

Enteral Baclofen for Persistent Stimulus-Induced Rigidity in Severe Neonatal Tetanus: Facilitating Weaning from Prolonged Mechanical Ventilation in a Low-Resource NICU – A Case Report

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Case Report

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Abstract

Background:

Neonatal tetanus is a life-threatening, vaccine-preventable disease that still occurs in low-resource settings, such as Somalia, often following unhygienic umbilical cord care during home deliveries. Management in intensive care is complex, especially when toxin-mediated motor overactivity coexists with suspected seizures and sepsis, and persistent rigidity prevents weaning from mechanical ventilation. This case is notable for the successful use of high-dose enteral baclofen to control refractory rigidity and enable extubation in severe neonatal tetanus in a low-resource neonatal intensive care unit setting.

Case presentation:

An 8-day-old term male neonate born at home to an unimmunized multigravida mother presented with respiratory distress, severe trismus, generalized rigidity, high fever, hemodynamic instability, and recurrent tonic–clonic seizures. Bedside electroencephalography was not performed because transfer was unsafe owing to ventilator dependence. Broad-spectrum antimicrobials, vancomycin and meropenem, were initiated for presumed sepsis, along with a single dose of human tetanus immunoglobulin and scheduled intravenous diazepam for tetanic spasms. Empirical antiepileptic therapy with phenobarbital and levetiracetam was administered for suspected cortical seizures. Over an eight-week intensive care course, fever and clinically apparent seizure-like episodes resolved, allowing for the discontinuation of antiepileptic drugs. However, marked axial and limb hypertonia with stimulus-induced spasms persisted and contributed to two failed extubation attempts, despite improved sepsis markers. Brain magnetic resonance imaging revealed only subtle chronic susceptibility foci without acute lesions. The introduction and careful titration of high-dose enteral baclofen, combined with low-dose oral diazepam and targeted physiotherapy, led to a sustained reduction in rigidity and spasms, successful weaning from intravenous benzodiazepines, extubation to room air, and progression to full oral feeding. The infant was discharged on tapering oral baclofen, with residual lower limb deformities and planned physiotherapy follow-up.

Conclusions:

This case illustrates that in severe neonatal tetanus with persistent rigidity in a low-resource setting, adjunctive high-dose enteral baclofen may help control post-acute muscular hypertonia and facilitate liberation from prolonged mechanical ventilation when advanced monitoring is limited.

1. Background

Neonatal tetanus is a severe but preventable neurological disorder caused by the neurotoxin tetanospasmin, produced by *Clostridium tetani* [1, 2]. The global burden has declined due to maternal immunization and improved clean delivery and umbilical cord care; however, neonatal tetanus remains a major threat in settings with low vaccine coverage and limited access to skilled perinatal care [3, 4]. In 2021, an estimated 7,700 newborns died from neonatal tetanus, an 84% reduction since 2000; however, several countries have not yet eliminated maternal and neonatal tetanus, underscoring persistent gaps in maternal immunization, antenatal care, skilled birth attendance, and hygienic cord-care practices [5].

Neurological manifestations of tetanus arise from the disruption of inhibitory neurotransmission in the central nervous system: tetanospasmin impairs the presynaptic release of γ -aminobutyric acid and glycine, producing motor neuron hyperexcitability, generalized rigidity, and painful muscle spasms [6–8]. In neonates, the disease typically follows a brief period of normal feeding and then progresses to poor suck, generalized stiffness, and stimulus-induced spasms. Severe cases develop dysphagia, laryngospasm, apnea, respiratory failure, and autonomic instability, which often require intensive care and mechanical ventilation [9, 10]. Management includes toxin neutralization, antimicrobial therapy, strict control of neuromuscular activity, ventilatory support, nutritional optimization, and prevention of complications of prolonged critical illness [10, 11].

Persistent rigidity and stimulus-induced spasms, despite improvements in systemic instability, pose major clinical challenges. Spasms may mimic convulsive seizures, especially when electroencephalography is unavailable, and many neonates require deep, prolonged sedation and extended mechanical ventilation, creating a dilemma between suppressing tetanic activity and avoiding complications of long-term sedation and delayed weaning [12–14]. Evidence to guide specific pharmacologic strategies for tetanus-related neuromuscular hyperactivity remains limited [10, 15, 16].

