

ORIGINAL ARTICLE

Cardiorespiratory Trajectory, ROX Index Extubation Failure, and Post-Tracheostomy Liberation in Pediatric Odontogenic Ablett Grade IV Tetanus Complicated by Cardiac Arrest: An Observational Analytic Case Study

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ARTICLE INFO	ABSTRACT
ARTICLE TYPE Original Article ARTICLE HISTORY Received: August 19, 2026 Revised: September 15, 2026 Accepted: October 10, 2026 Available online: October 27, 2026 Published: October 30, 2026 KEYWORDS Ablett Grade IV tetanus; Odontogenic infection; Pediatric intensive care; ROX index; Surgical tracheostomy CORRESPONDING AUTHOR Indra Ihsan E-mail: indraihsan@yahoo.co.id DOI 10.59345/sjped.v4i2.376 LICENSE CC BY-NC-SA 4.0 COPYRIGHT © 2026 The Author(s). Published by Phlox Institute: Indonesian Medical Research Organization.	Background: Ablett Grade IV tetanus from an odontogenic portal carries high mortality in resource-limited settings because of laryngospasm, autonomic instability and prolonged ventilation. Objective: To describe the cardiorespiratory course, extubation failure by the Respiratory Rate-Oxygenation (ROX) index, and post-tracheostomy liberation in an unimmunized, severely undernourished child with odontogenic Grade IV tetanus. Methods: This single-patient case report followed a boy through a 20-day hospital course within 37 days of follow-up (October 1 to November 6, 2024) at Dr. M. Djamil General Hospital, Padang, Indonesia (ethics protocol CMHC/EC/IRB/2026/0418). Oxygenation indices, the ROX index, the neutrophil-to-lymphocyte ratio (NLR) and the systemic immune-inflammation index (SII) were calculated from serial records. Results: The unimmunized boy, aged 4 years 2 months, weighed 11.0 kg (weight-for-age Z-score -3.0 SD) and had rampant dental caries, 1.0 cm trismus, opisthotonus, aspiration bronchopneumonia and hypertension (130/85 mmHg). On Day 2, reflex spasms rose to 10–12 episodes/day (NLR 7.36; SII $3,946.9 \times 10^3/\mu\text{L}$), followed by apneic cardiac arrest (SpO₂ 80%, PaO₂/FiO₂ 106.7 mmHg). Extubation to high-flow nasal cannula on Day 7 failed within 2 hours (SpO₂ 82%, ROX index 2.2). After meropenem for ceftriaxone-resistant <i>Pseudomonas</i> sp. bacteremia and elective surgical tracheostomy on Day 9, he was weaned to a T-piece within 24 to 48 hours and continuous sedatives were stopped, and enteral baclofen was started. Spasms had resolved by Day 19; decannulation was scheduled for Day 39. Conclusion: In this child, surgical tracheostomy after ROX-documented extubation failure was followed by rapid ventilator liberation and sedative de-escalation. Causation cannot be shown.

1. Introduction

Tetanus remains a life-threatening neuromuscular and dysautonomic infectious emergency. It is caused by *Clostridium tetani*, a spore-forming, obligate anaerobic Gram-positive bacillus.^{1,2} Global immunization campaigns using diphtheria-tetanus-pertussis (DTP) vaccines have cut worldwide tetanus mortality from approximately 275,000 deaths in 1990 to 34,684 deaths in 2019. The disease persists nonetheless. It still causes a disproportionate burden of preventable pediatric morbidity and mortality across low- and middle-income countries (LMICs).^{1–3} Global burden analyses covering 1990 to 2021 show that incidence among children under five years of age fell to 17,788 annual cases (mortality rate 1.34 per 100,000). In resource-constrained tropical regions, however, incidence remains up to 135-fold higher than in high-income nations, and inpatient case-fatality rates range between 20.0% and 45.0%.^{2–4} Hospital-based surveillance in Indonesia recorded a 33.9% case-fatality rate among tetanus hospitalizations (38 deaths vs. 74 survivors out of 112 cases). Of these patients, 98.2% had incomplete or unknown immunization histories. World Health Organization (WHO) country surveillance recorded 391 pediatric and neonatal tetanus notifications nationwide in 2019.^{4,5} Several factors add to the problem. Sociocultural barriers, vaccine hesitancy, reliance on non-medical traditional practitioners, and incomplete primary DTP series completion leave pockets of

preschool children with no protective anti-tetanus toxoid immunoglobulin G (IgG) titers ($> 0.01 \text{ IU/mL}$).^{6,7}

C. tetani spores classically inoculate devitalized soft tissues through penetrating lower-extremity puncture wounds, lacerations, burns, or unsterile umbilical cord care. Yet approximately 5.9% to 7.0% of clinical tetanus cases present without any identifiable external cutaneous trauma.^{8,9} In such cryptogenic presentations, the oral and maxillofacial cavity is a frequently overlooked, occult portal of entry.^{8,9} Epidemiological syntheses of oral and odontogenic tetanus indicate that 73.0% of intraoral cases follow dental manipulation or extraction. Another 23.0% arise spontaneously from deep carious lesions (caries dentis), necrotic pulp chambers, or periodontal abscesses.^{8,9} In unimmunized children with poor oral hygiene, deep carious cavities create an ideal anaerobic microenvironment with low oxidation-reduction potential and abundant necrotic cellular debris. Dormant *C. tetani* spores can germinate there into vegetative bacilli. These bacilli elaborate two principal exotoxins: tetanolysin, which accelerates local tissue necrosis, and tetanospasmin, a potent 150-kDa zinc-dependent metalloprotease neurotoxin.^{8–10} Odontogenic inoculation occurs in immediate anatomical proximity to the trigeminal (cranial nerve V), facial (cranial nerve VII), glossopharyngeal (cranial nerve IX), and vagal (cranial nerve X) motor nuclei. Retrograde axonal transport to the brainstem therefore covers a shorter distance. The progression from isolated trismus (lockjaw) and pharyngeal dysphagia to generalized

axial rigidity and life-threatening laryngospasm is hastened.^{9,10}

Tetanospasmin is cleaved into two chains. The 100-kDa heavy chain mediates high-affinity binding to polysialogangliosides (GT1b and GD1b) on presynaptic alpha-motor nerve terminals. The 50-kDa catalytic light chain undergoes dynein-driven retrograde axonal transport at 1.0 to 3.0 mm/hour to central nervous system inhibitory interneurons.^{10,11} Within spinal Renshaw cells and brainstem inhibitory nuclei, the zinc metalloprotease light chain selectively cleaves vesicle-associated membrane protein-2 (VAMP-2 / synaptobrevin-2) at the Gln⁷⁶-Phe⁷⁷ peptide bond. This abolishes the soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) complex that synaptic vesicle exocytosis of gamma-aminobutyric acid (GABA) and glycine requires.^{10,11} Postsynaptic inhibition is lost. Alpha- and gamma-motor neuron firing goes unopposed. Clinically this appears as sustained masseter spasm (trismus), risus sardonius, board-like abdominal rigidity, paraspinal opisthotonus, and violent reflex tetanic spasms triggered by minimal tactile or auditory stimuli.^{11,12} When retrograde trans-synaptic migration extends to the preganglionic sympathetic neurons of the intermediolateral cell column (thoracolumbar segments T1 to L2), loss of central GABAergic restraint precipitates Ablett Grade IV tetanus. This grade is defined by paroxysmal sympathetic hyperactivity (autonomic storm), labile Stage 2 hypertension, sinus tachycardia, hyperpyrexia, and profuse catecholamine surges that predispose to myocardial exhaustion and sudden cardiac arrest.^{12,13}

Airway and ventilatory management in pediatric Ablett Grade IV tetanus is a major intensive care challenge.¹²⁻¹⁴ Nearly 80.0% of patients with severe generalized tetanus require invasive mechanical ventilation for a median duration exceeding three weeks. The reasons are recurrent laryngospasm, diaphragmatic rigidity, copious sialorrhea, and the deep pharmacological sedation needed to suppress tetanic convulsions.¹²⁻¹⁴ Prolonged endotracheal intubation in a child with severe trismus and active spasms frequently provokes reflex glottic adduction, tube biting or dislodgement, and impaired mucociliary clearance. It also brings nosocomial ventilator-associated pneumonia (VAP) or bloodstream infection with multidrug-resistant Gram-negative bacilli such as *Pseudomonas aeruginosa*.^{14,15} Cohort investigations suggest that early surgical tracheostomy reduces VAP incidence and accelerates ventilator weaning compared to prolonged translaryngeal intubation. Quantitative longitudinal analyses remain scarce in the global pediatric literature. They would integrate objective extubation failure indices, such as the Respiratory Rate-Oxygenation (ROX) index, with multi-receptor sedative de-escalation and nutritional rehabilitation in severely undernourished preschool children with odontogenic Grade IV tetanus.^{15,16}

This report describes, in daily detail, 20 inpatient days within 37 days of total clinical surveillance. The patient was an unimmunized, severely wasted (weight-for-age Z-score -3.0 SD) 4-year-2-month-old boy with odontogenic Ablett Grade IV tetanus. He survived apneic cardiac arrest and failed translaryngeal extubation to high-flow nasal cannula (HFNC) with an objective ROX index of 2.2. He also developed ceftriaxone-resistant *Pseudomonas* sp. bacteremia. Even so, he was liberated from positive-pressure mechanical ventilation within 24 to 48 hours of Day 9 surgical tracheostomy, and continuous intravenous sedatives were stopped with enteral baclofen cross-

titration. The primary aim of this case report is to evaluate the quantitative cardiorespiratory, hematologic-inflammatory, neuro-sedative, and nutritional trajectories across eight clinical epochs. The intent is to describe the airway and critical care course and to discuss its implications for severe pediatric tetanus in resource-limited tertiary referral settings.

2. Methods

Study Design

This was a hospital-based, single-subject (n = 1), longitudinal case report with dense serial time-series tracking. It was conducted in the Pediatric Intensive Care Unit (PICU) and Pediatric High Care / Ward of Dr. M. Djamil General Hospital, Faculty of Medicine, Universitas Andalas, Padang, West Sumatera, Indonesia. Follow-up ran from October 1, 2024 (Hospital Day 1, T0) through outpatient polyclinic surveillance on November 6, 2024 (Day 37, T8). The report follows the Consensus-based Clinical Case Reporting (CARE) guidelines.

Ethics Governance

Formal ethical approval was granted by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number 2024/0418). It covered collection, longitudinal analysis, and publication of anonymized clinical, radiological, laboratory, and anthropometric chart data. Institutional academic supervision came from the Department of Child Health, Faculty of Medicine, Universitas Andalas, and the Department of Pediatric, Dr. M. Djamil General Hospital, Padang, West Sumatera, Indonesia. Written informed consent was obtained from the patient's legal guardians (both parents) prior to surgical tracheostomy, intensive care procedures, and manuscript preparation. All study procedures complied with the ethical principles of the Declaration of Helsinki (2013 Fortaleza revision). They also complied with the International Committee of Medical Journal Editors (ICMJE) privacy directives. These included permanent solid black bar anonymization over all patient identifying headers and superior craniofacial/orbital borders on thoracic radiographs.

Participant Eligibility

The study participant was a 4-year-2-month-old (50-month-old) male preschool child. He was referred from RSUD Dr. Adnaan WD Payakumbuh to Dr. M. Djamil General Hospital on October 1, 2024. Inclusion required fulfillment of the World Health Organization clinical case definition for generalized tetanus. This definition covers acute onset of hypertonia, painful muscular contractions of the masseter and cervical muscles, and generalized spasms without other apparent medical cause. Cases were stratified as Ablett Grade IV (very severe generalized tetanus characterized by severe trismus, generalized spasticity, prolonged reflex spasms, apneic spells, tachypnea or tachycardia > 120 beats/minute, and overt autonomic nervous system instability). Secondary diagnostic eligibility required clinical and dental verification of an odontogenic portal of entry (rampant caries dentis, ICD-10 K02.9). External cutaneous puncture wounds, fractures, burns, or otogenic suppuration had to be completely absent. The host also had documented zero-dose DTP immunization status (ICD-10 Z28.3) and severe somatic wasting (ICD-10 E63.9).

Longitudinal Observation Protocol

Clinical, hemodynamic, ventilatory, hematologic, biochemical, microbiological, and nutritional monitoring was carried out across eight standardized longitudinal

epochs. Epoch T0 (Day 1, October 1, 2024) comprised the Emergency Department primary survey using the Pediatric Assessment Triangle [PAT], WHO anthropometric Z-score plotting, baseline hematology/electrolytes, pre-referral chest radiography, and administration of 3,000 IU intramuscular human tetanus immunoglobulin [HTIG]. Epochs T1a and T1b (Day 2, October 2, 2024, 08:00 and 21:00 WIB) covered a morning spasm surge, repeat hematology, dental consultation, and a nighttime apneic cardiac arrest. They also included cardiopulmonary resuscitation [CPR], endotracheal intubation with a 4.5 mm cuffed tube at 13 cm depth, return of spontaneous circulation [ROSC], post-arrest arterial blood gas [ABG] and lactate analysis, and initiation of pressure-controlled synchronized intermittent mandatory ventilation [PSIMV+] plus continuous midazolam and ketamine infusions. Epoch T2 (Days 3–5, October 3–5, 2024) involved ventilator pressure/oxygen weaning and transition from ketamine to continuous propofol infusion. Epoch T3 (Days 6–7, October 6–7, 2024) included a spontaneous breathing trial [SBT] and extubation to HFNC. Acute extubation failure occurred at 2 hours, with ROX index quantification. Emergency re-intubation followed. Epoch T4 (Days 8–9, October 8–9, 2024) comprised repeat hematology, coagulation [PT, APTT, INR], renal function, isolation of ceftriaxone-resistant *Pseudomonas* sp. on blood culture, antibiotic escalation to intravenous meropenem, and vitamin K administration. Elective surgical tracheostomy followed on October 9, with cannula tip verification at the Th3 vertebral level. Epoch T5 (Days 10–13, October 10–13, 2024) covered post-tracheostomy weaning (Day 11) from PSIMV+ to T-piece oxygen at 2.0 to 1.0 L/min. Continuous midazolam and propofol infusions were stopped, and enteral baclofen was started. Epoch T6 (Days 14–19, October 14–19, 2024) involved weaning to 0.5 L/min T-piece and room air, complete cessation of intravenous diazepam on Day 14, enteral nasogastric feeding escalation to 960–1,200 mL/day, and hospital discharge on Day 19–20. Epochs T7 and T8 were Outpatient Pediatric Polyclinic evaluations on October 21 [Day 21] and November 6, 2024 [Day 37]. They culminated in scheduled tracheostomy decannulation on November 8, 2024 [Day 39].

