

Post-Traumatic Cephalic Tetanus Complicated by Respiratory Failure: A Case Report and Comprehensive Literature Review

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Abstract

Case Report

Cephalic tetanus is a rare but potentially life-threatening form of tetanus characterized by cranial nerve involvement and a high risk of progression to generalized disease. Despite widespread vaccination, sporadic cases still occur, particularly in elderly patients with contaminated traumatic wounds. We report a case of post-traumatic cephalic tetanus in a 65-year-old polytraumatized patient, complicated by respiratory failure requiring intensive care management. This article discusses diagnostic challenges, prognostic scoring, therapeutic strategies, and outcomes in light of current scientific evidence.

Keywords: Cephalic tetanus, Cranial nerve palsy, Trismus, *Clostridium tetani*, Intensive care, Vaccination.

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INTRODUCTION

Tetanus is an acute neurotoxic disease caused by the exotoxin tetanospasmin produced by *Clostridium tetani*, an obligate anaerobic, spore-forming bacillus. Although preventable by vaccination, tetanus remains a public health issue in low- and middle-income countries and among inadequately immunized elderly populations. Cephalic tetanus accounts for less than 3% of cases and typically follows craniofacial or contaminated peripheral wounds. It is associated with early cranial nerve palsies, trismus, and an increased risk of respiratory compromise.

CASE PRESENTATION

A 65-year-old male with a history of hypothyroidism, poorly controlled hypertension, and chronic smoking was admitted following a 20-meter fall into a well. Initial injuries included lumbar and pelvic fractures, rib fractures with pulmonary contusion, a sutured thigh wound, an open scrotal wound, and necrotic lesions of the left great toe. Ten days after admission, the patient developed progressive trismus, dysarthria, and xerostomia. ENT and maxillofacial evaluations excluded traumatic etiologies. Given the presence of contaminated wounds and a compatible incubation period, a diagnosis of cephalic tetanus was established.



The clinical course was complicated by generalized myoclonic spasms and acute respiratory arrest, necessitating orotracheal intubation, mechanical ventilation, deep sedation, and neuromuscular blockade. The Dakar score was calculated at 2, suggesting a mild form, though respiratory involvement mandated aggressive ICU care.

DISCUSSION

Cephalic tetanus is a rare but particularly severe localized form of tetanus, representing approximately 1–3% of all tetanus cases, and is defined by predominant

cranial nerve involvement following craniofacial trauma or contaminated wounds. Despite its classification as a localized entity, cephalic tetanus is associated with a disproportionately high risk of progression to generalized tetanus and severe respiratory failure, frequently requiring prolonged intensive care management. In contemporary series, mortality remains significant, particularly among elderly patients, those with delayed presentation, and individuals with incomplete or absent vaccination, highlighting the continued clinical relevance of this disease in modern intensive care practice.

The pathophysiology of cephalic tetanus is mediated by tetanospasmin, an extremely potent neurotoxin produced by *Clostridium tetani*. Following inoculation, the toxin binds irreversibly to peripheral motor and autonomic nerve terminals and is transported retrogradely along axons to the central nervous system. At the synaptic level, tetanospasmin cleaves synaptobrevin (vesicle-associated membrane protein), thereby inhibiting the presynaptic release of inhibitory neurotransmitters, primarily gamma-aminobutyric acid (GABA) and glycine. This loss of inhibitory control results in sustained motor neuron hyperexcitability, tonic muscle contraction, and profound autonomic dysregulation. In cephalic tetanus, early involvement of cranial nerve nuclei—most commonly the facial (VII), oculomotor (III), glossopharyngeal (IX), and vagus (X) nerves—accounts for the characteristic presentation with trismus, facial palsy, dysphagia, ophthalmoplegia, and impaired airway protective reflexes.

Cranial nerve involvement has consistently been identified as a marker of severe disease and a strong predictor of progression to generalized tetanus. Multiple observational studies and case series report that up to two-thirds of patients with cephalic tetanus subsequently develop generalized spasms. Bulbar dysfunction, laryngospasm, and rigidity of respiratory musculature significantly increase the risk of acute respiratory failure, aspiration, and sudden airway obstruction. Consequently, cephalic tetanus should be regarded as a high-risk presentation warranting early admission to the intensive care unit and close respiratory monitoring.