Baclofen, a γ -aminobutyric acid-B receptor agonist, may provide an adjunctive option by enhancing inhibitory neurotransmission and potentially counteracting tetanospasmin-induced neuronal disinhibition [17, 18]. However, data are sparse, derived mainly from older children and adults, and focused on intrathecal rather than enteral administration; therefore, the role of enteral baclofen in neonatal tetanus with prolonged rigidity and ventilator dependence remains poorly defined [18–20].

We report a case of severe neonatal tetanus in Somalia complicated by persistent stimulus-induced spasms and prolonged mechanical ventilation, illustrating the diagnostic and therapeutic challenges in a low-resource neonatal intensive care unit and describing the adjunctive use of enteral baclofen to facilitate control of rigidity and liberation from ventilatory support.

2. Case presentation

2.1 Perinatal and Family History

An 8-day-old term male neonate, born to a 22-year-old gravida 4, para 4 mother, was admitted to the neonatal intensive care unit (NICU) with progressive feeding refusal, generalized rigidity, and recurrent

stimulus-induced spasms. The infant was delivered at home via an uncomplicated spontaneous vaginal delivery assisted by a traditional birth attendant. Antenatal care was minimal, limited to a single ultrasound at 38 weeks of gestation, and the mother had received no tetanus-containing vaccines (zero lifetime doses of tetanus toxoid/tetanus–diphtheria vaccine). There was no reported history of maternal infections or chronic medical illnesses, and the previous three children were healthy.

2.2 Umbilical Care and Early Postnatal Course

The umbilical cord was cut with a non-sterile sharp instrument and tied with an unclean thread on day 0 of life. In the days following birth (days 1–3 of life), the umbilical stump developed foul-smelling purulent discharge with surrounding erythema and edema, but no medical care was sought. The infant was breastfed normally until day 4 of life, when the parents noted excessive crying and episodes of stiffness triggered by handling and loud noises. By day 6, according to the parents, the infant developed difficulty opening his mouth, compatible with trismus, leading to progressive difficulty with latching and suckling. On day 7, the spasms became severe and paroxysmal, associated with marked rigidity and cyanosis, prompting emergency presentation and NICU admission on day 8 of life.

2.3 Examination on Admission

On admission, the neonate appeared acutely ill and had significant respiratory distress. Anthropometric measurements showed a weight of 2.75 kg (10th–25th percentile), length of 49 cm, and a head circumference of 33.5 cm. Vital signs revealed hyperpyrexia (rectal temperature 38.9°C), tachycardia (heart rate 176 beats/min), and tachypnea (respiratory rate 72 breaths/min) with severe intercostal and subcostal retractions. Blood pressure was 82/52 mmHg, and oxygen saturation was 83% on room air, frequently dropping below 70% during spasms.

Neurological examination revealed the classic features of severe neonatal tetanus (Grade III/Score IV). The infant was in a hyperextended posture with profound axial and appendicular rigidity. Craniofacial findings included marked facial muscle spasms (risus sardonicus), tightly closed eyelids, a deeply furrowed brow, and severe trismus (“lockjaw”), with a complete absence of sucking and rooting reflexes. Sustained rigidity involved the neck, trunk, and limbs, producing pronounced opisthotonos, clenched fists, and wrist flexion. The deep tendon reflexes were brisk, with bilateral ankle clonus. Minimal tactile, acoustic, or light stimulation reproducibly provoked painful generalized tonic spasms lasting 15–30 seconds, leading to transient chest wall fixation, cyanosis, and profound desaturation.

Cardiorespiratory and abdominal examinations confirmed an advanced systemic compromise. Respiratory assessment revealed markedly increased work of breathing with rigid thoracoabdominal motion and equal but strained air entry. Cardiovascular examination revealed persistent tachycardia with normal heart sounds, no murmurs, bounding peripheral pulses, and a capillary refill time of 2 seconds. The abdomen was board-like and diffusely rigid. The umbilical stump was unhealed and necrotic with periumbilical erythema and edema and active, foul-smelling purulent discharge, consistent with an infected portal of entry.