The data were taken from the patient's records at the eight epochs described above.

Comparative Framework

To contextualize single-patient clinical kinetics without extrapolating beyond the observations, the patient's longitudinal parameters were evaluated against three reference standards. First were age-, sex-, and height-specific pediatric physiological reference norms. These were the WHO Child Growth Standards Z-score curves for boys aged 2 to 5 years and the Fourth Report / AAP pediatric blood pressure percentiles for a 4-year-old boy of 94.0 cm height: 50th percentile [P50] = 95/55 mmHg, 90th percentile [P90] = 104/65 mmHg, 95th percentile [P95] = 107/68 mmHg, and 99th percentile [P99] = 113/72 mmHg. Second were validated pediatric critical care respiratory thresholds. These included the Pediatric Acute Lung Injury Consensus Conference (PALICC) PaO₂/FiO₂ and SpO₂/FiO₂ cutoffs and the pediatric HFNC ROX index threshold (ROX = [SpO₂/FiO₂]/RR, where values < 4.88 signify high risk of extubation failure). Third was a structured literature benchmark. It compared clinical course, airway timing, and survival against contemporary published adult and pediatric tetanus cohorts from 2021 to 2026.

Biostatistical Analysis

Because the report concerns a single patient, inferential hypothesis testing (p-values, t-tests, analysis of variance, chi-square tests) was not performed. Deterministic longitudinal calculations were applied to the serial parameters instead. Relative trajectory magnitude was quantified as Percentage Delta Change, defined as $\Delta\% = [(X_t - X_0) / X_0] \times 100\%$. Here X₀ is the baseline admission (or pre-intervention peak) value. X_t is the value at epoch t. The linear rate of clinical resolution or escalation across an interval $\Delta t = t_2 - t_1$ was computed as Resolution Velocity, $v = (X_{t_2} - X_{t_1}) / \Delta t$. Hemodynamic perfusion load was quantified with Mean Arterial Pressure, MAP = [SBP + 2(DBP)] / 3. Pulse Pressure, PP = SBP – DBP, was quantified likewise. Pulmonary gas exchange was tracked through the arterial oxygen tension-to-inspired oxygen fraction ratio (PaO₂/FiO₂), the non-invasive saturation-to-inspired oxygen ratio (SpO₂/FiO₂), and the Respiratory Rate-Oxygenation Index, ROX = (SpO₂ / FiO₂) / RR. Systemic hematologic inflammation was quantified with the Neutrophil-to-Lymphocyte Ratio, NLR = (% Segmented Neutrophils + % Band Neutrophils) / % Lymphocytes, and the Systemic Immune-Inflammation Index, SII = Platelet Count ($\times 10^3/\mu\text{L}$) $\times$ NLR. Somatic nutritional energetics were modeled with the Schofield pediatric Basal Metabolic Rate equation for boys aged 3 to 10 years, BMR (kcal/day) = $19.49 \times \text{Weight (kg)} + 130.3 \times \text{Height (m)} + 414.9$. This was adjusted for critical illness hypercatabolism via Resting Energy Expenditure, REE = BMR $\times$ Stress Factor (1.5) $\times$ Activity Factor (0.8). It was then compared against Recommended Dietary Allowance targets for Ideal Body Weight (IBW = 14.0 kg), Target Energy = IBW $\times$ 90 kcal/kg/day.

3. Results

Baseline Demographic, Odontogenic Portal, and Emergency Triage Ascertainment

The patient was a 4-year-2-month-old (50-month-old) boy from Payakumbuh, West Sumatera, Indonesia. He was referred from RSUD Dr. Adnaan WD Payakumbuh and admitted to Dr. M. Djamil General Hospital on October 1, 2024 (Epoch T0, Hospital Day 1). Alloanamnesis from the parents (father 35 years old, senior high school graduate, private sector employee; mother 34 years old, senior high school graduate, housewife) revealed a 6-day pre-hospital prodrome. This began with cough, fever, sore throat, coryza, and inability to expectorate tenacious phlegm. On Day –3 prior to admission (the third febrile day), progressive lockjaw (trismus) developed. Interincisal mouth opening was restricted to the width of one finger (approximately 1.0 cm). That was a –75.0% reduction compared to the normal preschool opening of 3.5 to 4.0 cm. Severe dysphagia and dysarthria accompanied it. Hypertonia then spread fast. Within 24 hours of trismus onset (Day –2 prior to admission), it involved the cervical, paraspinal, abdominal, and appendicular musculature. This produced pronounced backward arching of the neck and spine (opisthotonus) and recurrent generalized tonic spasms, 3 to 4 times per day, each persisting for more than 1.0 minute. On arrival in the Emergency Department, an acute generalized tetanic convulsion was witnessed. The child then regained full consciousness (Glasgow Coma Scale [GCS] 15: E4M6V5).^{16,17}

Detailed epidemiological and physical examination excluded any history or cutaneous evidence of penetrating puncture wounds, lacerations, burns, fractures, hematomas, animal bites, or otitis media. The ear canals were dry and intact bilaterally. Intraoral inspection, however, demonstrated rampant dental caries (caries dentis, ICD-10

K02.9) across multiple primary molars without active gingival hemorrhage. This established an anaerobic odontogenic portal of entry in a household with poor environmental hygiene and sanitation.^{17,18} Perinatal records confirmed full-term spontaneous vaginal delivery at 40 to 41 weeks of gestation with a birth weight of 3,300 g. Immunization verification disclosed a major vaccination gap (ICD-10 Z28.3). Only neonatal birth immunizations and a single dose of oral polio vaccine (OPV) two months prior to presentation had been received, with zero doses of Diphtheria-Pertussis-Tetanus (DTP) vaccine (0/4 recommended doses; -100.0% immunization deficit).

Baseline pediatric triage with the Pediatric Assessment Triangle (PAT) classified the child as critically ill, with central nervous system and neuromuscular instability. Findings were active hypertonus, preserved eye contact and consolability, absence of stridor or retractions, and warm acral perfusion with capillary refill time < 2.0 seconds. Table 1 details the baseline demographic, socio-environmental, anthropometric, immunization, and primary survey parameters at T0.

Table 1. Baseline Demographic, Socio-Environmental, Anthropometric, Immunization, and Primary Survey Profile at Emergency Presentation (T0: October 1, 2024).

Clinical Domain & Parameter	Observed Patient Value at T0 (Day 1)	Pediatric Reference Interval / Standard	Quantitative Deviation / Z-Score / Ratio	Pathophysiological & Diagnostic Interpretation
Demographics & Referral Origin	Male, 4 yr 2 mo (50 mo)	Preschool age stratum (24–60 mo)	Referred from RSUD Dr. Adnaan WD Payakumbuh	High-vulnerability unimmunized preschool child from West Sumatera
Parental & Socio-Environmental Profile	Father: 35 yr (High school, private employee); Mother: 34 yr (High school, housewife)	WHO household sanitation & hygiene standards	Permanent ventilated house; poor sanitation	Environmental spore exposure compounded by delayed preventive dental & vaccine care
Immunization & Vaccine Status	Birth vaccines + 1 dose OPV (2 mo prior); DPT = 0 doses	3 primary DTP doses + booster (anti-tetanus IgG > 0.01 IU/mL)	0 / 4 recommended DTP doses (-100.0% deficit)	Complete absence of humoral neutralizing anti-tetanus toxin immunity (ICD-10 Z28.3)
Portal of Entry & Incubation Prodrome	Rampant dental caries (caries dentis); zero external puncture wounds	Intact oral mucosa and caries-free primary dentition	Prodrome: Day -6 cough/fever; Day -3 trismus; Day -2 spasms	Anaerobic necrotic pulp/periodontal microenvironment enabling <i>C. tetani</i> spore germination (ICD-10 K02.9)
Anthropometry & Somatic Growth	Weight: 11.0 kg; Height: 94.0 cm; BMI: 12.45 kg/m ² ; Height age: 3 yr 2 mo	Ideal Body Weight (IBW): 14.0 kg; Target Height: 156.5–173.5 cm	WAZ = -3.0 SD; HAZ: -3.0 < SD < -2.0; WHZ = -3.0 SD (-21.4% vs. IBW)	Severe undernutrition / wasting (ICD-10 E63.9) with diminished physiological and diaphragmatic reserve
Nutritional Energetics & Fluid Target	BMR: 751.77 kcal/day; REE: 902.1 kcal/day; Target RDA: 1,260.0 kcal/day	90 kcal/kg IBW/day; Fluid: 100–110 mL/kg/day (1,100–1,210 mL/day)	Initial 10% fluid restriction: 950 mL/24 h (KaEN 1B at 37 mL/h)	Hypercatabolic tetanic state (stress factor 1.5) requiring staged enteral NGT escalation
Primary Survey: Airway & Cranial Nerves	Patent airway, no stridor; severe trismus (interincisal opening ≈ 1.0 cm)	Interincisal opening ≥ 3.5–4.0 cm; intact swallowing reflex	-75.0% reduction in mouth opening (1 fingerbreadth); thick secretions	Presynaptic VAMP/synaptobrevin-2 cleavage in trigeminal motor nucleus (CN V) & pharyngeal dysphagia
Primary Survey: Breathing & Oxygenation	RR: 24–26 breaths/min; bilateral fine rales (+/+); SpO ₂ : 93–95% on room air	RR: 20–28 breaths/min; vesicular sounds; SpO ₂ ≥ 97% (FiO ₂ 0.21)	SpO ₂ /FiO ₂ = 442.9–452.4; placed on nasal O ₂ 2.0 L/min (FiO ₂ ≈ 0.28)	Early aspiration bronchopneumonia (ICD-10 J18.0) secondary to sialorrhea and impaired cough clearance
Primary Survey: Circulation & Dysautonomia	BP: 130/85 mmHg (MAP: 100.0 mmHg); HR: 130–145 bpm; Temp: 38.9 °C; CRT < 2 s	Age/height BP percentiles: P50 = 95/55, P90 = 104/65, P95 = 107/68, P99 = 113/72 mmHg	SBP +15.0% above P99 (+36.8% above P50); DBP +18.1% above P99	Stage 2 sympathetic hypertension, sinus tachycardia & hyperpyrexia defining Ablett Grade IV tetanus (ICD-10 A35)
Primary Survey: Disability & Motor Tone	GCS 15 (E ₄ M ₆ V ₅); pupils 2–3 mm isochoric; neck stiffness (+); abdominal rigidity (+); opisthotonus (+)	Normal axial/appendicular tone; zero spontaneous or reflex spasms	3–4 generalized spasms/day (> 1 min each) pre-admission; 1 ED spasm	Spinal Renshaw cell glycinergic and GABAergic disinhibition with preserved cortical consciousness

Abbreviations: BMI, body mass index; BMR, basal metabolic rate; BP, blood pressure; CN V, trigeminal nerve; CRT, capillary refill time; DBP, diastolic blood pressure; DOB, date of birth; DPT/DTP, diphtheria-pertussis-tetanus vaccine; ED, emergency department; FiO₂, fraction of inspired oxygen; GCS, Glasgow Coma Scale; HAZ, height-for-age Z-score; HR, heart rate; IBW, ideal body weight; MAP, mean arterial pressure; NGT, nasogastric tube; OPV, oral polio vaccine; RDA, recommended dietary allowance; REE, resting energy expenditure; RR, respiratory rate; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation; VAMP, vesicle-associated membrane protein; WAZ, weight-for-age Z-score; WHZ, weight-for-height Z-score.

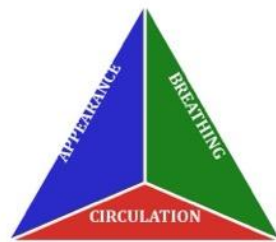
Autonomic Instability, Serial Thoracic Radiography, and WHO Anthropometric Z-Score Bioenergetics

Baseline physical examination at T0 confirmed Ablett Grade IV (very severe) generalized tetanus complicated by overt sympathetic autonomic storm and early aspiration bronchopneumonia.^{17,18} Blood pressure surged to 130/85 mmHg. This corresponded to a Mean Arterial Pressure (MAP) of 100.0 mmHg and a pulse pressure of 45.0 mmHg. Age- and height-matched pediatric blood pressure standards for a 94.0 cm boy were P50 = 95/55 mmHg, P90 = 104/65 mmHg, P95 = 107/68 mmHg, and P99 = 113/72 mmHg. Against these, admission systolic pressure exceeded the 99th percentile by +17.0 mmHg (+15.0% above P99 and +36.8% above P50). Diastolic pressure exceeded P99 by +13.0 mmHg (+18.1% above P99 and +54.5% above P50), confirming Stage 2 sympathetic hypertension. Sinus tachycardia reached 130 to 145 beats/minute (stabilizing to 100–110 beats/minute on the ward following initial intravenous diazepam bolus therapy). Core axillary temperature peaked at 38.9 °C. Respiratory rate was 24 to 26 breaths/minute, with peripheral oxygen saturation

(SpO₂) of 93.0% to 95.0% on ambient room air (FiO₂ 0.21; baseline SpO₂/FiO₂ ratio = 442.9 to 452.4). Immediate supplementation followed with nasal cannula oxygen at 2.0 L/minute (FiO₂ ≈ 0.28). Thoracic auscultation revealed symmetrical normothorax movement with bilateral fine inspiratory rales (+/+) and thick pooled oropharyngeal secretions, without wheezing or stridor. Figure 1 illustrates the emergency PAT triage and the pre-referral anteroposterior (AP) and lateral thoracic radiographs obtained at RSUD Dr. Adnaan WD Payakumbuh (verifying right upper lung field consolidation with bilateral perihilar infiltrates compatible with bronchopneumonia, ICD-10 J18.0). It also shows the post-stabilization/tracheostomy AP thoracic radiograph at Dr. M. Djamil General Hospital (confirming normal cardi thoracic contours, bilateral perihilar bronchopneumonic patches, and optimal positioning of the tracheostomy cannula tip at the third thoracic vertebra, Th3) and the serial SpO₂/FiO₂ oxygenation trajectory.

