

# Jacksonian March Revisited? A Case of Local Tetanus with Generalized Spread

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## Abstract

A 41 year-old male with no prior history of seizure disorder presented to the emergency department complaining of four "seizure" episodes which began a few hours prior to arrival. Within minutes of triage, one of the episodes was witnessed and inconsistencies with seizure activity including an absence of clonus, consciousness throughout the event and lack of a post-ictal state were noted. Further examination led to discovery of an untreated laceration on the patient's left hand which had been sustained 1-week prior on a rusted metal fence. More extensive history also revealed recurrent episodes of muscle spasm in his left hand which preceded the generalized attacks. A presumptive diagnosis of wound tetanus with secondary, generalized manifestation was made and the patient was treated with local and intramuscular tetanus immunoglobulin as well as intravenous metronidazole and diazepam. The patient was admitted to the Neurologic Intensive Care Unit and, after 1 week of therapy, made a full recovery. While rare in the developed world, acute tetanus remains a disease associated with a significant morbidity and mortality (even in the United States). The high rate of lapsed adult immunization as well as the prevalence of insufficient effective antibodies even in those sufficiently immunized mandates vigilance on the part of the emergency physicians-particularly in patients with atypical neurological presentations.

## Narrative

A 41 year-old male with a past medical history of insulin-dependent diabetes mellitus presented to the Emergency Department (ED) reporting recurrent "seizure" activity which began a few hours prior to arrival. After a witnessed episode of full-body tonic activity at triage the patient was brought directly to the resuscitation bay, where he was able to provide additional history. He stated that he suffered his first attack while resting at home that evening, and his second a few minutes later while preparing to be driven to the ED. He reported being fully alert during the attacks but unable to move or speak because of full-body spasms. They were becoming increasingly painful. Both he and his fiancé denied any alteration of his mental status in the post-episode period.

Shortly after arrival in the resuscitation bay, the patient suffered another attack involving a full-body spasm with leftward deviation of the head and flexion of the upper extremities. The episode began with a forceful contraction of the patient's left hand and subsequently spread to his entire body, reminiscent of a Jacksonian march seen with certain forms of epilepsy. After approximately 45 seconds, the episode subsided. He had no loss of bladder or bowel control with this event and there was no confusion or post-ictal state upon its completion. While preparations were made to transport the patient for a Computed Tomography (CT) scan of the head, a secondary assessment revealed an open poorly-healing laceration on the palmar surface of the left hand approximately 3 cm in length. It was bandaged loosely, exhibited granulation tissue, and had no evidence of purulent drainage or a foul odor. The patient reported he had cut his hand on a metal fence approximately one week ago and had not sought medical attention. He noted a loss of sensation in his hand along with localized muscle spasms extending up his arm that began 3 days after the injury. The patient could not recall his last tetanus immunization update.

Further physical examination demonstrated flaccid paralysis of the left upper extremity, an inability to shrug the left shoulder, and decreased sensation to light touch throughout that extremity. Work-up including head CT and laboratory analysis (complete blood count, extended metabolic panel, drug screens) was negative. The patient was presumptively diagnosed with a subacute, localized tetanus infection

which had spread to become generalized. Wound cultures were obtained, the wound was thoroughly irrigated with normal saline and 500 units of tetanus immune globulin (a sum representing the total supply available in the ED at that time) were administered, at least half at the wound site. His acute spasms were treated with Intravenous (IV) diazepam and antibiotic coverage (IV metronidazole) for potential *Clostridium tetani* colonization was given.

The patient was admitted to the Neurologic Intensive Care Unit (NICU) for on-going care. While awaiting bed availability in the ED, he continued to suffer recurrent tetanic episodes, one of which was captured in the accompanying video (obtained with the patient's written permission). Further supplies of tetanus immune globulin were obtained from local hospitals in the region and, over the next 24 hours, an additional 2,500 units of tetanus immune globulin were administered. In the interim, his spasms were treated with IV diazepam and, by hospital day 2, all tonic motor activity had completely resolved. By day 3, he had regained full muscle and sensory function in his left upper extremity, though transient paraesthesias remained. He remained hemodynamically stable throughout his hospitalization was discharged on day 7 with no adverse sequelae.

## Discussion

This case report illustrates the potential pitfalls of tetanus diagnosis and the need for emergency physicians to remain suspicious when encountering inconsistent neurological presentations. According to

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the most recent surveillance data released by the Centers for Disease Control (CDC) fewer than 25 cases of tetanus are diagnosed each year in the US [1] but the related case-fatality rate remains high at 13.2% [2]. Death from tetanus typically occurs in those older than 60 years for whom immunization had lapsed or had never been established. However, according to Talan et al. in 2004, nearly 1 in 10 adults aged 18 or older lack immunity [3]. Even with an up to date immunization status, effective tetanus antibodies may be absent. Indeed, 10% of tetanus cases reported between 2001-2008 were in adults with greater than or equal to 4 previous doses of tetanus toxoid, 25% of whom had received a dose of toxoid within the past 10 years [2].

More commonly, tetanus infection presents as a systemic condition with increased tone and episodic spasm, initially affecting the muscles of the head and neck and subsequently spreading caudally. Characteristic features of these episodic spasms include contorting facial contractions (risus sardonius) and forced flexion of the arms with an arching of the back (opisthotonus). Tetanus localized near the site of a peripheral injury, as noted in our patient with muscle spasms initially confined to the left upper extremity, is much rarer but can progress to generalized involvement [4]. The key features which initiated concern for the diagnosis in this case were the untreated peripheral wound, the absence of a post-ictal state or loss of bladder or bowel control, and the rarity of new-onset seizures in a patient of this age.

The most common cause of death in untreated tetanus patients is ventilatory failure related to spasm of the respiratory musculature. In patients who are hospitalized with appropriate ventilatory management, the next most common cause of death is autonomic instability. The latter is due to both direct effects of the tetanus toxin on autonomic nervous system activity and secondary effects of forceful muscle contraction on systemic blood pressure. Death can result from hypotension, especially in the setting of volume loss from prolonged spasms and diaphoresis. Hyperpyrexia is also common and when present significantly increases the risk of death.

As highlighted by our case, the treatment of tetanus infections involves administration of IM tetanus immunoglobulin prior to wound debridement, parenteral metronidazole or intramuscular penicillin,

and control of spasms and autonomic instability with benzodiazepines or neuromuscular blockade. Though not used in our patient, magnesium sulfate may be helpful to control autonomic function and muscle activity [5]. For patients with hemodynamic instability unresponsive to deep sedation and magnesium, clonidine should be considered. Historically beta blockers have been used, but they have now been shown to be associated with a significant risk of cardiac arrest [6]. Infection with *Clostridium tetani* does not confer immunity, so all surviving patients must receive tetanus toxoid at presentation, at 6 weeks, and at 6 months following recovery.

Although tetanus remains a rare disease in the United States, it is a potentially fatal infection that warrants vigilance on the part of emergency physicians. Whereas a textbook patient who has trismus and progressive opisthotonus may be easily recognized, other presentations can mimic more common neurological disorders such as seizure. As with anything else, anchoring on a single diagnosis can have disastrous consequences and we hope this case will prompt consideration of tetanus when atypical features of a seemingly typical neurologic disorder are present.

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