

Non-Wound Tetanus Mimicking Botulism in a Low-Resource Setting: Early Recognition and Successful Intensive Care Management

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Introduction: Tetanus is a vaccine-preventable, toxin-mediated neurologic emergency caused by *Clostridium tetani*. Although classically associated with contaminated wounds, the portal of entry may be trivial or unrecognized, resulting in so-called non-wound presentations that can delay diagnosis. Early tetanus may also mimic other toxin-mediated syndromes, including botulism, particularly when cranial or bulbar symptoms predominate.

Case Presentation: A 41-year-old man in Somalia presented with rapidly progressive trismus, neck stiffness, dysarthria, perioral paresthesia, and marked masseter rigidity over 12 hours. He had no limb weakness or focal neurologic deficits, and brain MRI and CT angiography were unremarkable. Initial history raised concern for botulism because of recent laser eye surgery and ingestion of home-canned vegetables. Repeated history-taking later revealed extensive post-flood debris cleanup with possible unnoticed hand punctures. His most recent documented tetanus vaccination was in 2020, but prior immunization history was incomplete. He received tetanus toxoid, intramuscular human tetanus immunoglobulin, intravenous metronidazole, and ICU observation. He improved within 24 hours and recovered fully by 48 hours.

Conclusion: This case illustrates that tetanus can present without an identifiable wound and may closely mimic botulism when bulbar symptoms and misleading exposure histories are present. In patients with acute trismus and painful rigidity, tetanus should remain a leading consideration despite normal neuroimaging and absent wounds. Early empiric antitoxin, antibiotics, vaccination, and close monitoring can be decisive, and repeated environmental exposure history, especially after flooding or debris handling, may reveal the missing diagnostic link. Importantly, tetanus occurred despite a recent documented vaccination, highlighting uncertainty about protection when prior immunization history is incomplete.

Keywords: tetanus, non-wound tetanus, trismus, botulism mimic, ICU, tetanus immunoglobulin

Introduction

Tetanus is a potentially life-threatening neurologic disorder caused by tetanospasmin produced by *Clostridium tetani*. Although it is vaccine-preventable, tetanus continues to occur worldwide, particularly in settings where immunization coverage is incomplete and booster doses are inconsistently received. It typically follows contamination of a wound, often minor or unnoticed, and is characterized by muscular rigidity and painful spasms, classically presenting with trismus and neck stiffness. Early recognition and prompt supportive care are essential because deterioration may be rapid and severe cases may require intensive care monitoring and airway support.^{1,2}

However, the portal of entry is not always apparent at presentation. Previous reports indicate that non-wound tetanus, although uncommon, remains clinically relevant and may delay diagnosis when no visible lesion is identified.³ The diagnostic challenge becomes greater when tetanus presents with cranial or bulbar features such as dysarthria, because it may resemble other toxin-mediated or neurologic conditions, including botulism.³ Botulism can also present with cranial nerve involvement and dysphagia or dysarthria, and exposure histories such as ingestion of improperly preserved food

may further complicate early clinical judgment.⁴ In addition, atypical forms of tetanus, including localized or cephalic tetanus, may mimic focal neurologic disease.⁵

Here, we report a diagnostically challenging case from Somalia of non-wound tetanus presenting with botulism-like features and a competing exposure history, in which early empiric therapy and close intensive care observation were associated with rapid clinical improvement and recovery. This case highlights the importance of maintaining clinical suspicion for tetanus despite the absence of an obvious wound, particularly in low-resource settings where diagnosis is often clinical and exposure histories may initially be misleading. It also underscores the value of repeated environmental and vaccination history-taking and timely initiation of antitoxin, antibiotics, and supportive care when tetanus is suspected.

Case Presentation

A 41-year-old man arrived at the emergency department of a tertiary hospital in Somalia with symptoms that had progressed rapidly over 12 hours. He complained of jaw tightness (trismus), neck stiffness, slurred speech (dysarthria), and tingling around the mouth. He was alert, cooperative, and fully oriented on assessment (GCS 15). Neurologic examination showed pronounced rigidity of the masseter muscles, with no cranial nerve palsy, ptosis, ophthalmoplegia, pupillary abnormality, limb weakness, sensory loss, or other focal deficits. There was no descending flaccid weakness, which made botulism less likely despite the history of home-canned food ingestion.

He denied recent injuries, intramuscular injections, dental work, or invasive procedures. However, he reported undergoing laser eye surgery two days earlier and consuming home-canned vegetables about two weeks before the onset of symptoms. When his history was revisited, he recalled that his home had flooded one month earlier. During extensive cleanup, he handled debris for prolonged periods and acknowledged the possibility of minor puncture wounds to his hands that may have gone unnoticed. Detailed examination of the skin and extremities did not reveal an identifiable wound or other clear portal of entry. His most recent documented tetanus toxoid vaccination was in 2020; however, prior immunization history was incomplete and unclear.