(a) Emergency Triage: Pediatric Assessment Triangle (PAT at T₀)
Active Hypertonus, Patent Airway, Normal Perfusion →
CNS Instability

Pediatric Assessment Triangle (PAT)



Breathing

- **Breath sound** : no stridor
- **Position** : No sniffing, no tripod
- **Retraction** : No epigastrium retraction
- **Nasal flare** : No nasal flare

Appearance (TICLS)

- **Tonus** : Active, hypertonus
- **Interactiveness** : interactive (+)
- **Consolability** : Consolable (+)
- **Look or gaze** : Eye contact (+)
- **Speech and cry** : Cry (+)

Circulation

Mottled (-), Pale (-),
Cyanotic (-)

Conclusion : CNS problem

(c) Post-Stabilization / Tracheostomy
AP Thoracic Radiograph
Cannula Tip Verified at Th₃ Vertebral
Level | Bilateral Perihilar Infiltrates



Cannula tip verified at Th₃ vertebral level |
normal cardiothoracic ratio

(b) Baseline AP & Lateral Chest Radiograph (T₀: Oct 1, 2024)
Right Upper Lobe Consolidation & Perihilar Infiltrates
(Bronchopneumonia)



Aspiration bronchopneumonia (ICD-10 J18.0) | SpO₂ 93–95% (room air)

(d) Serial Oxygenation (SpO₂/FiO₂) & FiO₂ Trajectory
T-Piece Liberation Within 24–48 Hours After Day 9 Tracheostomy

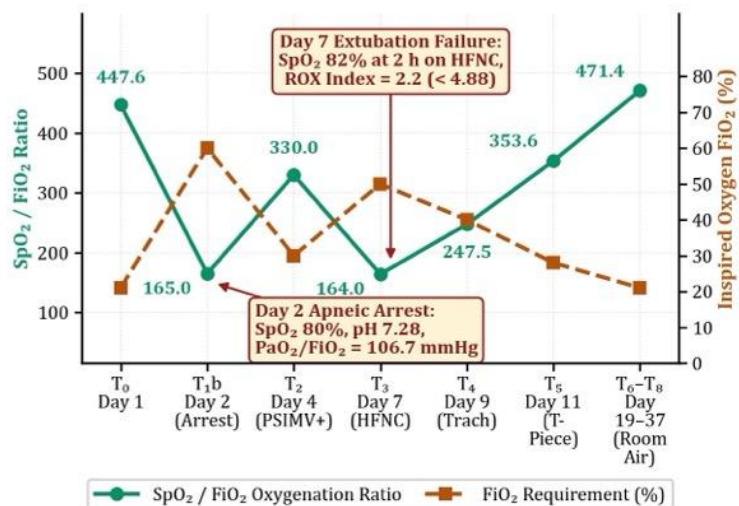


Figure 1. Emergency Department Pediatric Assessment Triangle (PAT) Triage, Thoracic Radiographs With Anonymized Margins, and Quantitative Oxygenation Trajectory. (a) Emergency Department PAT triage diagram at T₀ demonstrating active hypertonus, patent airway without stridor, and normal peripheral circulation, localizing primary instability to the central nervous system. (b) Baseline anteroposterior (AP) and lateral chest radiographs at RSUD Dr. Adnaan WD (T₀: October 1, 2024) demonstrating right upper lung field consolidation and bilateral perihilar infiltrates (bronchopneumonia, ICD-10 J18.0), with permanent solid black anonymization bars masking superior craniofacial margins and patient identity headers. (c) Post-resuscitation / tracheostomy AP chest radiograph at Dr. M. Djamil General Hospital confirming normal cardiac silhouette, bilateral perihilar bronchopneumonic patches, and tracheostomy cannula tip at the Th₃ vertebral level. (d) Serial SpO₂/FiO₂ oxygenation ratio and FiO₂ trajectory across Epochs T₀ to T₈, highlighting Day 2 apneic arrest (PaO₂/FiO₂ = 106.7 mmHg), Day 7 HFNC extubation failure (ROX index = 2.2), and rapid 24-hour T-piece weaning post-tracheostomy.

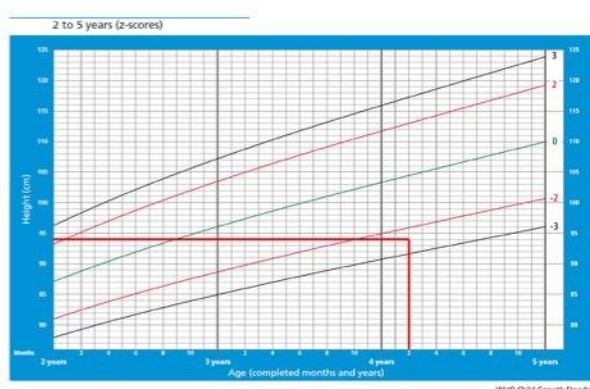
Anthropometric evaluation at admission (T₀) revealed severe comorbid somatic undernutrition (ICD-10 E63.9), plotted against World Health Organization (WHO) Child Growth Standards for boys aged 2 to 5 years.^{19,20} The child weighed 11.0 kg (unchanged from pre-illness baseline). Standing height was 94.0 cm. These values yielded a Body Mass Index (BMI) of 12.45 kg/m², a height age of 3 years 2 months, and an Ideal Body Weight (IBW) of 14.0 kg (–21.4% body mass deficit relative to IBW). Parental statures (father 162.0 cm, mother 155.0 cm) established a genetic target height range of 156.5 to 173.5 cm. Plotting on official WHO growth charts confirmed a Weight-for-Height Z-score (WHZ) of –3.0 SD (severe wasting). The Height-for-Age Z-score (HAZ) lay between –3.0 SD and –2.0 SD (moderate stunting), and the Weight-for-Age Z-score (WAZ) was –3.0 SD (severe underweight). Nutritional bioenergetic modeling used the Schofield equation. It yielded a Basal Metabolic

Rate (BMR) of 19.49(11.0) + 130.3(0.94) + 414.9 = 214.39 + 122.48 + 414.90 = 751.69 kcal/day. The acute-phase Resting Energy Expenditure (REE, incorporating a tetanus stress factor of 1.5 and bedrest activity factor of 0.8) was 902.1 kcal/day (751.77 × 1.5 × 0.8). The catch-up Recommended Dietary Allowance (RDA) target was 14.0 kg × 90 kcal/kg/day = 1,260.0 kcal/day. Standard maintenance fluid requirements were 100 to 110 mL/kg/day (1,100 to 1,210 mL/day). To prevent cerebral edema during acute tetanic convulsions, however, total daily fluid intake was initially restricted by 10.0% to 950.0 mL/24 hours (administered as intravenous KaEN 1B at 37.0 mL/hour on Day 1 while nil per os [NPO]). Staged nasogastric tube (NGT) liquid formula escalation followed, from 150.0 mL/day (6 × 25 mL) on Day 2 to 1,200.0 mL/day (6 × 200 mL; +700.0% enteral volume expansion) at nutritional stabilization, as depicted in Figure 2.

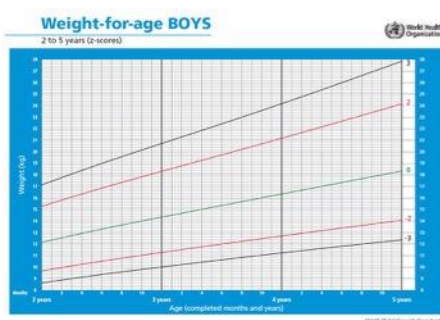
(a) WHO Weight-for-Height Z-Score (WHZ = -3.0 SD) — Weight: 11.0 kg at Height: 94.0 cm (Ideal Body Weight: 14.0 kg)



(b) WHO Height-for-Age Z-Score ($-3.0 < \text{HAZ} < -2.0$ SD) — Height: 94.0 cm at Age 4 yr 2 mo (Height Age: 3 yr 2 mo)



(c) WHO Weight-for-Age Z-Score (WAZ = -3.0 SD) — Severe Wasting (BMI = 12.45 kg/m^2 ; -21.4% Below Ideal Weight)



(d) Staged Enteral NGT Volume Escalation (0 → 1,200 mL/day) — Transition From 10% Fluid Restriction (950 mL) to Full Enteral Nutrition

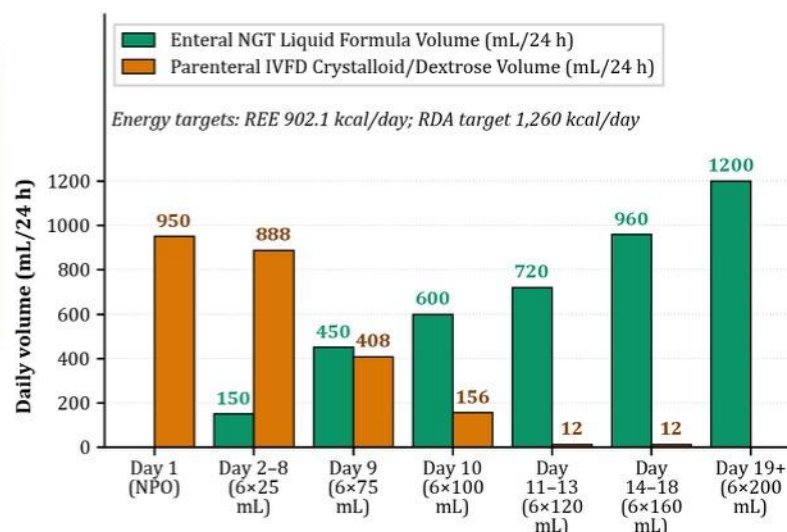


Figure 2. Baseline World Health Organization (WHO) Child Growth Standards Z-Score Plots (Boys 2–5 Years) and Longitudinal Enteral Nutritional Escalation. (a) WHO Weight-for-Height plot confirming WHZ = -3.0 SD (11.0 kg at 94.0 cm; severe wasting, ideal body weight 14.0 kg). (b) WHO Height-for-Age plot confirming $-3.0 < \text{HAZ} < -2.0$ SD (94.0 cm at age 4 years 2 months; height age 3 years 2 months). (c) WHO Weight-for-Age plot confirming WAZ = -3.0 SD (BMI = 12.45 kg/m^2). (d) Staged transition from initial 10% fluid-restricted parenteral therapy (950 mL/24 h) and Day 2 trophic nasogastric feeding (150 mL/day) to full enteral nutrition (1,200 mL/day; +700.0% increase) toward the 1,260.0 kcal/day RDA target (REE = 902.1 kcal/day).

Longitudinal Cardiorespiratory Trajectory, ROX Index Extubation Failure, and Post-Tracheostomy Weaning

Immediate emergency pharmacotherapy on Day 1 (T0) comprised several agents. Intramuscular Human Tetanus Immunoglobulin (HTIG) 3,000 IU single dose neutralized unbound circulating tetanospasmin. Intravenous ceftriaxone 550 mg was given every 12 hours (100.0 mg/kg/day). Intravenous metronidazole eradicated vegetative *C. tetani* bacilli (initiated at 90 mg q6h / 350 mg TID on Day 1 and standardized to 110 mg every 6 hours [40.0 mg/kg/day] from Day 2 onward). Intravenous paracetamol 150 mg was given as needed. Slow intravenous bolus diazepam 5.0 mg was given six times daily (30.0 mg/day = 2.73 mg/kg/day).^{21,22} The child stabilized initially on the ward on Day 1 (HR 100–110 beats/minute, RR 26 breaths/minute, temperature 37.0°C , and a single brief seizure < 1.0 minute). Progression then accelerated sharply on the morning of Hospital Day 2 (October 2, 2024, 08:00 WIB; Epoch T1a). Tactile reflex tetanic spasms surged to 10

to 12 episodes/day (+200.0% to +242.9% increase over pre-admission frequency). These were accompanied by persistent 1.0 cm trismus, copious thick oral secretions, bilateral pulmonary rales, intermittent opisthotonus, and four-limb spasticity. Pediatric dentistry consultation on Day 2 confirmed rampant caries dentis as the primary infectious focus. Definitive tooth extraction was recommended once intensive care stabilization was achieved. At 21:00 WIB on Day 2 (Epoch T1b), a violent generalized tetanic convulsion precipitated acute laryngospasm, prolonged apnea, cyanotic desaturation to SpO₂ 80.0% ($\Delta\% = -14.9\%$ from baseline 94.0%), and pulseless cardiac arrest. Immediate Pediatric Advanced Life Support cardiopulmonary resuscitation (CPR) and emergency translaryngeal endotracheal intubation (size 4.5 mm cuffed endotracheal tube [ETT] secured at 13.0 cm lip depth) achieved Return of Spontaneous Circulation (ROSC). Mechanical ventilation began in Pressure-Controlled Synchronized Intermittent Mandatory Ventilation (PSIMV+) mode. Settings were Peak Inspiratory Pressure (Pins) 12.0 cmH₂O, respiratory rate 25

breaths/minute, inspiratory-to-expiratory (I:E) ratio 1:2, Positive End-Expiratory Pressure (PEEP) 6.0 cmH₂O, FiO₂ 60.0% (0.60), and flow trigger 3.0 L/minute. This restored peripheral saturation to SpO₂ 99.0% (PaO₂/FiO₂ = 64.0 / 0.60 = 106.7 mmHg; SpO₂/FiO₂ = 165.0). Table 2 details the

complete longitudinal trajectory of hemodynamic, respiratory, ventilator, and neuromuscular parameters across Epochs T0 through T8.

Table 2. Longitudinal Trajectory of Hemodynamic Parameters, Respiratory Mechanics, Ventilator Settings, and Neurological Spasm Activity Across Eight Observation Epochs (T0 to T8: Days 1 to 37).