2.4 Initial Management and Intensive Care

Given severe respiratory distress and recurrent stimulus-induced spasms, the infant was emergently intubated on day 8 of life (day 1 of admission) and commenced on mechanical ventilation in synchronized intermittent mandatory ventilation mode. Disease-specific therapy included a single intramuscular dose of 500 IU human tetanus immunoglobulin to neutralize unbound toxin and broad-spectrum intravenous antimicrobials with meropenem (180 mg/day, administered as 60 mg every 8 hours; for 14 days) and vancomycin (90 mg/day, administered as 30 mg every 8 hours; for 14 days) to eradicate *Clostridium tetani* and treat potential concomitant sepsis. Oral feeding was initially withheld because of severe abdominal distension and respiratory compromise; enteral nutrition was later reintroduced via an orogastric tube as the abdominal findings improved.

From a neurological standpoint, the infant exhibited both classical tetanic spasms and distinct generalized tonic-clonic convulsive episodes with marked autonomic instability during weeks 1–3 of admission. Bedside electroencephalography was not feasible because of critical ventilator dependence and hemodynamic fragility. Therefore, empirical antiepileptic therapy with oral phenobarbital (16 mg/day, administered as 8 mg every 12 hours via nasogastric tube; maintained for 3 weeks) and intravenous levetiracetam (60 mg/day, administered as 30 mg every 12 hours; maintained for 3 weeks) was initiated to treat suspected cortical seizures, while continuous intravenous ketamine infusion (60 mg/day, maintained for 5 days) and intermittent intravenous diazepam (1.0 mg/day, administered as 0.5 mg every 12 hours; maintained for 4 weeks) were titrated to control tetanus-induced rigidity and spasms and to facilitate mechanical ventilation.

2.5 Investigations

Serial laboratory assessments, including inflammatory markers and blood cultures, were performed to evaluate sepsis, guide therapy, and monitor treatment response. Microbiological culture of the purulent umbilical stump swab identified the presence of *Staphylococcus aureus* and anaerobic bacteria. The laboratory values presented in (Table 1) were obtained at the time of admission. Given the prolonged critical course and repeated failed extubation attempts, neuroimaging was performed. Contrast-enhanced brain MRI revealed linear low-signal susceptibility foci along the periphery of the occipital horn of the left lateral ventricle, consistent with chronic calcific or hemosiderin deposition, without evidence of acute parenchymal pathology. The cerebral hemispheres, basal ganglia, thalami, and brainstem were otherwise structurally unremarkable, with no diffusion restriction or abnormal contrast enhancement noted.

Table 1
Baseline Laboratory Parameters at NICU Admission.

Parameter	Patient Value	Reference Range
Hemoglobin	17.7 (g/dL)	11.7–18.0 (g/dL)
WBCs	20 390 (cells/ μ L)	4 000–10 000 (cells/ μ L)
Absolute Neutrophil	14 880 (cells / μ L)	2 000–7 000 (cells / μ L)
Absolute Lymphocyte	3 020 (cells / μ L)	800–4 000 (cells / μ L)
Absolute Monocyte	2 400 (cells / μ L)	120–1 120(cells / μ L)
Absolute Eosinophil	10 (cells / μ L)	20–500 (cells / μ L)
Platelets	319 000 (cells/ μ L)	150 000–450 000 (cells/ μ L)
MPV	12.4 (fL)	7.8–11.0 (fL)
CRP	23.2 (mg/dL)	< 6.0 (mg/dL)
Creatinine	0.74 (mg/dL)	0.35–1.10 (mg/dL)
Urea	108 (mg/dl)	10–45 (mg/dl)
SGPT	18 (UI/L)	0–45 (UI/L)
SGOT	112 (UI/L)	0–35 (UI/L)
Total Bilirubin	5.58 (mg/dL)	0.30–1.35 (mg/dL)
Ionized Calcium (cCa ²⁺)	1.39 (mmol/L)	1.10–1.35 (mmol/L)
Sodium (Na ⁺)	151 (mEq/L)	135–150 (mEq/L)
Potassium (K ⁺)	5.65 (mmol/L)	3.5–5.5 (mEq/L)
Blood Glucose	73.8 (mg/dL)	60–100 (mg/dL)
Blood pH	7.346	7.35–7.45
$\backslash \text{varvec}p \backslash \text{varvec}C \backslash \text{varvec}O_2$	24.5 (mmHg)	35–45 (mmHg)