A tetanus toxoid booster was administered immediately on admission. Empiric treatment was started with intravenous metronidazole (500 mg every 8 hours) and intramuscular human tetanus immunoglobulin (500 IU). No sedatives, benzodiazepines, muscle relaxants, ventilatory support, or additional invasive supportive interventions were required during ICU observation. Symptoms improved substantially within 24 hours, with complete resolution of trismus and dysarthria by 48 hours. Laboratory studies revealed mild leukocytosis ($11.2 \times 10^9/L$) and elevated creatine kinase (479 U/L), while inflammatory markers and renal and liver function tests were within normal ranges. He was observed in the ICU for 48 hours and discharged without symptoms. At follow-up on 1 weeks, he remained asymptomatic without recurrence.

Discussion

This case illustrates how tetanus can mimic other toxin-mediated neurologic syndromes during the early stage of illness, particularly when the presenting history is incomplete and the classic history of a contaminated wound is absent. Although tetanus is classically associated with an identifiable inoculation site, the portal of entry may be trivial, healed, or entirely unrecognized. For this reason, diagnosis remains primarily clinical and is based on characteristic bedside findings, especially trismus and painful muscle rigidity, rather than on neuroimaging or laboratory confirmation alone. Accordingly, normal MRI or CT findings do not exclude tetanus, and treatment should not be delayed when the clinical pattern is strongly suggestive.¹ This diagnostic challenge is especially relevant in low-income and lower-middle-income settings, where specialized confirmatory testing may be unavailable and treatment decisions often depend on clinical examination and exposure history.¹

In this patient, the absence of an obvious recent injury initially lowered suspicion for tetanus. However, non-wound or unrecognized-wound tetanus is well documented and may delay diagnosis when no visible portal of entry is found.² Only after repeated history-taking did the patient recall extensive debris handling after household flooding, providing a plausible environmental exposure. Flood-related cleanup often involves contact with soil-contaminated materials, and small punctures or abrasions to the hands may pass unnoticed.^{1,3} This case therefore reinforces the importance of

revisiting the history when early clinical findings are compatible with tetanus, with particular attention to environmental exposures and seemingly minor skin injuries.³

Botulism was an important competing diagnosis in this case because the patient presented with dysarthria and perioral paresthesia and also reported recent consumption of home-canned vegetables. Both tetanus and botulism are toxin-mediated disorders that may present with bulbar symptoms and rapid progression, creating early diagnostic overlap.⁴ However, the clinical pattern favored tetanus. Painful rigidity and marked masseter spasm are more characteristic of tetanus, whereas botulism typically presents with descending flaccid weakness.^{1,4} Thus, although the exposure history initially suggested botulism, the bedside neurologic examination was more consistent with tetanus (Table 1).

Other differential diagnoses were also considered. Acute dystonic reactions were less likely because there was no relevant medication exposure and the presentation was dominated by persistent trismus and rigidity rather than episodic abnormal posturing. Hypocalcemic tetany was also less likely in the absence of generalized muscle cramps, carpopedal spasm, or biochemical evidence of electrolyte disturbance. Meningismus or meningoenzephalitis was considered less probable because the patient remained alert, fully oriented, and without signs of progressive encephalopathy or systemic infection. Likewise, focal neurologic disease was less likely given the absence of focal deficits and the normal brain MRI and CT angiography findings. Taken together, the predominance of painful rigidity, preserved consciousness, and the absence of descending flaccid weakness supported tetanus over these alternatives.⁴

The patient's dysarthria, in the absence of limb weakness and with normal neuroimaging, also created the impression of a focal cranial process. Localized or cephalic tetanus has been reported to mimic cranial neuropathies and other focal neurologic syndromes, often prompting extensive imaging and vascular evaluation.⁵ In this case, cranial MRI and CT angiography were useful in excluding structural pathology, but they did not account for the overall clinical syndrome. This highlights an important diagnostic principle: normal imaging should not divert attention from toxin-mediated disorders when the bedside examination remains strongly suggestive.^{1,5}

Management was based on established tetanus treatment principles: neutralization of unbound toxin, reduction of bacterial burden, restoration of active immunity, and close monitoring for deterioration. Early administration of human tetanus immunoglobulin is essential because it neutralizes circulating toxin before irreversible binding to nervous tissue

Table 1 Differential Diagnostic Considerations in This Case: Tetanus versus Botulism