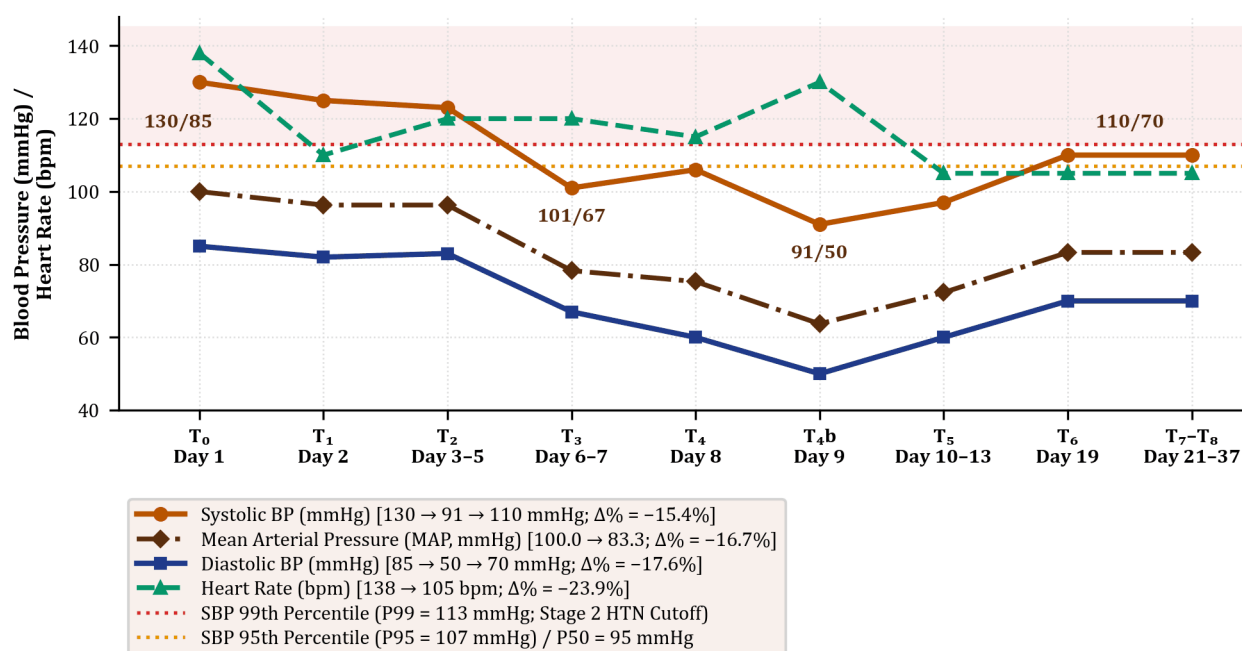
Epoch & Clinical Date	Airway & Ventilatory Support Mode	Ventilator / Oxygen Parameters & ROX / P/F Metrics	BP (mmHg) [MAP] & HR (bpm)	Temp (°C) & SpO ₂ (%)	Neuromuscular Tone, Trismus & Daily Spasm Burden	Key Clinical Event & Percentage Delta Change (Δ% vs. T0)
T0: Day 1 (Oct 1, 2024)	Spontaneous breathing; Nasal cannula O ₂ 2.0 L/min	FiO ₂ ≈ 0.28 (room air SpO ₂ 93–95%); RR 24–26/min; bilateral rales (+/+)	130/85 [100.0]; HR 130–145 (ward: 100–110)	38.9 → 37.0 °C; SpO ₂ 93–95%	GCS E ₄ M ₆ V ₅ ; Trismus 1.0 cm; opisthotonus (+); 3–4 spasms/day pre-adm (1 on ward)	Baseline Ablett Grade IV admission; HTIG 3,000 IU IM + IV diazepam, ceftriaxone & metronidazole initiated
T1a: Day 2 AM (Oct 2, 2024, 08:00)	Spontaneous breathing; Nasal cannula O ₂ 2.0 L/min	FiO ₂ ≈ 0.28; RR 26/min; copious thick oral secretions; rales (+/+)	125/82 [96.3]; HR 100–110	37.0 °C; SpO ₂ 95–96%	GCS E ₄ M ₆ V ₅ ; Trismus 1.0 cm; opisthotonus (+); 10–12 tactile spasms/day (< 1 min)	Peak pre-intubation spasm surge (+200.0% to +242.9% vs. pre-admission baseline); dental consult confirmed caries
T1b: Day 2 PM (Oct 2, 2024, 21:00)	Emergency ETT (size 4.5 mm, depth 13 cm); PSIMV+ mode	Pins 12 cmH ₂ O, PEEP 6 cmH ₂ O, RR 25/min, I:E 1:2, FiO ₂ 60% (0.60), F.T. 3.0; PaO ₂ /FiO ₂ = 106.7 mmHg	Post-ROSC: 123/83 [96.3]; HR 120	37.0 °C; SpO ₂ 80% (arrest) → 99% (MV)	Sedated; acute apneic spasm & cardiac arrest; ROSC achieved via CPR + intubation	Fulminant laryngospasm & hypoxemic arrest (SpO ₂ Δ% = –14.9% at nadir, restored +5.3% to 99% on PSIMV+)
T2: Days 3–5 (Oct 3–5, 2024)	Endotracheal tube (4.5 mm); PSIMV+ mode	Day 3: Pins 12, FiO ₂ 60%; Days 4–5: Pins 8 cmH ₂ O, PEEP 6 cmH ₂ O, RR 25/min, I:E 1:1.7, FiO ₂ 30% (S/F = 330.0)	123/83 [96.3]; HR 120	37.0 °C; SpO ₂ 99%	Sedated (Midazolam + Ketamine → Propofol); persistent tactile spasms & limb spasticity	Inspiratory pressure reduced –33.3% (12 → 8 cmH ₂ O) & FiO ₂ reduced –50.0% (60% → 30%); MAP Δ% = –3.7%
T3: Days 6–7 (Oct 6–7, 2024)	SBT → Extubation to HFNC (2 h) → Re-intubation (PSIMV+)	PSIMV+: Pins 8, PEEP 6, FiO ₂ 30%, VtE 7–8 mL/kg; HFNC failure at 2 h: SpO ₂ 82%, ROX index = 2.2	101/67 [78.3]; HR 120	36.7 °C; SpO ₂ 99% (82% on HFNC)	GCS E ₄ M ₆ B ₄ R ₁ ; reduced tactile spasms, but post-extubation spasms & retractions forced re-intubation	ROX index 2.2 (< 4.88 cutoff) suggested upper airway/secretion obstruction; SBP Δ% = –22.3%, MAP Δ% = –21.7%
T4: Days 8–9 (Oct 8–9, 2024)	ETT PSIMV+ → Elective Surgical Tracheostomy (Oct 9, tip at Th3)	Day 8: Pins 10, PEEP 5, I:E 1:2, FiO ₂ 50%; Day 9: Pins 8, PEEP 5, I:E 1:1.7, FiO ₂ 40%, VtE 7–8 mL/kg	Day 8: 106/60 [75.3]; Day 9: 91/50 [63.7]; HR 110–140	Day 8: 37.6 °C; Day 9: 38.7 °C (37.5 °C exam); SpO ₂ 99%	GCS E ₄ M ₆ B ₄ R ₁ ; decreased tactile spasms; intermittent back opisthotonus & four-limb spasticity	Pseudomonas sp. bacteremia treated with Meropenem; Surgical tracheostomy secured airway; MAP Δ% = –36.3%
T5: Days 10–13 (Oct 10–13, 2024)	Day 10: Tracheostomy PSIMV+; Day 11: T-piece 2 L/min; Day 12: T-piece 1 L/min	Day 10: Pins 8, PEEP 5, FiO ₂ 40%; Days 11–13: Spontaneous breathing via T-piece 2.0 → 1.0 L/min O ₂	Day 10: 97/54 [68.3]; Days 11–13: 97/60 [72.3]; HR 100–110	37.5 °C (afebrile ≥ 3 d); SpO ₂ 99%	GCS E ₄ M ₆ V ₄ ; continuous IV sedation stopped; oral Baclofen 3 × 2.5 mg started; tactile spasms decreased	Ventilator weaning to T-piece within 48 h of tracheostomy (Day 11); HR Δ% = –23.6%, MAP Δ% = –27.7% vs. T0
T6: Days 14–19 (Oct 14–19, 2024)	Tracheostomy with T-piece 0.5 L/min (Day 15) → Room air (Day 19)	Spontaneous respiration via tracheostomy; RR 24–28/min; FiO ₂ 0.21; SpO ₂ /FiO ₂ = 471.4	Days 14–18: 97/60 [72.3]; Day 19: 110/70 [83.3]; HR 100–110	37.5 → 37.0 °C (afebrile > 5 d); SpO ₂ 99%	GCS E ₄ M ₆ V ₄ ; zero tactile spasms on Day 19; residual mild limb tone managed on oral Baclofen; IV diazepam stopped	100.0% spasm resolution; discharged home on Day 19–20 with oral Cefixime 2 × 60 mg & Baclofen 3 × 2.5 mg
T7 & T8: Days 21 & 37 (Oct 21 & Nov 6, 2024)	Outpatient Tracheostomy on Room Air (Decannulation planned Nov 8, Day 39)	Spontaneous room-air breathing (FiO ₂ 0.21); RR 24–28/min; clear vesicular breath sounds (rales 0/0)	110/70 [83.3]; HR 100–110	37.0 °C; SpO ₂ 99%	GCS E ₄ M ₆ V ₄ ; Trismus 0% (normal opening); Spasticity 0%; Opisthotonus 0%; normal physiological reflexes	Complete neurological, autonomic & pulmonary recovery (SBP Δ% = –15.4%, MAP Δ% = –16.7%, Spasms Δ% = –100.0%)

Abbreviations: BP, blood pressure; CPR, cardiopulmonary resuscitation; ETT, endotracheal tube; FiO₂, fraction of inspired oxygen; F.T., flow trigger; GCS, Glasgow Coma Scale; HFNC, high-flow nasal cannula; HR, heart rate; HTIG, human tetanus immunoglobulin; I:E, inspiratory-to-expiratory ratio; MAP, mean arterial pressure; MV, mechanical ventilation; PEEP, positive end-expiratory pressure; Pins, peak inspiratory pressure; PSIMV+, pressure-controlled synchronized intermittent mandatory ventilation; ROSC, return of spontaneous circulation; ROX, respiratory rate-oxygenation index; RR, respiratory rate; SBT, spontaneous breathing trial; S/F, SpO₂/FiO₂ ratio; VtE, expiratory tidal volume.

Across Days 3 to 5 (Epoch T2, October 3–5, 2024), continuous neuro-sedative infusions were titrated to suppress reflex spasms and autonomic hyperactivity while maintaining ventilator synchrony.^{23–25} Immediately post-ROSC on Day 2–3, sedation was maintained with continuous intravenous midazolam (16.5 mg diluted in 25.0 mL normal saline; initiated at 4.0 mL/hour post-arrest and maintained at 1.0 mL/hour on Day 3). Continuous intravenous ketamine (275.0 mg in 25.0 mL normal saline at 1.0 mL/hour = 1.0 mg/kg/hour) was added, together with escalated intravenous diazepam boluses of 3.0 mg twelve times daily (36.0 mg/day = 3.27 mg/kg/day). Pulmonary compliance improved by Days 4 to 5. Ventilator weaning to Pins 8.0 cmH₂O (Δ% = –33.3% vs. post-arrest 12.0 cmH₂O), PEEP 6.0 cmH₂O, RR 25 breaths/minute, I:E ratio 1:1.7, and FiO₂ 30.0% (Δ% = –50.0% vs. 60.0%; SpO₂/FiO₂ improved +100.0% from 165.0 to 330.0) was then possible. Ketamine was transitioned to a continuous intravenous propofol infusion at 48.0 µg/kg/min (3.0 mL/hour, where 1.0 mL/hour ≈ 16.0 µg/kg/min), alongside midazolam 4.0 µg/kg/min and tapered diazepam boluses (2.0 mg ten times daily = 20.0 mg/day). On Days 6 to 7 (Epoch T3, October 6–7, 2024), blood pressure normalized to 101/67 mmHg (MAP 78.3 mmHg; Δ% = –21.7% vs. T0 MAP 100.0 mmHg).

Propofol was tapered by –66.7% from 48.0 to 16.0 µg/kg/min. Midazolam ran at 4.0–5.0 µg/kg/min, and diazepam was tapered from 2.0 mg 8×/day [16.0 mg/day] on Day 6 to 1.0 mg 6×/day [6.0 mg/day] on Day 7. The child passed a spontaneous breathing trial (VtE 7.0–8.0 mL/kg) and was extubated to High-Flow Nasal Cannula (HFNC). Within exactly 2.0 hours post-extubation, however, unopposed pharyngeal and laryngeal muscle spasms, copious viscid secretions, intercostal retractions, and acute hypoxemia (SpO₂ dropping to 82.0%) supervened. The objective Respiratory Rate-Oxygenation (ROX) index was 2.2 (severely below the 4.88 threshold required for HFNC success), which mandated emergent re-intubation. Following parental counseling and written informed consent, elective surgical tracheostomy was performed on October 9, 2024 (Day 9, Epoch T4). Within 24 to 48 hours post-tracheostomy (Day 11, Epoch T5), the patient was liberated from positive-pressure mechanical ventilation onto a T-piece at 2.0 L/minute O₂ (weaned to 1.0 L/minute on Day 12, 0.5 L/minute on Day 15, and ambient room air by Day 19 with SpO₂ 99.0% [SpO₂/FiO₂ = 471.4]). All tactile spasms resolved completely (Δ% = –100.0%) by Day 19. Trismus and limb spasticity resolved completely by Outpatient Day 21, as illustrated in Figure 3.