Abbreviations: **WBC:** white blood cell count; **MPV:** mean platelet volume; **CRP:** C-reactive protein; **SGOT/AST:** serum glutamic oxaloacetic transaminase/aspartate aminotransferase; **SGPT/ALT:** serum glutamic pyruvic transaminase/alanine aminotransferase; **pCO₂:** Partial Pressure of Carbon Dioxide.

Parameter	Patient Value	Reference Range
Bicarbonate (HCO_3^-)	13.0 (mmol/L)	21–28 (mmol/L)
Base Excess (cBase)	-11.6 (mmol/L)	-2.0 to + 2.0 (mmol/L)
Blood Lactate	5.9 (mmol/L)	0.30–1.35 (mg/dL)
Blood culture	Negative	
Umbilical stump culture	<i>Staphylococcus aureus</i> and anaerobic bacteria isolated	
Abbreviations: WBC: white blood cell count; MPV: mean platelet volume; CRP: C-reactive protein; SGOT/AST: serum glutamic oxaloacetic transaminase/aspartate aminotransferase; SGPT/ALT: serum glutamic pyruvic transaminase/alanine aminotransferase; pCO2: Partial Pressure of Carbon Dioxide.		

2.6 Clinical Course

Over the ensuing 3–4 weeks of intensive care, fever and clinically overt generalized tonic-clonic seizures resolved completely. Anti-epileptic medications were gradually tapered and ultimately discontinued without seizure recurrence. Despite systemic stabilization and normalization of laboratory indices, the infant continued to experience persistent axial and limb spasms, which progressively resulted in fixed axial and limb deformities with a clinical appearance resembling cerebral palsy.

Two separate attempts at ventilator weaning and extubation were unsuccessful in the patient. Both trials were complicated by upper airway stiffness, chest wall hypertonia, and glottic spasm shortly after extubation, necessitating reintubation. To mitigate potential laryngeal edema and bronchial hyperreactivity associated with prolonged intubation, intravenous dexamethasone (0.75 mg) was administered prior to subsequent weaning attempts.

Because persistent hypertonia and stimulus-induced spasms remained the principal barriers to ventilator liberation, the antispasticity regimen was optimized during weeks 4–7 of admission. High-dose enteral baclofen was initiated via nasogastric tube (6 mg/day, administered as 2 mg every 8 hours), alongside a maintenance schedule of low-dose oral diazepam (1.0 mg/day, administered as 0.5 mg every 12 hours; maintained for 2 weeks) with the aim of reducing neuromuscular rigidity without precipitating central respiratory depression. Following the initiation of baclofen, there was a rapid and sustained reduction in axial and limb hypertonia, with marked attenuation of stimulus-triggered spasms.

2.7 Outcome and Follow-up

After improved control of rigidity, the infant was successfully extubated to continuous positive airway pressure and weaned to room air without supplemental oxygen in week 7 of admission. Enteral feeds were progressively advanced from orogastric tube feeding to full oral bottle feeding and breastfeeding. At discharge, approximately eight weeks after admission, the patient was afebrile, hemodynamically

stable, breathing spontaneously in room air, and tolerating full oral nutrition. Neurologically, he demonstrated marked recovery, with resolution of opisthotonos and generalized tonic–clonic seizures, although a degree of residual axial and limb hypertonia persisted, showing gradual improvement. He was discharged on a tapering regimen of oral baclofen and scheduled for outpatient follow-up, including physical therapy and continued neurological assessment (Fig. 1). During follow-up, adherence was ensured through nurse-administered dosing and caregiver instruction, and tolerability was monitored clinically without any significant adverse effects. No adverse or unanticipated events related to baclofen or other interventions were observed. The timeline of clinical presentation, diagnostic events, and management is summarized in Table 2.