Diagnosis of cephalic tetanus is primarily clinical and should not be delayed by laboratory investigations, as microbiological confirmation is frequently negative and lacks sensitivity. The diagnosis should be suspected in any patient presenting with cranial nerve dysfunction, trismus, or dysphagia following head or facial injury, particularly when vaccination status is unknown or incomplete. Prognostic scoring systems have been developed to assist in severity assessment and risk stratification. The Dakar score, although simple and widely used in resource-limited settings, provides only moderate prognostic accuracy. In contrast, the Tetanus Severity Score (TSS) has demonstrated superior predictive value for mortality,

duration of mechanical ventilation, and length of ICU stay across multiple validation studies. Nonetheless, expert consensus emphasizes that these tools should complement, rather than replace, experienced clinical judgment.

Respiratory complications remain the leading cause of mortality in cephalic tetanus. Mechanisms include laryngospasm, chest wall rigidity, impaired cough and secretion clearance, aspiration pneumonia, and autonomic instability leading to hypoventilation or sudden cardiovascular collapse. Several intensive care studies advocate for early elective endotracheal intubation in patients with bulbar involvement or rapidly progressive trismus, as emergency airway management in tetanus is associated with a high rate of complications and mortality. In severe cases, prolonged mechanical ventilation with deep sedation and neuromuscular blockade is often required to suppress spasms and ensure ventilatory synchrony.

Current management of tetanus is based on five core therapeutic principles: neutralization of circulating toxin, eradication of *Clostridium tetani*, control of muscle spasms, management of autonomic dysfunction, and comprehensive supportive intensive care. Human tetanus immunoglobulin (HTIG) is essential for neutralization of unbound toxin and should be administered as early as possible, recognizing that it does not reverse toxin already bound to neural tissue. Surgical wound debridement remains mandatory to eliminate ongoing toxin production. Antimicrobial therapy with metronidazole is currently preferred over penicillin due to equivalent anaerobic coverage and the absence of GABA antagonism; several studies have demonstrated reduced mortality and shorter ICU stays with metronidazole-based regimens.

Control of muscle spasms represents a major therapeutic challenge and often necessitates a multimodal pharmacologic strategy. Benzodiazepines remain first-line therapy owing to their potentiation of GABAergic inhibition. In refractory cases, continuous infusions of sedative agents combined with neuromuscular blocking agents are frequently required. Such strategies, while effective, necessitate prolonged mechanical ventilation and are associated with an increased risk of critical illness myopathy, neuropathy, and nosocomial infections, underscoring the importance of early physiotherapy and rehabilitation planning.

Autonomic dysfunction is a hallmark of severe tetanus and is characterized by labile hypertension, tachyarrhythmias, hyperthermia, diaphoresis, and profound catecholamine surges. Magnesium sulfate has been extensively studied as an adjunctive therapy and has demonstrated efficacy in reducing autonomic instability, decreasing sedative requirements, and improving hemodynamic control. Additional agents, including beta-blockers, clonidine, and dexmedetomidine, have been

used with variable success, although careful titration is essential to avoid cardiovascular collapse.

Early tracheostomy is frequently advocated in patients with severe tetanus requiring prolonged ventilation. Observational studies suggest that early tracheostomy facilitates airway management, reduces sedation requirements, improves pulmonary hygiene, and may shorten the duration of mechanical ventilation and ICU stay, although high-quality randomized data remain limited.

Despite advances in intensive care, cephalic tetanus continues to be associated with significant morbidity and mortality. Poor prognostic factors consistently identified across studies include advanced age, delayed presentation, severe autonomic dysfunction, need for mechanical ventilation, and progression to generalized tetanus. This case underscores the critical importance of early recognition, aggressive airway management, meticulous supportive care, and adherence to evidence-based ICU protocols. It also highlights the enduring public health importance of vaccination and booster programs in preventing this entirely avoidable disease.

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