(a) Sympathetic Autonomic Storm (Stage 2 HTN) & Hemodynamic Stabilization



(b) Daily Tetanic Spasm Burden & Interincisal Mouth Opening Trajectory

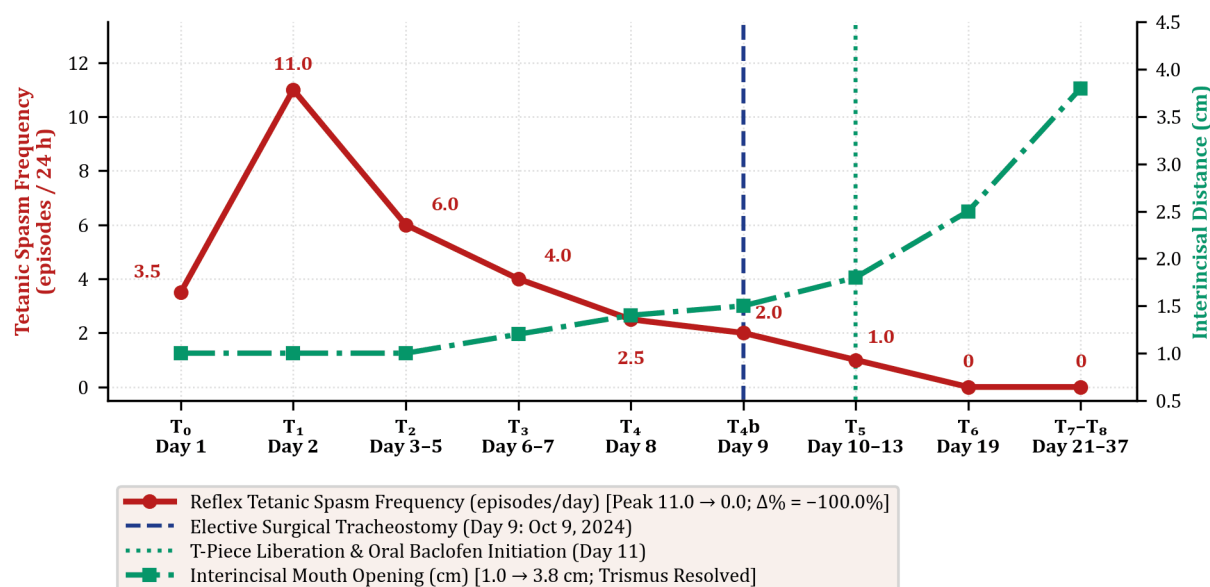


Figure 3. Longitudinal Autonomic Hemodynamic Trajectory and Neuromuscular Tetanic Spasm / Trismus Resolution Kinetics Across Eight Observation Epochs (Days 1 to 37). (a) Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) plotted against age- and height-specific pediatric percentiles (P50 = 95/55, P90 = 104/65, P95 = 107/68, P99 = 113/72 mmHg), demonstrating baseline Stage 2 sympathetic hypertension (130/85 mmHg; MAP 100.0 mmHg) and progressive stabilization to 110/70 mmHg (MAP 83.3 mmHg; Δ% = -16.7%). (b) Daily reflex tetanic spasm frequency (peaking at 10–12 episodes/day on Day 2 before declining by -100.0% to zero by Day 19) and interincisal mouth opening recovery (1.0 cm trismus at T₀ to 3.8 cm normal opening by Day 21).

Hematologic-Inflammatory Indices, Arterial Blood Gas Kinetics, *Pseudomonas Bacteremia*, and Sedative Titration

Table 3 summarizes the serial hematologic, derived inflammatory index, arterial blood gas, biochemical, coagulation, and microbiological profiles across the hospitalization. At admission (Day 1, October 1, 2024), the complete blood count showed marked leukocytosis of $22.35 \times 10^3/\mu\text{L}$ ($22,350/\text{mm}^3$; +123.5% above the upper normal limit of $10.00 \times 10^3/\mu\text{L}$). It also showed reactive

thrombocytosis of $524.0 \times 10^3/\mu\text{L}$ ($524,000/\text{mm}^3$; +16.4% above the $450.0 \times 10^3/\mu\text{L}$ threshold). Hemoglobin was 12.1 g/dL, hematocrit 37.3%, erythrocytes $4.80 \times 10^6/\mu\text{L}$, and reticulocytes 1.41%. The indices were borderline microcytic-hypochromic (MCV 77.7 fL, MCH 25.2 pg, MCHC 32.4 g/dL), reflecting underlying chronic nutritional deficiency.^{26–28} The baseline leukocyte differential was 0% basophils, 0% eosinophils, 1% band neutrophils, 77% segmented neutrophils, 15% lymphocytes, and 7% monocytes. From it, the baseline Neutrophil-to-Lymphocyte

Ratio (NLR) was $(77 + 1) / 15 = 5.20$, and the Systemic Immune-Inflammation Index (SII) was $524.0 \times 5.20 = 2,724.8 \times 10^3/\mu\text{L}$. On Day 2 (October 2, 2024, prior to nighttime cardiac arrest), total leukocytes changed minimally to $21.03 \times 10^3/\mu\text{L}$ ($\Delta\% = -5.9\%$). Platelet count rose to $536.0 \times 10^3/\mu\text{L}$ ($+2.3\%$), hemoglobin declined to 11.5 g/dL (-5.0%), and reticulocytes rose to 1.78% ($+26.2\%$), while the differential shifted sharply leftward to 0/0/6/75/11/8, a $+500.0\%$ surge in immature band neutrophils from 1% to 6% with lymphopenia of 11%, which drove the NLR up by $+41.5\%$ to 7.36 and the SII up by $+44.9\%$ to $3,946.9 \times 10^3/\mu\text{L}$. Immediately after Day 2 nighttime resuscitation and ROSC, arterial blood gas and

electrolyte analysis showed acute mixed respiratory and metabolic acidosis. Values were pH 7.28, PaCO₂ 45.0 mmHg, PaO₂ 64.0 mmHg (PaO₂/FiO₂ = 106.7 mmHg on FiO₂ 0.60), bicarbonate (HCO₃⁻) 21.6 mmol/L, base excess -5.6 mmol/L, and arterial oxygen saturation (SaO₂) 91.0%. Hyperlactatemia measured 2.2 mmol/L. Sodium (Na⁺) was 137 mmol/L, chloride (Cl⁻) 102 mmol/L, and calcium (Ca²⁺) 9.2 mg/dL. Post-arrest hypokalemia followed. Potassium (K⁺) dropped by -24.4% from 4.5 to 3.4 mmol/L, and it was subsequently corrected to 3.7 mmol/L [$+8.8\%$] via electrolyte-supplemented intravenous fluid therapy.

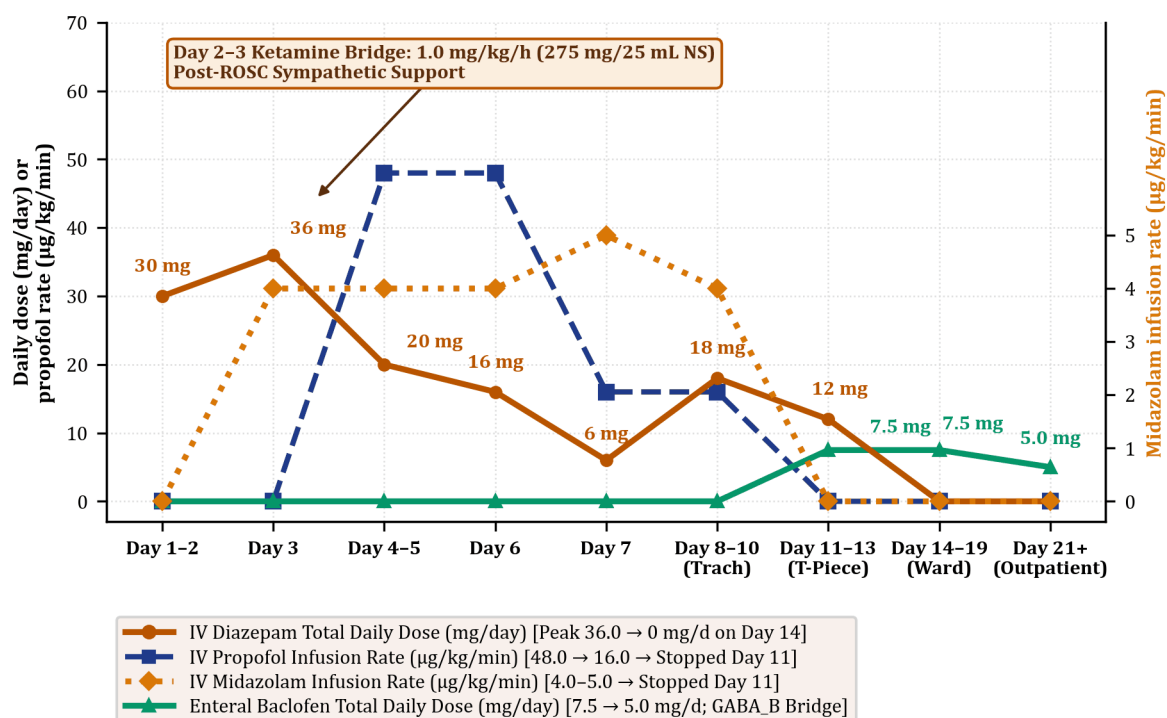
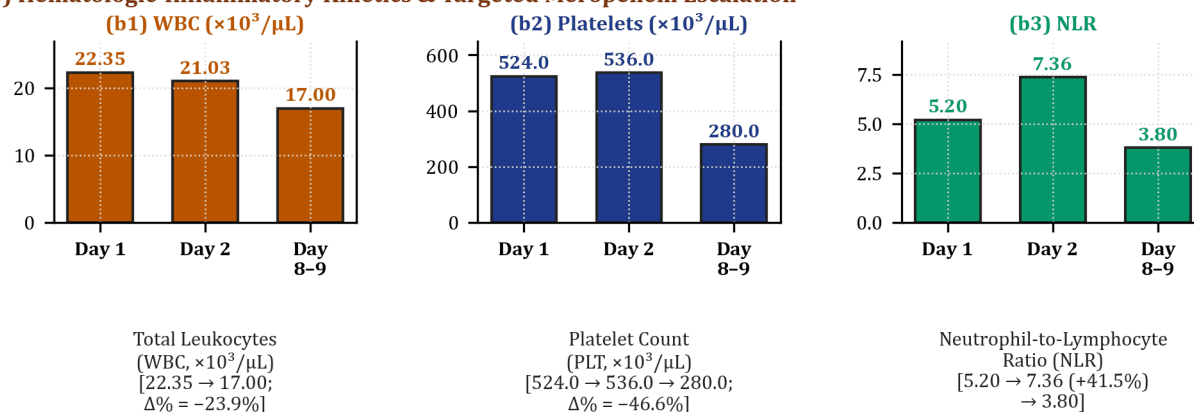
Table 3. Serial Hematologic, Derived Inflammatory Index, Arterial Blood Gas, Electrolyte, Coagulation, and Microbiological Kinetics (October 1 to October 9, 2024).

Laboratory & Microbiological Parameter	Day 1 Baseline (Oct 1, 2024)	Day 2 Pre-/Post-Arrest (Oct 2, 2024)	Day 8–9 Pre-Tracheostomy (Oct 8–9, 2024)	Pediatric Reference Interval	Relative Delta Change ($\Delta\%$ vs. Initial) & Clinical Interpretation
Hemoglobin (Hb, g/dL)	12.1	11.5	11.2	12.0–14.0 g/dL	$\Delta\% = -7.4\%$ (Day 1 to Day 8); mild anemia of critical illness
Hematocrit (Hct, %)	37.3%	36.4% (calc.)	34.5%	37.0–43.0%	$\Delta\% = -7.5\%$ (Day 1 to Day 8); parallel to hemoglobin trajectory during intensive care
Erythrocytes (RBC, $\times 10^6/\mu\text{L}$) & Reticulocytes (%)	$4.80 \times 10^6/\mu\text{L}$; Retic 1.41%	$4.58 \times 10^6/\mu\text{L}$; Retic 1.78%	Not repeated (stable)	RBC 3.4–5.0 $\times 10^6/\mu\text{L}$; Retic 0.8–2.8%	RBC $\Delta\% = -4.6\%$; Reticulocyte compensatory increase $\Delta\% = +26.2\%$ (Day 1 to Day 2)
Erythrocyte Indices (MCV / MCH / MCHC)	77.7 fL / 25.2 pg / 32.4 g/dL	79.5 fL / 25.1 pg / 31.6 g/dL	Described as normocytic normochromic (values not reported)	MCV 82–92 fL; MCH 27–31 pg; MCHC 32–36 g/dL	Borderline microcytic/hypochromic baseline consistent with chronic undernutrition (WAZ -3.0 SD)
Total Leukocytes (WBC, $\times 10^3/\mu\text{L}$)	$22.35 \times 10^3/\mu\text{L}$ (22,350/mm ³)	$21.03 \times 10^3/\mu\text{L}$ (21,030/mm ³)	$17.00 \times 10^3/\mu\text{L}$ (17,000/mm ³)	$5.00\text{--}10.00 \times 10^3/\mu\text{L}$	$\Delta\% = -5.9\%$ (Day 2) and -23.9% (Day 8 vs. Day 1); persistent leukocytosis prompted blood culture
Platelet Count (PLT, $\times 10^3/\mu\text{L}$)	$524.0 \times 10^3/\mu\text{L}$ (524,000/mm ³)	$536.0 \times 10^3/\mu\text{L}$ (536,000/mm ³)	$280.0 \times 10^3/\mu\text{L}$ (280,000/mm ³)	$150.0\text{--}450.0 \times 10^3/\mu\text{L}$	$\Delta\% = +2.3\%$ (Day 2 peak reactive thrombocytosis) $\rightarrow -46.6\%$ (Day 8 normalization into reference range)
Leukocyte Differential (Bas/Eos/Band/Seg/Lym/Mon, %)	0 / 0 / 1 / 77 / 15 / 7	0 / 0 / 6 / 75 / 11 / 8	Neutrophilic predominance	Band < 5%; Seg 40–60%; Lym 25–45%	Marked bandemia ($+500.0\%$ increase in band forms: 1% $\rightarrow$ 6%) & lymphopenia (15% $\rightarrow$ 11%) on Day 2
Neutrophil-to-Lymphocyte Ratio (NLR) & SII ($\times 10^3/\mu\text{L}$)	NLR = 5.20; SII = 2,724.8	NLR = 7.36; SII = 3,946.9	Declining inflammatory trajectory	Pediatric baseline NLR < 2.0; SII < 600	NLR $\Delta\% = +41.5\%$ & SII $\Delta\% = +44.9\%$ on Day 2, preceding the night-time cardiorespiratory arrest
Serum Electrolytes (Na ⁺ / K ⁺ / Cl ⁻ / Ca)	138 / 4.5 / 104 mmol/L; Ca ²⁺ 10.4 mg/dL	Post-arrest: 137 / 3.4 / 102 mmol/L; Ca ²⁺ 9.2 mg/dL	135 / 3.7 / 98 mmol/L	Na ⁺ 135–145; K ⁺ 3.5–5.1; Cl ⁻ 97–111 mmol/L; Ca ²⁺ 9.2–10.5 mg/dL	K ⁺ $\Delta\% = -24.4\%$ post-arrest (3.4 mmol/L) corrected to 3.7 mmol/L ($+8.8\%$) via electrolyte-enriched IVFD
Post-Arrest Arterial Blood Gas & Lactate (Oct 2, 21:00)	Not indicated (SpO ₂ 93–95%)	pH 7.28; PaCO ₂ 45 mmHg; PaO ₂ 64 mmHg; HCO ₃ ⁻ 21.6 mmol/L; BE -5.6 mmol/L; SaO ₂ 91%; Lactate 2.2 mmol/L	Resolved on PSIMV+ & T-piece (SpO ₂ 99%)	pH 7.35–7.45; PaCO ₂ 35–45; PaO ₂ 80–100 mmHg; Lactate < 2.0 mmol/L	Mixed acute respiratory/metabolic acidosis & hyperlactatemia ($+10.0\%$ above 2.0 mmol/L cutoff) post-arrest
Renal Function & Coagulation Profile (Oct 8)	Baseline normal	Baseline normal	Urea 13.3 mg/dL; Cr 0.3 mg/dL; PT 15.3 s (1.5 $\times$); APTT 28.9 s; INR 1.44	Urea 10–40 mg/dL; Cr 0.2–0.7 mg/dL; PT 10–12 s; INR 0.8–1.1	Preserved renal clearance; sepsis-induced vitamin K-responsive coagulopathy treated with Vitamin K 3 mg IV q24h
Microbiological Cultures & Antimicrobial Susceptibility	Sputum & Blood cultures: Sterile (no growth)	Endotracheal aspirate surveillance	Blood culture (Oct 9): Pseudomonas sp. (Ceftriaxone-R, Meropenem-S)	Sterile bloodstream	Targeted antibiotic escalation from Ceftriaxone (550 mg BID) to Meropenem (450 mg TID) resolved fever in 48 h

Abbreviations: APTT, activated partial thromboplastin time; BE, base excess; Cr, creatinine; Hb, hemoglobin; Hct, hematocrit; INR, international normalized ratio; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; NLR, neutrophil-to-lymphocyte ratio [(Segmented + Band Neutrophils) / Lymphocytes]; PaCO₂, partial pressure of arterial carbon dioxide; PaO₂, partial pressure of arterial oxygen; PLT, platelets; PT, prothrombin time; R, resistant; RBC, red blood cells; S, susceptible; SaO₂, arterial oxygen saturation; SII, systemic immune-inflammation index [PLT $\times$ NLR]; WBC, white blood cells.