Table 2

Timeline of clinical presentation, diagnostic events, and management in this episode of care.

Clinical Phase	Time frame / Day of Life	Clinical Presentation & Diagnostic Events	Therapeutic Interventions & Management
Prenatal & Birth	Day 0 (Birth)	Born via spontaneous vaginal delivery at home assisted by a traditional birth attendant; unimmunized mother with zero lifetime tetanus toxoid doses and minimal antenatal care.	Umbilical cord was cut with a non-sterile sharp instrument and tied with an unclean thread; no immediate medical care was sought.
Asymptomatic Period	Days 1–3 of life	Breastfed normally with no reported feeding difficulties or abnormal movements	No specific medical interventions; routine home care only
Symptom Onset	Days 4–6 of life	Excessive crying and generalized stiffness triggered by stimuli, trismus with poor latching and suckling, and a malodorous umbilical stump with purulent discharge, erythema, and oedema.	Home observation; no medical treatment received.
Emergency Presentation & NICU Admission	Day 8 of life (severe spasms began Day 7)	Acute respiratory distress, severe trismus, generalized rigidity, hyperpyrexia, severe tachycardia, cyanosis, and overt GTC seizure episodes, with elevated inflammatory markers. Clinical findings were consistent with severe neonatal tetanus.	Emergency intubation and mechanical ventilation in SIMV mode; single IM dose of HTIG; IV vancomycin and meropenem; phenobarbital and levetiracetam; IV diazepam and ketamine.
Acute NICU Phase & Stabilization	Weeks 1–3 of admission	Persistent stiffness and stimulus-induced spasms; abdominal distension gradually improved; bedside EEG unfeasible due to critical ventilator dependence. Umbilical stump culture grew <i>Staphylococcus aureus</i> and anaerobic bacteria	Mechanical ventilation and sedation continued. Nutrition initiated and advanced via OG tube.
Resolution of Clinical Seizures	Weeks 3–4 of admission	Fever and clinical GTC seizures resolved completely; persistent axial and limb hypertonia/spasms remained.	Systematic tapering and complete discontinuation of AEDs (Phenobarbital and Levetiracetam).
Persistent Spasms, Failed Extubation & Targeted Therapy	Weeks 4–7 of admission	Persistent spasms caused fixed axial and limb deformities resembling cerebral palsy. Two failed extubation attempts due to upper-airway stiffness, chest-wall hypertonia, and glottic spasm; contrast-enhanced brain MRI showed chronic susceptibility foci along the periphery of the left occipital horn,	Re-intubation after failed trials; IV Dexamethasone bridge therapy introduced prior to extubation attempts to reduce laryngeal edema. Parenteral sedatives were progressively reduced. Addition and incremental titration of

Clinical Phase	Time frame / Day of Life	Clinical Presentation & Diagnostic Events	Therapeutic Interventions & Management
		compatible with calcific or hemosiderin deposition, without acute parenchymal pathology. High-dose oral Baclofen combined with low-dose Diazepam rapidly and sustainably reduced hypertonia and spasms.	oral Baclofen combined with low-dose oral Diazepam.
Extubation & Oral Feeding Progression	Late NICU course (prior to Week 8)	Successful extubation to continuous positive airway pressure followed by weaning to room air without supplemental oxygen; resolution of opisthotonos and generalized tonic-clonic seizures, with residual axial and limb hypertonia.	Progressive transition from OG tube feeds to full oral bottle-feeding and breastfeeding.
Discharge & Follow-Up	Week 8 of admission	Afebrile and stable on room air, tolerating full oral feeds, with improving residual hypertonia and no significant adverse effects.	Discharged on tapering oral baclofen with physical therapy and neurological follow-up; adherence and tolerability were monitored.

Abbreviations: AED, anti-epileptic drug; CPAP, continuous positive airway pressure; EEG, electroencephalography; GTC, generalized tonic–clonic; IV, intravenous; MRI, magnetic resonance imaging; NICU, neonatal intensive care unit; OG, orogastric; SIMV, synchronized intermittent mandatory ventilation; TIG, tetanus immunoglobulin.