Feature	Tetanus	Botulism
Pathophysiology/mechanism	Neurotoxin blocks inhibitory neurotransmission (GABA/glycine) → disinhibition and sustained muscle contraction	Neurotoxin blocks acetylcholine release at the neuromuscular junction → impaired muscle activation
Typical muscle tone	Painful rigidity (spasticity), often starting with jaw/neck	Flaccid weakness/paralysis, often with prominent bulbar complaints
Pattern of progression	May begin locally (eg, jaw/neck) and can progress to generalized rigidity and spasms	Often begins with cranial nerve/bulbar symptoms and progresses descending to limbs
Reflexes	Often normal to increased (hyperreflexia may be present)	Often reduced (hyporeflexia), depending on severity
Autonomic/ocular clues	Pupils usually normal; autonomic instability may appear later in severe disease	Pupillary changes may occur (often dilated or sluggish) and other autonomic features can be present
Key bedside clue	Trismus and masseter spasm strongly support tetanus	Weakness dominates; rigidity and trismus are not typical
Incubation/timing	Usually days to weeks after minor or unnoticed contamination/inoculation	Usually hours to days after ingestion of preformed toxin (foodborne)
Core treatment	HTIG (as early as possible) + metronidazole + vaccination + supportive/ICU monitoring	Botulinum antitoxin + supportive care (including ventilation if needed)
Consciousness	Usually preserved unless complicated by hypoxia, sedation, or severe autonomic instability	Usually preserved; sensorium typically remains intact

Note: The symbol (→) indicates "leads to" in the pathophysiological description.

occurs, while metronidazole helps eliminate the organism and reduce ongoing toxin production.¹ A booster dose of tetanus toxoid was also appropriate, as natural infection does not reliably confer immunity and this patient's prior immunization history was incomplete.⁶ The rapid clinical improvement observed in this case was atypical for tetanus and should therefore be interpreted cautiously. One possible explanation is that treatment was initiated before substantial toxin binding and before progression to generalized spasms or autonomic instability. It is also possible that the patient had an early or localized form of tetanus, which may follow a less severe course than generalized disease. Even so, ICU observation remained appropriate because tetanus may worsen abruptly, and severe cases can require airway protection, sedation, and intensive supportive care.^{1,7}

This case also carries an important prevention message. Tetanus persists because *Clostridium tetani* spores remain widely distributed in the environment, and prevention depends on complete immunization and timely booster doses across the lifespan.² Although this patient had a documented tetanus vaccination in 2020, earlier immunization records were unavailable, limiting confidence in the extent of his prior protection. In many low-resource settings, incomplete vaccination documentation is a common challenge. This case therefore highlights the importance of maintaining complete and up-to-date tetanus immunization, particularly among individuals with potential environmental exposures.^{6,7}

This report has several limitations. First, no definite portal of entry was identified. Second, prior immunization history was incomplete, limiting certainty regarding pre-existing protection. Third, as is common in many low-resource settings, diagnosis was made clinically and specific confirmatory testing for tetanus or botulism was not available. These limitations should be considered when interpreting the unusually rapid clinical response.

Overall, this case highlights that tetanus should remain high on the differential diagnosis in patients presenting with trismus and painful rigidity, even when no visible wound is identified and neuroimaging is normal. Exposure histories may suggest alternative toxin-mediated syndromes, but they should not outweigh examination findings that favor rigidity rather than flaccid weakness. Early empiric treatment is justified when clinical suspicion is reasonable, because delay may permit disease progression and increase the risk of ICU-level complications. Repeated, focused history-taking, especially after flooding or debris handling, may uncover the relevant exposure even when the initial history is unrevealing.³

Conclusion

This case shows that tetanus can present without an obvious wound and may closely resemble botulism, particularly when exposure history (such as home-canned foods) points in another direction. In patients with rapidly progressive trismus and muscle rigidity, tetanus should remain a leading consideration even when neuroimaging is normal and a clear portal of entry is not found. Early administration of human tetanus immunoglobulin, metronidazole, and a tetanus toxoid booster, together with ICU observation, was followed by complete recovery.

Importantly, the patient's likely environmental exposure during post-flood debris cleanup and the uncertainty around prior vaccination records reflect real-world challenges in many settings. This case highlights the critical role of complete and up-to-date tetanus immunization, as gaps or uncertainty in vaccination status may leave individuals vulnerable even when recent doses are documented. It also emphasizes the value of repeated, detailed history-taking and the need to strengthen routine booster coverage, since prevention depends on sustained immunity rather than reliance on identifying a specific wound at presentation.

Abbreviations

CK, Creatine kinase; CT, Computed tomography; CTA, Computed tomography angiography; ED, Emergency department; GCS, Glasgow Coma Scale; ICU, Intensive care unit; IM, Intramuscular; IV, Intravenous; IU, International units; MRI, Magnetic resonance imaging; WBC, White blood cell count.

Ethics and Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying images. In accordance with our institutional Dr sumait Hospital/Simad University policy, ethical committee approval was not required for publication of this single-patient case reports.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no conflicts of interest related to this study.

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