By Day 8 (October 8, 2024), follow-up laboratory evaluation showed a -23.9% reduction in leukocyte count to $17.00 \times 10^3/\mu\text{L}$ (17,000/mm³). Platelets normalized by -46.6% to $280.0 \times 10^3/\mu\text{L}$ (280,000/mm³). Hemoglobin was 11.2 g/dL ($\Delta\% = -7.4\%$ vs. Day 1) and hematocrit 34.5%. Renal function was normal (urea 13.3 mg/dL, creatinine 0.3 mg/dL), as were electrolytes (Na⁺ 135 mmol/L, K⁺ 3.7 mmol/L, Cl⁻ 98 mmol/L). However, a secondary fever peak of 37.6 °C appeared on Day 8 and escalated to 38.7 °C on Day 9. It came with sepsis-induced coagulopathy, characterized by a prolonged Prothrombin Time (PT) of 15.3 seconds (1.5 $\times$ normal reference), Activated Partial Thromboplastin Time (APTT) of 28.9 seconds, and International Normalized Ratio (INR) of 1.44. Blood culture results returned on Day 9 isolated Pseudomonas sp., which was resistant to ceftriaxone and susceptible to meropenem (whereas initial admission blood and sputum cultures had been sterile). Ceftriaxone (550 mg IV BID) was discontinued at once on

Day 9 and escalated to intravenous meropenem 450 mg three times daily (1,350 mg/day = 122.7 mg/kg/day). Intravenous vitamin K 3.0 mg once daily was given alongside (Days 9–10). Fever fell to 37.5 °C within 24 hours. Apyrexia was then sustained ($\leq 37.0\text{--}37.5$ °C for > 5 consecutive days) from Day 11 through hospital discharge. Surgical tracheostomy on Day 9, together with meropenem and falling spasm frequency, was followed by cessation of the continuous intravenous midazolam and propofol infusions on Day 11 and a move to enteral baclofen (2.5 mg three times daily = 7.5 mg/day via NGT), so that intravenous diazepam boluses were tapered from 1.5 mg 12 $\times$ /day (18.0 mg/day, Days 8–10) to 1.5 mg 8 $\times$ /day (12.0 mg/day, Days 11–13) before being completely discontinued on Day 14 ($\Delta\% = -100.0\%$), as depicted in Figure 4.

(a) Stepwise Neuro-Sedative De-Escalation & Oral Baclofen Cross-Titration**(b) Hematologic-Inflammatory Kinetics & Targeted Meropenem Escalation**

Day 1 (Oct 1): Admission (Ceftriaxone) | Day 2 (Oct 2): Pre-Arrest Peak (Left Shift 6% Bands)
Day 8-9 (Oct 8-9): Pre-Tracheostomy (Meropenem Escalation)

Day 8-9 Sepsis & Coagulation Audit: Blood Culture: *Pseudomonas* sp. (Ceftriaxone-R, Meropenem-S); Escalated to Meropenem 450 mg IV TID (122.7 mg/kg/d); PT 15.3 s (1.5×), APTT 28.9 s, INR 1.44 → Vit K 3 mg IV; Defervescence (38.7 → 37.0 °C) achieved within 48 hours

Figure 4. Multimodal Neuro-Sedative Pharmacotherapy Titration and Hematologic-Inflammatory Biomarker & Carbapenem Escalation Trajectory. (a) Stepwise titration of intravenous diazepam boluses (peak 36.0 mg/day on Day 3 → discontinued Day 14), continuous midazolam (4.0-5.0 µg/kg/min) and ketamine (1.0 mg/kg/h) infusions, continuous propofol infusion (48.0 → 16.0 µg/kg/min), and Day 11 cross-titration to enteral GABA_B agonist baclofen (7.5 → 5.0 mg/day) immediately after surgical tracheostomy. (b) Serial total leukocyte count (WBC: 22.35 → 21.03 → 17.00 × 10³/µL; Δ% = -23.9%), platelet count (PLT: 524.0 → 536.0 → 280.0 × 10³/µL; Δ% = -46.6%), and Neutrophil-to-Lymphocyte Ratio (NLR: 5.20 → 7.36 [+41.5% pre-arrest surge] → 3.80), alongside Day 9 antibiotic escalation from ceftriaxone to meropenem (450 mg IV TID) and vitamin K (3.0 mg IV) for ceftriaxone-resistant *Pseudomonas* sp. bacteremia and coagulopathy (PT 15.3 s, INR 1.44).

Multimodal Pharmacotherapy and Global Literature Benchmarking

The complete multimodal pharmacotherapy and nutritional schedule is detailed in Table 4. On clinical grounds, rampant dental caries in a child with zero DTP doses, with no external wound, otitis media or fracture and with trismus preceding generalized spasms by one day, was judged the most likely portal of entry for the Ablett Grade IV tetanus; sterile

baseline blood and sputum cultures did not point to another source. High-dose GABAergic and propofol sedation and translaryngeal intubation were life-saving after the Day 2 cardiac arrest. They may also have suppressed cough clearance and favored lower-airway bacterial colonization, which preceded the Day 9 bloodstream infection. Surgical tracheostomy and meropenem escalation on Day 9 were followed by clinical improvement.

Table 4. Multimodal Neuro-Sedative, Anticonvulsant, Antimicrobial, and Enteral Nutritional Titration Protocol.

Therapeutic / Analytical Domain	Agent, Regimen, or Causality Instrument	Longitudinal Dosing / Scoring Trajectory (Days 1 to 37)	Pharmacological Target / Mechanistic Rationale	Verified Clinical Outcome / Attribution Verdict
Toxin Neutralization & Eradication	Human Tetanus Immunoglobulin (HTIG) + IV Metronidazole	HTIG: 3,000 IU IM single dose (Day 1); Metronidazole: 90–110 mg IV q6h (Days 1–13; 40 mg/kg/day)	HTIG neutralizes unbound circulating tetanospasmin; Metronidazole eradicates anaerobic C. tetani vegetative bacilli	Halted further neurotoxin translocation from odontogenic caries locus
Antimicrobial Stewardship & Escalation	IV Ceftriaxone → IV Meropenem → Oral Cefixime	Ceftriaxone 550 mg IV BID (Days 1–8) → Meropenem 450 mg IV TID (Days 9–19) → Cefixime 60 mg PO BID (Day 19+)	Empiric bronchopneumonia coverage escalated to carbapenem upon isolating Ceftriaxone-resistant Pseudomonas sp.	Complete defervescence within 48 h (38.7 → 37.5 → 37.0 °C; ≥ 5 days afebrile) & resolution of rales
Primary GABAA Agonist Bolus Titration	Intravenous Diazepam (Slow Bolus)	Day 1–2: 5 mg × 6 (30 mg/d) → Day 3: 3 mg × 12 (36 mg/d) → Day 4–5: 2 mg × 10 (20 mg/d) → Day 6: 2 mg × 8 → Day 7: 1 mg × 6 → Day 8–10: 1.5 mg × 12 (18 mg/d) → Day 11–13: 1.5 mg × 8 (12 mg/d) → Day 14: Stopped (Δ% = –100.0%)	Positive allosteric modulation of central and spinal GABAA chloride channels to counter Renshaw cell disinhibition	Controlled acute generalized spasms; completely weaned by Day 14 following oral Baclofen cross-titration
Continuous ICU Sedative Infusions	IV Midazolam + IV Ketamine → IV Propofol	Midazolam: 4.0–5.0 µg/kg/min (Days 2–10); Ketamine: 1.0 mg/kg/h (Days 2–3); Propofol: 48 µg/kg/min (Days 4–6) tapered to 16 µg/kg/min (Days 7–10); All infusions stopped Day 11	Synergistic GABAA potentiation (Midazolam/Propofol) and NMDA glutamate antagonism + sympathetic stabilization (Ketamine)	Maintained ventilator synchrony and blunted catecholamine surges; 100% discontinued 24 h post-tracheostomy
Enteral GABAB Rehabilitation Agonist	Oral / NGT Baclofen	Days 11–19: 2.5 mg TID (7.5 mg/day = 0.68 mg/kg/day); Day 21+ outpatient: tapered to 2.5 mg BID (5.0 mg/day)	Presynaptic GABAB receptor activation inhibiting spinal monosynaptic and polysynaptic α-motor neuron excitation	Enabled complete cessation of IV benzodiazepines/propofol and full resolution of spasticity by Day 21
Enteral Nutritional & Fluid Escalation	Staged Nasogastric Liquid Feeding + IVFD + Vitamin K	NPO (Day 1) → 6×25 mL (150 mL/d, Day 2) → 6×75 mL (450 mL/d, Day 9) → 6×100 mL (600 mL/d, Day 10) → 6×120 mL (720 mL/d, Day 11–13) → 6×160 mL (960 mL/d, Day 14–19) → 6×200 mL (1,200 mL/d, +700.0% vs. Day 2); Vit K 3 mg IV OD (Days 9–10)	Prevents mucosal atrophy, reverses –3.0 SD wasting catabolism (REE 902.1 kcal/day; target RDA 1,260 kcal/day), and corrects INR 1.44	100% enteral tolerance without aspiration; coagulopathy corrected and somatic recovery initiated

Abbreviations: ADR, adverse drug reaction; BID, twice daily; GABA, gamma-aminobutyric acid; HTIG, human tetanus immunoglobulin; ICU, intensive care unit; IM, intramuscular; INR, international normalized ratio; IV, intravenous; IVFD, intravenous fluid drip; NGT, nasogastric tube; NMDA, N-methyl-D-aspartate; NPO, nil per os; OD, once daily; PO, per os; q6h, every 6 hours; RDA, recommended dietary allowance; REE, resting energy expenditure; TID, three times daily; WHO-UMC, World Health Organization-Uppsala Monitoring Centre.

To place the clinical impact of our airway and intensive care strategy against international standards, the index patient's clinical profile, tracheostomy timing, ventilator weaning kinetics, and survival outcome were benchmarked against contemporary tetanus cohorts and case investigations published between 2021 and 2026, as synthesized in Table 5.^{9,12,17,29–32} The retrospective ICU cohort by Naz et al. (2021; N = 48 adult tetanus patients, 89.5% tracheostomized, overall survival 81.3%) is a landmark reference. In that cohort, early tracheostomy (performed within ≤ 7 days of ICU admission in 50.0% of patients) achieved 100.0% survival (24/24 survived vs. 10/19 [52.6%] in late tracheostomy). It also gave 0.0% incidence of ventilator-associated pneumonia (0/16 VAP cases vs. 16/16 [100.0%] in late tracheostomy) and a lower requirement for prolonged mechanical ventilation (34.5% vs. 65.5%). Delayed translaryngeal intubation, by contrast, led to

endotracheal tube blockage or dislodgement requiring emergency re-intubation in 70.6% (12/17) of patients. Our pediatric patient showed a similar sequence of events. Prolonged translaryngeal intubation through Day 7 culminated in copious sputum stasis and immediate post-extubation HFNC failure (ROX index = 2.2), requiring emergency re-intubation. Surgical tracheostomy on Day 9 (7 days after initial intubation) was followed by a different course. The child was liberated from positive-pressure mechanical ventilation onto a T-piece within 24 to 48 hours (Day 11). Discharge followed on Day 19–20. Trismus and spasticity had resolved completely by Day 21. Elective decannulation was scheduled for Day 39, without any neurological sequelae.

Table 5. Global Comparative Benchmarking of the Present Pediatric Odontogenic Grade IV Tetanus Case Against Contemporary Published Tetanus Cohorts and Case Investigations (2021–2026).