3. Discussion and Conclusions

Neonatal tetanus remains a largely preventable cause of neonatal mortality, disproportionately affecting settings with gaps in maternal immunization, antenatal care, hygienic delivery, and cord-care practices. Global initiatives have driven a substantial reduction in neonatal tetanus deaths since 2000; however, recent analyses of maternal and neonatal tetanus elimination efforts indicate that progress has stalled or reversed in several high-burden countries, particularly those with fragile health systems and recurrent disruptions to vaccination and maternal health services [2, 5]. This case from Somalia exemplifies how such systemic weaknesses can culminate in a life-threatening but avoidable infection in a resource-limited neonatal intensive care context.

Clinically, the evolution from an initial period of normal feeding to progressive poor suck, generalized rigidity, and stimulus-induced spasms is characteristic of neonatal tetanus as defined by the World Health Organization [5]. Severe forms frequently require intensive care, with airway protection, deep sedation, and prolonged mechanical ventilation to control spasms, prevent respiratory failure, and manage autonomic instability. The markedly prolonged ventilator dependence in this infant, despite resolution of sepsis parameters and negative blood cultures, underscores how toxin-mediated neuromuscular hyperactivity can dominate the clinical course even after control of the infectious focus,

particularly where advanced neuromonitoring is unavailable and titration of sedative and antispastic regimens must rely on clinical observation alone [21].

Reports from low-resource settings emphasize that case fatality in neonatal and pediatric tetanus remains high where access to invasive ventilation, neuromuscular blockade, and continuous monitoring is constrained [22, 23]. In such contexts, clinicians commonly rely on combinations of benzodiazepines, barbiturates, magnesium sulfate, and other agents to manage spasms and autonomic dysfunction, with variable success, and improvise prolonged critical care strategies within a constrained infrastructure [21, 22]. Our experience adds to the literature by detailing the complexities of long-term ventilatory support in a low-resource neonatal ICU, including repeated failed extubation attempts driven by persistent rigidity and chest-wall involvement despite apparent systemic stabilization.

Distinguishing tetanic spasms from epileptic seizures in neonates is particularly challenging in the absence of electroencephalography, leading many teams to initiate broad antiepileptic therapy in parallel with sedatives and muscle relaxants administration. Contemporary critical care descriptions of tetanus management highlight multimodal control of neuromuscular hyperactivity and autonomic storms; however, evidence to guide specific pharmacologic approaches to tetanus-related rigidity in neonates remains sparse [21, 23]. In this setting, the use of enteral baclofen as adjunctive antispastic therapy was individualized, aiming to enhance inhibitory neurotransmission and facilitate weaning from deep sedation and mechanical ventilation; however, its optimal dosing, timing, and long-term neurodevelopmental impact in neonatal tetanus require further systematic evaluation.

From a public-health perspective, every episode of neonatal tetanus signals a cascade of preventable failures: lack of adequate tetanus-toxoid-containing vaccination among women of reproductive age, insufficient antenatal care, and unhygienic delivery and cord-care practices. The WHO and partner agencies stress that sustaining and expanding maternal immunization, maintaining clean delivery and cord-care practices, and closely monitoring high-risk districts are essential to prevent the resurgence of maternal and neonatal tetanus, particularly following disruptions such as the COVID-19 pandemic and other humanitarian crises [2, 5]. UNICEF and global partners similarly emphasize that eliminating neonatal tetanus requires integrated strategies that combine robust immunization programs with improved access to skilled birth attendants and strengthened community awareness of safe umbilical cord care [5]. Our report reinforces that, even when intensive care can occasionally salvage severely affected neonates, prevention through sustained vaccination coverage and safe birth practices remains the most effective, equitable, and context-appropriate strategy.

This single-center case report cannot establish a causal link between enteral baclofen and clinical improvement, as recovery likely reflects the combined effects of toxin clearance and comprehensive intensive care. Distinguishing tetanic spasms from epileptic seizures is challenging in the absence of electroencephalography, which may have influenced the interpretation of the treatment response. Long-term neurodevelopmental outcomes were not systematically assessed; therefore, subtle sequelae may

have been missed. Finally, findings from one neonatal intensive care unit in a resource-limited setting may not be generalizable to other contexts.

