnose.¹⁵ Diagnosis is based purely on clinical observation,¹⁶ and very little has been added to the description of clinical features through the centuries. Clinical tetanus comprises 4 symptomatic types: generalized, local, cephalic, and neonatal.⁷ Laryngeal spasm may lead to sudden death from asphyxia.⁷

The commonest presenting symptom is trismus, but patients often develop simultaneous dysphagia as well as pain and stiffness of the neck musculature.¹⁷ Markedly increased tone in the central muscles (face, neck, chest, back, and abdomen) with superimposed generalized spasms and relative sparing of the hands and feet strongly suggests tetanus.¹³

Sustained contractions of facial musculature causes "risus sardonicus"—the so-called "sneering grin" expression (Fig. 2).^{3,18} Rigidity progresses in a descending manner, with the short cranial nerves being affected first. With severe trismus there is opisthotonos, the most dramatic manifestation of the disease, caused by generalized spasm and resulting in the flexion of arms, extension of the legs, and rigidity of the abdominal wall, followed by rigidity of the trunk and limbs.

The spatula test^{10,19} is a simple bedside test to diagnose tetanus, with a sensitivity of 94% and specificity of 100%. The posterior pharyngeal wall is touched with a spatula and a reflex spasm of masseters occurs (positive test) instead of the normal gag reflex (negative test).

Enzyme immunoassays for antitoxin levels are useful but not readily available. A level of 0.01 IU/mL or greater is considered protective, making the diagnosis of tetanus less likely. However, toxin-neutralizing antibodies can be "overwhelmed" if the burden of toxin is high enough, and clinical tetanus, albeit with a mild clinical course, can develop.^{15,20,21} The activity of creatine phosphokinase may be increased in some cases, but it is not pathognomonic.⁷

A high index of clinical suspicion is therefore necessary, especially in patients with risk factors,¹⁰ which include diabetes, chronic wounds (skin ulcers, abscesses, gangrene), parenteral drug abuse (notably "skin popping"), burns, childbirth, recent surgery, unimmunized individuals, and elderly, especially above 60 years of age.^{16,22,23} The median time to onset of generalized tetanus after injury is 7 days.¹³

Treatment requires admission to the ICU, because airway control and mechanical ventilation, neuromuscular blockade, and management of sympathetic overactivity (SOA) are the mainstay of management. ICU management has considerably reduced the mortality rate, from 44% to 15%.⁴

As always, the initial priority of management is airway control and maintenance of ventilation. Fifty percent of mortality associated with tetanus can be attributed to the respiratory complications of the disease.²⁴⁻²⁶ Respiratory failure may occur as a result of muscle rigidity and reflex spasm that characterizes the disease or secondary to hypoxia following atelectasis and pneumonia.³ Tracheostomy is recommended for securing the airway, because the presence of an endotracheal tube is a strong stimulus for spasms.^{16,27,28}

Passive immunotherapy with human tetanus immunoglobulin (HTIG) helps to bind the free tetanospasmin not yet within the nervous system and eliminates as much of the toxic burden as possible.^{16,29} If the patient has received a dose of absorbed tetanus vaccine during the 5 years before injury, a booster dose should be administered; if not, a complete vaccination regimen should be planned.³⁰

All partially immunized and unimmunized adults should receive vaccine, as should those recovering from tetanus. The primary series for adults consists of 3 doses. A booster dose is required every 10 years. Because toxin released during a tetanus infection is insufficient to provide immunity, active immunization must be initiated at the time of presentation either with a booster or a full vaccination series.¹³ The exact effective dose of HTIG has not yet been established and remains a controversy. Acceptable doses range from 500 IU to 10,000 IU^{31,32}; some have noted that 500 IU of HTIG was as effective as the standard dose of 5,000 IU,³³ and others have argued that doses of 3,000-6,000 IU are

optimal.^{34,35} Additional doses are unnecessary, because the half-life of HTIG is long.¹³

Thorough debridement of the offending wound with removal of devitalized tissue and any foreign bodies is essential to eradicate the source of toxin production. The efficacy of antibiotics is unclear, because the disease is caused by a toxin. Penicillin has been traditionally used,^{3,9,17} but because of its potential to act synergistically with tetanospasmin as a GABA antagonist its use has fallen into disfavor. Instead, 500 mg IV metronidazole every 6 hours is now recommended as the first-line antibiotic^{3,36} and has been shown to reduce the progression of the disease.³⁷

Supportive care includes sedation, neuromuscular blockade, and management of autonomic instability (SOA). Antispasticity drugs are key agents. A continuous infusion of midazolam (a GABA-agonist benzodiazepine) is preferred and is employed as a standard in most units,^{31,38} but other agents, such as propofol, vecuronium, pancuronium, dantrolene, and baclofen, have been used.

SOA is common in severe tetanus and manifests as labile hypertension, tachycardia, dysrhythmias, peripheral vascular constriction, profuse sweating, fever, increased carbon dioxide output, increased catecholamine excretion, and, in some cases, late development of hypotension.³⁹ Various drugs have been used to manage cardiovascular disturbances but no single drug or combination of drugs has consistently been shown to be successful in controlling them.⁴⁰ Labetalol (beta-blocker) infusion and morphine sulphate infusions are used to control SOA. Recent studies⁴¹ have shown a multiple beneficial role for magnesium sulphate infusions. In addition to controlling spasms well, it has been shown not only to minimize SOA but also to minimize the need for heavy sedation and mechanical ventilation, thus reducing the mortality from associated complications such as pulmonary sepsis, bronchospasms, atelectasis, deep vein thrombosis, pulmonary embolism, and gastric hemorrhage. However, it is important to titrate the dose against the preservation of the patellar reflex, and maintaining serum concentrations within the therapeutic range is essential.

Supportive care is essential until recovery occurs by the formation of new synapses; in our patient this happened in 30 days. Despite the long-term possibility of nerve palsies, neuropathies, and psychologic after-effects, patients can survive tetanus and return to their predisease state of health, as in this case.

CONCLUSION

Our patient presented with the classic signs and symptoms (trismus and dysphagia) of generalized tetanus with an obvious focus of infection and belonged

to 2 high-risk categories (diabetes and postsurgery). Although there was a clinical suspicion of tetanus, it was not strong enough to prompt ICU admission. A spatula test might have tilted the index of suspicion but could not be performed owing to limited mouth opening. Flucloxacillin was continued for another 24 hours along with metronidazole and this could have had a deleterious effect. An early aggressive approach to initial wound management and immunization may have reduced the toxic burden of tetanospasmin resulting in a likely shorter ICU stay. All these areas of concern help to outline the need for awareness of this rare condition in order to avoid these common pitfalls in management.

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