Study & Geographic Setting	Study Design & Population	Portal of Entry & Vaccine Status	Severity Grade & Airway Strategy	Complications & Neuro-Sedative Regimen	Clinical Outcome & Key Comparative Insight
Present Study (2026) — Padang, West Sumatera, Indonesia	Case report (n = 1; 4 yr 2 mo male, WAZ –3.0 SD)	Odontogenic (rampant dental caries; zero external trauma); Unimmunized (0 DTP)	Ablett Grade IV; ETT on Day 2 (post-arrest), HFNC failure (ROX 2.2) on Day 7, Surgical Tracheostomy on Day 9	Apneic cardiac arrest (ROSC), Ceftriaxone-R Pseudomonas sp. bacteremia, INR 1.44; HTIG 3,000 IU, Midazolam, Ketamine, Propofol, Diazepam → Oral Baclofen	100% survival; weaned to T-piece 24 h post-tracheostomy (Day 11), discharged Day 19–20, zero neurological sequelae at Day 37
Naz et al. (2021) — Karachi, Pakistan	Retrospective ICU Cohort (N = 48 adults; median age 25.5 yr [IQR 22–35]; 91.7% male)	Lower limb (66.7%), upper limb (20.8%), cephalic (6.3%); 12.5% recent TT booster	Ablett Grade II (10.4%), III (79.2%), IV (10.4%); Tracheostomy in 89.5% (Early ≤ 7 d: 50.0%; Late > 7 d: 39.6%)	VAP in 33.3% (0% in early vs. 100% of VAP in late tracheostomy), ETT blockage/reintubation in 70.6% of delayed group; Diazepam, Propofol (25%), MgSO ₄	Overall survival 81.3% (100% in early vs. 52.6% in late tracheostomy); validates rapid MV weaning after tracheostomy
Mondkar et al. (2021) — Mumbai, India	Tertiary PICU retrospective cohort (N = 25 pediatric patients aged 6 months to 12 years; mean age 6.6 years)	Post-traumatic (52%) and otogenic (40%) portals; 15 unimmunized and 10 partially immunized	18 moderate and 7 severe tetanus; 8 primary tracheostomy and 11 primary endotracheal intubation	Pneumonia (58%), acute kidney injury after rhabdomyolysis (33%), sepsis (29%), DIC (25%); oral diazepam most common, then midazolam, vecuronium and magnesium sulfate	Mortality 32%; short onset period (< 48 hours) and acute kidney injury were associated with death (from the abstract of the cited article)
Akbar et al. (2022) — Makassar, Indonesia	Case Report & Literature Review (Odontogenic generalized tetanus)	Odontogenic infection (carious molar defect); incomplete tetanus immunization	Generalized tetanus; non-tracheostomized airway surveillance and intensive pharmacotherapy	Severe trismus, dysphagia, and generalized spasms managed with HTIG, metronidazole, and diazepam titration	Confirmed carious dental tissue as a critical clandestine portal of entry in Indonesian patients lacking trauma history
Fafolahan et al. (2023) — Abeokuta, Nigeria	Pediatric Case Series (N = 2 children: 14 yr female & 7 yr male)	Peripheral limb injuries in unimmunized children	Generalized tetanus managed with medical spasmodic plus structured inpatient pediatric physiotherapy	Severe limb hypertonicity and joint stiffness; treated with soft-tissue massage, passive stretching, and mobilization over 8–14 days	Achieved 4/5 Oxford muscle strength and functional independence; supports outpatient physiotherapy integration at Day 21

Study & Geographic Setting	Study Design & Population	Portal of Entry & Vaccine Status	Severity Grade & Airway Strategy	Complications & Neuro-Sedative Regimen	Clinical Outcome & Key Comparative Insight
Boer et al. (2024) — Netherlands / Global	Case Report & Diagnostic-Therapeutic Analysis	Minor peripheral portal in under-immunized host	Severe generalized tetanus requiring ICU mechanical ventilation and early tracheostomy	Refractory spasms and autonomic instability managed with multimodal GABAergic and sympatholytic therapy	Demonstrated that early tracheostomy prevents laryngeal trauma and facilitates ICU mobilization
Aiello et al. (2024) — Jacksonville, USA	Case report (62-year-old woman; generalized grade 4 tetanus)	Laceration; vaccination status not stated in the abstract	Interventions beyond tetanus immunoglobulin and antibiotics were needed for progressive spasms	Intrathecal baclofen (lumbar drain, then tunneled catheter with external pump) after oral baclofen failed	Transition from intrathecal to oral baclofen allowed discharge (adult patient; intrathecal, not oral, baclofen was the treatment)
Gu et al. (2026) — Hangzhou, China	Adult 10-year ICU cohort (N = 36 Ablett Grade III–IV severe tetanus patients)	Rural trauma and occult portals; low post-exposure prophylaxis uptake	Ablett Grade III–IV; majority required tracheostomy, deep sedation, and mechanical ventilation (median ICU stay 27.5 d)	Pulmonary infections in 80.6% and antimicrobial-resistant isolates in 55.6%; in-hospital mortality 16.7%	Confirms high burden of resistant nosocomial pathogens in Grade IV tetanus and survival benefit of targeted ICU care

Abbreviations: DTP, diphtheria-tetanus-pertussis vaccine; ETT, endotracheal tube; GABA, gamma-aminobutyric acid; HFNC, high-flow nasal cannula; HTIG, human tetanus immunoglobulin; ICU, intensive care unit; INR, international normalized ratio; IQR, interquartile range; MgSO₄, magnesium sulfate; MV, mechanical ventilation; PICU, pediatric intensive care unit; R, resistant; ROSC, return of spontaneous circulation; ROX, respiratory rate-oxygenation index; TT, tetanus toxoid; VAP, ventilator-associated pneumonia; WAZ, weight-for-age Z-score.

Adverse events

The complications of the course were the Day 2 apneic cardiac arrest, the failed extubation on Day 7, and ceftriaxone-resistant *Pseudomonas* sp. bacteremia with coagulopathy on Day 9, all described above. Enteral feeding was tolerated without aspiration events.

4. Discussion

Odontogenic Portal Pathogenesis and Multi-Tier Neuro-Autonomic Cascade

This case report shows that fulminant pediatric Ablett Grade IV generalized tetanus from an odontogenic portal of entry can end in full neurological and cardiorespiratory recovery. This held even though the course was complicated by severe baseline wasting (WAZ -3.0 SD, WHZ -3.0 SD), Stage 2 sympathetic hypertension (130/85 mmHg; MAP 100.0 mmHg), apneic cardiac arrest requiring cardiopulmonary resuscitation, failed translaryngeal extubation (ROX index 2.2), and ceftriaxone-resistant *Pseudomonas* sp. bacteremia. Recovery followed a synchronized multimodal intensive care protocol anchored by surgical tracheostomy, targeted carbapenem escalation, enteral GABAB agonist cross-titration, and aggressive nasogastric nutritional rehabilitation.^{33,34} In developing tropical nations such as Indonesia, pediatric tetanus persists as a sentinel indicator of immunization inequities and neglected primary dental care.³⁴ The Indonesian Ministry of Health incorporates DTP vaccination into the mandatory national Expanded Programme on Immunization (EPI). Our patient, however, had received zero doses of DTP vaccine by 4 years 2 months of age. His circulating anti-tetanospasmin IgG titer was below the minimum protective threshold of 0.01 IU/mL. Natural infection does not help here. Picogram quantities of neurotoxin are insufficient to elicit adaptive humoral

immunity in natural tetanus. Unimmunized children therefore remain entirely defenseless. Spores can germinate in minor or occult mucosal lesions. These children need structured catch-up DTP immunization prior to or following hospital convalescence.

A diagnostic point from this case is that rampant dental caries (caries dentis) was the most likely portal of entry in a preschool child with zero history of external trauma. Pediatricians in clinical practice often associate tetanus exclusively with rusty nails, soil-contaminated lower-limb punctures, or unsterile umbilical cord care. Diagnosis can then be delayed when a child presents with isolated cough, fever, and lockjaw. Carious dental cavities, however, account for 23.0% of oral tetanus cases. Severe generalized tetanus evolving from carious molars has been increasingly recognized in Indonesian patients. Deep carious lesions extend into the pulp chamber and periapical tissues. There they create an avascular, necrotic pocket with an extremely low oxidation-reduction potential. Ubiquitous *C. tetani* spores, ingested via contaminated fingers or food in households with poor sanitation, can then germinate and release tetanospasmin. Trigeminal sensory and motor nerve endings densely innervate the dental pulp and periodontal ligament, so intraoral tetanospasmin gains immediate entry to cranial nerve V axons. The toxin then travels a short retrograde axonal pathway to the pons and medulla. That route explains why our patient progressed from initial trismus on Day -3 to generalized opisthotonus and tonic convulsions within 24 hours. The five-tier pathophysiological, neuro-autonomic, cardiorespiratory, and surgical airway cascade governing this clinical trajectory is illustrated in Figure 5.

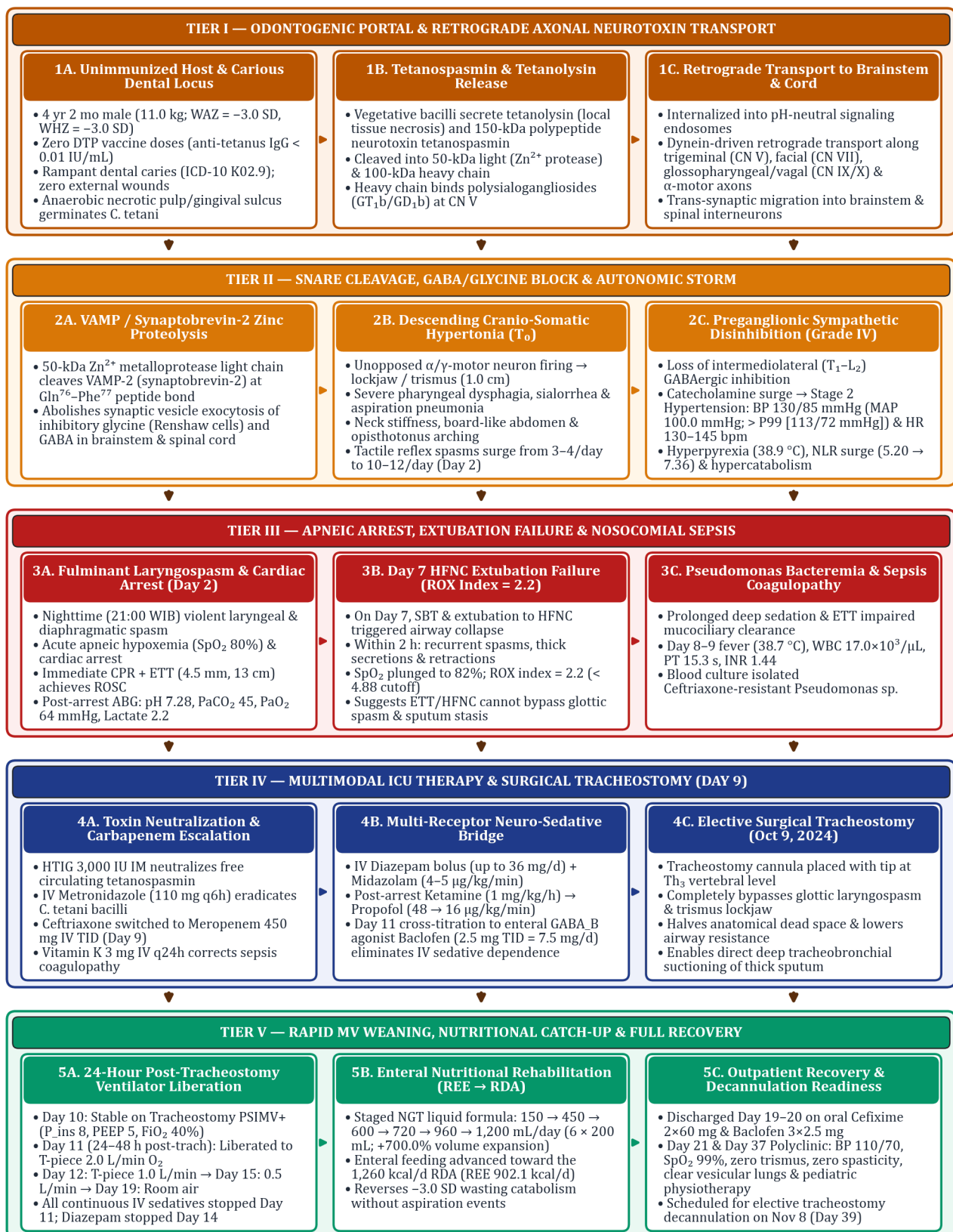


Figure 5. Proposed Five-Tier, Fifteen-Node Pathophysiological, Neuro-Autonomic, Cardiorespiratory, and Surgical Airway Cascade in Pediatric Odontogenic Aplett Grade IV Tetanus. Tier I: Unimmunized host (0 DTP doses, WAZ -3.0 SD), anaerobic *C. tetani* spore germination in rampant dental caries, and retrograde axonal transport of tetanospasmin along cranial nerve V. Tier II: Zinc metalloprotease cleavage of VAMP-2/synaptobrevin-2, loss of spinal Renshaw glycinergic and brainstem GABAergic inhibition, descending trismus/opisthotonus, and intermediolateral sympathetic storm (BP 130/85 mmHg > P99). Tier III: Day 2 nighttime apneic cardiac arrest (SpO₂ 80%, ROSC via CPR + 4.5 mm ETT), Day 7 HFNC extubation failure (ROX index = 2.2), and Day 8–9 ceftriaxone-resistant *Pseudomonas* sp. bacteremia with INR 1.44 coagulopathy. Tier IV: Multimodal ICU intervention combining HTIG (3,000 IU IM), metronidazole, meropenem escalation, GABAA/NMDA/GABAB sedation bridge, and Day 9 surgical tracheostomy (tip at Th3). Tier V: Rapid 24-hour ventilator weaning to T-piece (Day 11), enteral nutritional catch-up (1,200 mL/day), complete neurological recovery by Day 21, and elective decannulation readiness at Day 37–39. This cascade is proposed from the literature; the molecular steps were not measured in this patient.