This case highlights that survival with encouraging early neurological recovery is achievable in severe neonatal tetanus through timely diagnosis, meticulous respiratory and hemodynamic support, and structured spasm control in a resource-limited setting. The use of high-dose enteral baclofen as adjunctive antispastic therapy appeared to facilitate weaning from prolonged mechanical ventilation in the context of persistent rigidity, suggesting a potential role for carefully monitored, individualized use in similar challenging cases.

Patient perspective

As the parents of this baby, we were terrified when he became stiff, stopped feeding, and needed prolonged intensive care and a ventilator, and we often feared he would not survive. We are deeply grateful that his spasms improved with treatment, allowing him to breathe on his own again, and we now understand how important maternal tetanus vaccination and clean delivery practices are to prevent this in other newborns.

Abbreviations

AEDs

Anti-Epileptic Drugs

CPAP

Continuous Positive Airway Pressure

EEG

Electroencephalography

GTC

generalized tonic–clonic

HTIG

Human Tetanus Immunoglobulin

IM

Intramuscular

IV

Intravenous

MRI

Magnetic Resonance Imaging

NICU

Neonatal Intensive Care Unit

OG

Orogastric

SIMV

Synchronized Intermittent Mandatory Ventilation

UNICEF

United Nations Children's Fund

WHO

World Health Organization

Declarations

Ethics Approval and Consent to Participate

Ethical approval for individual case reports is not required at Mogadishu Somali Türkiye Recep Tayyip Erdoğan Training and Research Hospital; therefore, no institutional review board approval or reference number was obtained. Written informed consent for participation was obtained from the patient's legal guardian.

Consent for publication

Written informed consent for publication of this case report and the accompanying clinical details and images was obtained from the patient's legal guardian. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Clinical Trial Number

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

Abbreviations

AED, anti-epileptic drug; CPAP, continuous positive airway pressure; EEG, electroencephalography; GTC, generalized tonic-clonic; IV, intravenous; MRI, magnetic resonance imaging; NICU, neonatal intensive care unit; OG, orogastric; SIMV, synchronized intermittent mandatory ventilation; TIG, tetanus immunoglobulin.

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The authors have nothing to report.

Author Contribution

F.H.A. conceptualized and designed the study, coordinated the investigation, performed data curation and formal analysis, and wrote the original draft of the manuscript. M.Y.A. co-conceptualized the study, performed advanced data analysis and interpretation, supervised the project workflow, and critically wrote and revised the original manuscript. I.M.O., K.A.A., N.A.O., A.A.A., A.M.A., M.A.R., N.A.A., J.H.H., and F.A.F. contributed to the investigation, data collection, and literature review, and participated in writing, reviewing, and editing the manuscript. All authors read and approved the final version of the manuscript.

Data Availability Statement

Not applicable.

References

1. Li J, Liu Z, Yu C, Tan K, Gui S, Zhang S, et al. Global epidemiology and burden of tetanus from 1990 to 2019: a systematic analysis for the Global Burden of Disease Study 2019. *Int J Infect Dis.* 2023;132:118–26.
2. Yusuf N, Raza AA, Chang-Blanc D, Ahmed B, Hailegebriel T, Luce RR, et al. Progress and barriers towards maternal and neonatal tetanus elimination in the remaining 12 countries: a systematic review. *Lancet Glob Health.* 2021;9(11):e1610–e1617.
3. Sudarshan R, Sayo AR, Renner DR, de Saram S, Godbole G, Warrell C, et al. Tetanus: recognition and management. *Lancet Infect Dis.* 2025;25(11):e645-e657.
4. Mengesha MB, Weldegeorges DA, Assefa NE, Gebremeskel SG, Hidru HD, Teame H, et al. Tetanus toxoid immunization status and associated factors among mothers in Hawzen, Eastern Zone of Tigray, Ethiopia, 2019. *Open Public Health J.* 2020;13:281–8.
5. World Health Organization. Tetanus. <https://www.who.int/news-room/fact-sheets/detail/tetanus>. Accessed 13 August 2026.
6. Megighian A, Pirazzini M, Fabris F, Rossetto O, Montecucco C. Tetanus and tetanus neurotoxin: from peripheral uptake to central nervous tissue targets. *J Neurochem.* 2021;158(6):1244–53.
7. Rao Y, Xu X, Chen L, Wang D, Liu L, Jiang T, et al. Delirium in tetanus patients: a review of clinical features, pathophysiology, and integrated management strategies. *Ann Med.* 2025;57(1):2543980.
8. Abdulkadir AM, Abdullahi IM, Hassan FA, Elmi AH. Generalized tetanus in an unvaccinated adolescent: a case report on successful management in a resource-limited setting. *Int Med Case Rep J.* 2026;19:1–5.