Cardiorespiratory Mechanics of ROX Index Extubation Failure and Rapid Post-Tracheostomy Liberation

The cardiorespiratory events observed on Hospital Day 2 (apneic cardiac arrest at 21:00 WIB) and Hospital Day 7 (HFNC extubation failure with ROX index 2.2) offer physiological lessons about airway mechanics in severe pediatric tetanus.^{35–37} Two quantitative warning signals appeared together before the Day 2 arrest. Daily tactile spasms tripled, from 3–4 episodes/day to 10–12 episodes/day. The leukocyte differential also showed an acute left shift, with band neutrophils rising by +500.0% from 1% to 6% alongside lymphopenia of 11%. This drove the Neutrophil-to-Lymphocyte Ratio (NLR) from 5.20 to 7.36 (+41.5%) and the Systemic Immune-Inflammation Index (SII) from 2,724.8 to $3,946.9 \times 10^3/\mu\text{L}$ (+44.9%). Recent observational studies by Dai et al. (2024), Hai et al. (2024), and Lu et al. (2022) have validated elevated SII, heart rate variability alterations, and continuous physiological wearable sensing as sensitive predictors of severe tetanus progression and ICU autonomic instability, and these markers reflect catecholamine- and cytokine-mediated bone marrow demargination of immature neutrophils together with stress-induced lymphocyte apoptosis.^{35–37}

This case suggests why translaryngeal endotracheal intubation and post-extubation High-Flow Nasal Cannula (HFNC) support may be insufficient during the active phase of Ablett Grade IV tetanus.^{38,39} Our patient was extubated to HFNC on Day 7 after a successful spontaneous breathing trial and initial sedation taper. He decompensated within 2.0 hours. He had recurrent spasms, copious thick secretions, intercostal retractions, SpO₂ 82.0%, and a calculated ROX index of 2.2. In pediatric intensive care validation cohorts by Yuniar et al. (2024), a low ROX index at 60 to 120 minutes after HFNC initiation is a strong quantitative predictor of HFNC failure and need for positive-pressure re-intubation.³⁸ In Grade IV tetanus, an endotracheal tube passes through the oral cavity, pharynx, and vocal cords. It acts as a continuous nociceptive foreign-body stimulus against hyperexcitable trigeminal, glossopharyngeal, and superior laryngeal reflex arcs, and the result is repetitive reflex laryngospasm, which calls for high-dose continuous infusions of midazolam (4.0–5.0 $\mu\text{g/kg/min}$) and propofol (48.0 $\mu\text{g/kg/min}$).^{39–42} Once the ETT was removed on Day 7, residual tetanospasmin-mediated trismus (1.0 cm opening), pharyngeal dysphagia, and glottic hyperreactivity obstructed the upper airway at once. They also prevented clearance of thick bronchopneumonic secretions, which predisposes to nosocomial *Pseudomonas aeruginosa* and *Acinetobacter* ventilator-associated pneumonia and bacteremia.^{43,44} A violent generalized spasm supervened at 21:00 WIB on Day 2, and tonic contraction of the intrinsic laryngeal adductors and thoracoabdominal musculature occurred at the same moment and precipitated acute asphyxial hypoxemia (SpO₂ 80.0%), mixed acidosis (pH 7.28, PaCO₂ 45.0 mmHg, base excess –5.6 mmol/L, lactate 2.2 mmol/L), and cardiac arrest. Prompt pediatric CPR and intubation achieved immediate ROSC, because asphyxial pediatric arrests respond rapidly to oxygenation restoration.^{45–47} Elective surgical tracheostomy on Day 9 (with the cannula tip positioned at the Th3 vertebral level) offered a contrast. It bypassed the spastic oropharyngeal and glottic segments. Direct tracheobronchial suctioning became possible, and weaning onto a T-piece within 24 to 48 hours (Day 11) followed, with cessation of all continuous intravenous sedatives. Staged enteral nasogastric nutritional advancement followed, reaching 1,200 mL/day (toward the 1,260.0 kcal/day RDA target; REE 902.1 kcal/day) to reverse –3.0 SD wasting.^{47,48}

Molecular Neuropharmacology of Sequential GABAA, NMDA, and GABAB Receptor Modulation in Grade IV Tetanus

The neuro-sedative titration trajectory executed in this case offers a clear model for balancing spasm suppression against sedative-associated morbidity in the pediatric intensive care unit. Tetanospasmin irreversibly cleaves presynaptic VAMP-2 / synaptobrevin-2 within spinal Renshaw cells and brainstem inhibitory interneurons. Functional recovery of endogenous glycinergic and GABAergic exocytosis therefore takes approximately 2 to 4 weeks. This is the time needed for de novo axonal sprouting and synthesis of intact SNARE proteins. During the acute hyperexcitable window (Days 1 to 10), drugs must artificially restore postsynaptic chloride conductance or inhibit excitatory glutamatergic transmission. Intravenous diazepam (administered up to 36.0 mg/day = 3.27 mg/kg/day on Day 3) and continuous midazolam infusion (4.0–5.0 $\mu\text{g/kg/min}$) act as positive allosteric modulators at the benzodiazepine binding site (α/γ subunit interface) of postsynaptic GABAA receptors, and both raise the frequency of chloride channel opening in response to residual ambient GABA. Ketamine came next. After the Day 2 cardiac arrest, continuous low-dose intravenous ketamine was added (1.0 mg/kg/hour on Days 2–3), and it provided non-competitive antagonism of central N-methyl-D-aspartate (NMDA) glutamate receptors and blocked excitatory wind-up in the spinal dorsal horn. It also preserved systemic vascular resistance during post-ROSC myocardial stunning.

During Days 4 to 10, ketamine gave way to continuous intravenous propofol (48.0 $\mu\text{g/kg/min}$ on Days 4–6, tapered to 16.0 $\mu\text{g/kg/min}$ on Days 7–10). Propofol provided direct GABAA receptor agonism (prolonging chloride channel open duration at β -subunit transmembrane domains) and potent suppression of sympathetic catecholamine outflow, so that blood pressure fell from 123/83 mmHg (MAP 96.3 mmHg) to 101/67 mmHg (MAP 78.3 mmHg) by Day 6. Prolonged high-dose propofol (> 4.0 mg/kg/hour or > 66.7 $\mu\text{g/kg/min}$ for > 48 hours) in young children carries the well-known hazard of Propofol-Related Infusion Syndrome (PRIS). The syndrome is characterized by mitochondrial fatty acid oxidation uncoupling, metabolic acidosis, rhabdomyolysis, and cardiac failure. High-dose intravenous diazepam, for its part, contains a propylene glycol vehicle that can accumulate and cause hyperosmolar lactic acidosis. Propofol was therefore titrated rapidly downward to 16.0 $\mu\text{g/kg/min}$ on Day 7. Both propofol and midazolam were discontinued on Day 11. This was enabled by the Day 9 surgical tracheostomy and the simultaneous start of enteral baclofen (2.5 mg three times daily = 7.5 mg/day), and it was a major safety milestone. Baclofen is a structural p-chlorophenyl derivative of GABA. The drug selectively activates metabotropic G-protein-coupled GABAB receptors on presynaptic Ia sensory afferents and postsynaptic spinal interneurons. It inhibits voltage-gated calcium influx (N- and P/Q-type Ca²⁺ channels) and enhances inwardly rectifying potassium (GIRK) conductance. In this way it suppresses monosynaptic and polysynaptic extensor spasticity with less cortical respiratory depression than benzodiazepines. The targeted receptor switch permitted full cessation of intravenous diazepam by Day 14 and smooth outpatient rehabilitation on oral baclofen (tapered to 2.5 mg twice daily on Day 21).

Pathophysiology of Nosocomial Pseudomonas Bacteremia, Sepsis Coagulopathy, and Targeted Carbapenem Stewardship

Secondary fever (38.7 °C), persistent leukocytosis ($17.00 \times 10^3/\mu\text{L}$), vitamin K-responsive coagulopathy (PT 15.3 seconds, INR 1.44), and ceftriaxone-resistant *Pseudomonas* sp.

bloodstream infection emerged on Days 8 to 9. The picture shows how tetanic neuromuscular pathology and intensive care unit nosocomial risk act on each other. Patients with severe generalized tetanus show an extraordinarily high incidence of lower respiratory tract colonization and ventilator-associated pneumonia (33.3% to 80.6%). Standard semi-recumbent positioning alone does not control it. Several synergistic mechanisms drive this vulnerability. First, severe trismus and pharyngeal dysphagia cause continuous micro-aspiration of saliva colonized by oral biofilm bacteria from carious teeth. Second, repetitive opisthotonic spasms prevent stable 30° head-of-bed elevation and promote gastroesophageal reflux. Third, prolonged translaryngeal intubation and deep GABAergic sedation abolish the cough reflex and impair mucociliary escalator velocity. The outcome is stagnant pools of viscid bronchial secretions (sputum stasis).

In pediatric intensive care units across Southeast Asia and the Middle East, *Pseudomonas aeruginosa* and *Acinetobacter* species are the predominant Gram-negative pathogens in ventilated patients. They often harbor intrinsic or acquired chromosomal AmpC beta-lactamases, efflux pumps (MexAB-OprM), and porin (OprD) modifications. These confer full resistance to third-generation cephalosporins such as ceftriaxone. Empirical ceftriaxone (550 mg IV BID) given from Day 1 controlled early community-acquired aspiration flora. Ceftriaxone does not cover *Pseudomonas aeruginosa*, which is intrinsically resistant, so the isolate was probably already resistant rather than selected by treatment. The organism translocated across the inflamed alveolar-capillary membrane into the bloodstream and triggered extrinsic pathway coagulation factor consumption (prolonging PT to 15.3 seconds and INR to 1.44 while sparing platelets at $280.0 \times 10^3/\mu\text{L}$ and APTT at 28.9 seconds). Culture-guided antibiotic escalation followed immediately on Day 9, to high-dose intravenous meropenem (450 mg every 8 hours = 122.7 mg/kg/day). The regimen was paired with parenteral vitamin K (3.0 mg IV once daily for 2 days) to restore hepatic γ -carboxylation of Factors II, VII, IX, and X, while surgical tracheostomy cleared stagnant lower-airway secretions. These measures achieved rapid bloodstream sterilization and full defervescence within 48 hours.

Bioenergetics of Severe Wasting (−3.0 SD), Autonomic Hypercatabolism, and Multidisciplinary Rehabilitation

A third determinant of survival in this child was the proactive management of severe baseline undernutrition (weight 11.0 kg, height 94.0 cm, BMI 12.45 kg/m²; WAZ = −3.0 SD, WHZ = −3.0 SD, HAZ between −3.0 and −2.0 SD) during an intensely hypercatabolic illness. Children with severe wasting have depleted skeletal muscle glycogen and visceral protein reserves. Their diaphragm and intercostal muscles therefore fatigue quickly during sustained tetanic contractions. The metabolic load is also high. Ablett Grade IV tetanus imposes a heavy metabolic stress factor ($1.5 \times$ basal metabolic rate), driven by continuous isometric muscular work, hyperpyrexia (38.9 °C), and sympathetic catecholamine-induced glycogenolysis and proteolysis. The child's Resting Energy Expenditure rose to 902.1 kcal/day. His catch-up caloric requirement rose to 1,260.0 kcal/day (90 kcal/kg Ideal Body Weight of 14.0 kg).

In accordance with contemporary pediatric intensive care nutrition guidelines, early trophic enteral feeding via nasogastric tube ($6 \times 25 \text{ mL} = 150.0 \text{ mL/day}$) was started on Day 2. The aim was to preserve intestinal mucosal tight-junction integrity and prevent bacterial translocation. Intravenous fluids were restricted by 10.0% (950.0 mL/24 hours) during acute cerebral and hemodynamic instability.⁴⁸

Surgical tracheostomy secured the airway on Day 9. The autonomic storm subsided. Enteral liquid formula delivery was then advanced stepwise from 450.0 mL/day (Day 9) to 600.0 mL/day (Day 10), 720.0 mL/day (Days 11–13), 960.0 mL/day (Days 14–19), and finally 1,200.0 mL/day ($6 \times 200 \text{ mL}$; 109.1 mL/kg/day). Gastrointestinal tolerance was complete, without aspiration events. There were zero aspiration events. Structured outpatient pediatric physiotherapy (soft-tissue stretching, passive range-of-motion mobilization, and postural re-education) was added alongside low-dose oral baclofen and mucolytic ambroxol at Day 21. This prevented permanent myostatic joint contractures. It culminated in full functional restoration and readiness for tracheostomy decannulation (scheduled for Day 39).

Strengths and limitations

This report has limitations. It describes one patient, so there is no comparison group and the findings cannot be generalized. Tracheostomy, meropenem, enteral baclofen and the natural decline of spasms occurred together, so the improvement cannot be attributed to any single intervention. The mechanisms discussed (SNARE cleavage, receptor pharmacology, impaired mucociliary clearance) come from the literature and were not measured here. The inflammatory indices were available on two days before the arrest and cannot be called predictive, and some reference ranges were not pediatric. Its strengths are the dense serial documentation across eight epochs, the objective ROX-based description of extubation failure, the culture and susceptibility data, and follow-up to Day 37.

5. Conclusion

This case report suggests that unimmunized preschool children with rampant dental caries are vulnerable to fulminant odontogenic Ablett Grade IV generalized tetanus. This holds even without any external cutaneous trauma. Severe sympathetic dysautonomia (Stage 2 hypertension of 130/85 mmHg), surging tactile reflex spasms (10–12 episodes/day with NLR 7.36 and SII $3,946.9 \times 10^3/\mu\text{L}$), and copious oropharyngeal secretions precipitated nighttime apneic cardiac arrest on Day 2. They preceded post-extubation High-Flow Nasal Cannula failure on Day 7 (ROX index 2.2). The clinical trajectory improved after elective surgical tracheostomy on Day 9 (cannula tip at Th₃), coupled with culture-guided meropenem escalation for ceftriaxone-resistant *Pseudomonas* sp. bacteremia. Tracheostomy bypassed upper-airway laryngospasm and allowed deep tracheobronchial clearance. Within 24 to 48 hours (Day 11), the child was fully liberated from positive-pressure mechanical ventilation onto a T-piece, and continuous intravenous midazolam and propofol infusions were stopped. Intravenous diazepam ended by Day 14 via enteral baclofen cross-titration. Full enteral nutritional rehabilitation (1,200 mL/day) was reached. The child survived without neurological sequelae. Surgical tracheostomy may be considered early in pediatric Grade IV tetanus complicated by extubation failure or refractory laryngospasm. It should be accompanied by catch-up DTP immunization, definitive dental caries eradication, and community-wide vaccine education.

Declarations

Ethics approval and consent to participate

This report was approved by the CMHC Research Center Ethics Committee, Indonesia (protocol approval number 2024/0418) and conducted in accordance with the Declaration of Helsinki. Written informed consent was

obtained from both parents for treatment, participation and publication.

Consent for publication

Written informed consent for publication of anonymized clinical data, laboratory records, growth charts and thoracic radiographs with anonymized margins was obtained from the patient's parents (legal guardians).

Data availability

All longitudinal clinical, hemodynamic, ventilatory, hematologic, microbiological and nutritional data analyzed during this study are included in this article, Tables 1–5 and Figures 1–5.

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Conflict of interest

The authors declare no conflicts of interest.

Author contributions

D.R.: Conceptualization; Methodology; Data curation; Formal analysis; Investigation; Visualization; Writing – original draft. I.I.: Supervision; Project administration; Resources; Validation; Writing – review & editing. A.T.W.: Investigation; Resources; Supervision; Writing – review & editing. All authors approved the final manuscript.

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Use of artificial intelligence

No generative artificial intelligence tools were used in the preparation of this manuscript.

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