9. Hajaj H, Bahari H, Ayyad A, Messaoudi S, Amrani R. Neonatal tetanus still exists: a case report and review of literature. *Cureus*. 2024;16(5):e61410.
10. Onyeje CK, Onyeje BT. Successful management of neonatal tetanus with autonomic manifestations at a referral hospital in Nigeria: a case report. *BMC Pediatr*. 2025;25:846.
11. Bacha T, Abayneh M, Tefera DB. Neonatal tetanus case series: a tale of survival and tragedy at St Paul Hospital Millennium Medical College, Addis Ababa, Ethiopia. *Pediatr Health Med Ther*. 2024;15:257–64.
12. Brook I. Neonatal tetanus. *Pediatr Emerg Med J*. 2021;8(1):1–7.
13. Médecins Sans Frontières. Tetanus. <https://medicalguidelines.msf.org/en/viewport/CG/english/tetanus-16689919.html>. Accessed 19 August 2026.
14. Lequain H, Richard-Mornas A, Argaud L, Dargent A. Tetanus-induced rhythmic seizures mimicking the clinical and electroencephalographic presentation of status epilepticus. *BMJ Case Rep*. 2022;15(9):e250645.
15. Rodrigo C, Fernando D, Rajapakse S. Pharmacological management of tetanus: an evidence-based review. *Crit Care*. 2014;18(2):217.
16. Karnad DR, Gupta V. Intensive care management of severe tetanus. *Indian J Crit Care Med*. 2021;25(Suppl 2):S155–S160.
17. Boer M, de Voogd M, Niemeijer ND, van Hoeven L. Tetanus—a case report highlighting the challenges in diagnosis and treatment. *Trop Dis Travel Med Vaccines*. 2024;10:10.
18. Silver EM, Rianprakaisang T, Thornton SL, Ganatra HA. A pediatric case of generalized tetanus treated with intrathecal baclofen. *Toxicol Commun*. 2024;8(1):2404194.
19. Yen LM, Thwaites CL. Tetanus. *Lancet*. 2019;393(10181):1657–68.
20. Şık G, Aydoseli A, Çıtak A. Intrathecal baclofen use in the management of tetanus related spasm: a case report. *Turk J Pediatr*. 2019;61(1):126–9.
21. Abubakar B, Dunga J, Jibrin YB, Maina H, Buma BG, Maigari I. ICU management of tetanus. In: Shaikh N, Aslanidis T, editors. *ICU management and protocols*. London: *IntechOpen*; 2022. p. 1–14.
22. Nakubulwa C, Opio E, Alekat GS, Kibetenga M, Alaroker FOE. Neonatal tetanus with good outcomes at a regional referral hospital in Eastern Uganda: a case report. *J Med Case Rep*. 2022;16:54.
23. Bhagwat B, Sarangi BU, Walimbe A, Ramtenki R. Intensive care management and outcomes of severe paediatric tetanus in a tertiary care hospital. *Trop Doct*. 2026;56(1):135–9.

Figures



Figure 1

Post-treatment clinical photograph taken at the time of discharge following enteral Baclofen therapy and physical therapy, demonstrating complete resolution of facial spasticity, open eyes, full recovery of oral feeding capacity, and persistent mild lower extremity musculoskeletal deformity scheduled for outpatient physical therapy.