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Critical Care Approach to Severe Tetanus with Septic Shock: A Case Report

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A B S T R A C T

Introduction: Tetanus, caused by *Clostridium tetani* neurotoxin, remains a life-threatening condition, particularly in regions with suboptimal vaccination coverage. Severe tetanus often necessitates intensive care unit (ICU) admission due to profound muscle spasms, respiratory failure, and autonomic nervous system dysfunction. Concomitant septic shock further complicates management and worsens prognosis. This report details the critical care management of a patient presenting with severe tetanus complicated by septic shock. **Case presentation:** A 41-year-old male presented with generalized muscle rigidity, trismus, and recurrent severe spasms following a puncture wound from bamboo 10 days prior. He had no prior tetanus immunization history. Upon admission, he exhibited signs of respiratory distress (Sp90% on a 15L non-rebreather mask) and septic shock (tachycardia, hypotension requiring vasopressors, SOFA score 7). Diagnosis of severe tetanus (Ablett Grade III) with respiratory failure and septic shock was made. Management involved immediate intubation, mechanical ventilation, administration of human tetanus immunoglobulin (HTIG), intravenous metronidazole, aggressive sedation with benzodiazepines (diazepam infusion) and neuromuscular blockade (vecuronium infusion), hemodynamic support with intravenous fluids and noradrenaline infusion, early tracheostomy, and comprehensive supportive care including nutritional support and VTE prophylaxis. His ICU stay was complicated by autonomic instability and ventilator-associated pneumonia (VAP). **Conclusion:** Managing severe tetanus complicated by septic shock requires a prompt, multidisciplinary critical care approach. Key elements include securing the airway, controlling spasms and rigidity, neutralizing toxins, eradicating the source, managing autonomic instability, aggressive sepsis management according to current guidelines, and providing meticulous supportive care. Despite significant challenges, a favorable outcome is possible with comprehensive ICU management.

1. Introduction

Tetanus is an acute and potentially fatal neurological disorder caused by tetanospasmin, a potent neurotoxin produced by the anaerobic, spore-forming bacterium Clostridium tetani. The spores of this bacterium are ubiquitous in the environment, residing in soil and having the capacity to enter the human body through various means, including wounds, abrasions, or even minor breaches in the skin. Despite the existence of effective vaccination programs designed to prevent tetanus, it remains a significant public health issue, particularly in low- and middle-income countries (LMICs) where immunization coverage may be suboptimal and access to adequate medical care, especially intensive care units (ICUs), is limited. The persistent global occurrence of tetanus results in substantial morbidity and mortality, highlighting the importance of continued vigilance and effective

healthcare strategies to combat this disease. The clinical manifestations of tetanus are characterized by muscle rigidity and painful reflex spasms, which can vary in severity. Severe forms of tetanus, such as Ablett Grades III and IV, are associated with generalized, prolonged spasms, the potential for opisthotonos (severe hyperextension and arching of the back), respiratory compromise due to laryngospasm or chest wall rigidity, and significant autonomic nervous system (ANS) dysfunction. Autonomic nervous system dysfunction, also referred to as dysautonomia or autonomic storm, is a particularly concerning complication of severe tetanus. It manifests as fluctuations in heart rate and blood pressure, including severe hypertension and tachycardia alternating with profound hypotension and bradycardia, as well as hyperpyrexia (high fever) and diaphoresis (excessive sweating). The presence of ANS dysfunction significantly increases the risk of mortality in tetanus cases, underscoring the need for careful monitoring and management of these symptoms.[1-4]

The management of severe tetanus invariably necessitates admission to an Intensive Care Unit (ICU). This level of care is required to provide essential interventions such as airway management, which often includes intubation, mechanical ventilation, and in some cases, tracheostomy. Deep sedation and neuromuscular blockade are crucial to control the severe muscle spasms and rigidity that characterize tetanus. Furthermore, patients with severe tetanus may require cardiovascular support to manage hemodynamic instability, as well as meticulous supportive care to address the various complications of the disease and its treatment. The co-occurrence of septic shock in a patient with severe tetanus further complicates the clinical picture and presents additional challenges for management. While tetanus itself is a toxemia resulting from the effects of the bacterial toxin, the initial wound through which the bacteria entered the body can also be a source of bacterial infection, potentially leading to sepsis. Additionally, the prolonged ICU stays often required for tetanus treatment increase the risk of nosocomial infections, such as ventilator-associated pneumonia or catheter-related bloodstream infections, which can further contribute to the development of sepsis and septic shock. Septic shock is a life-threatening condition characterized by persistent hypotension requiring vasopressors despite adequate fluid resuscitation, along with evidence of organ dysfunction. Septic shock carries a high mortality rate on its own and adds another layer of complexity to the hemodynamic management of a patient already experiencing tetanus-induced autonomic instability.[5-7]

The management of concurrent life-threatening conditions such as severe tetanus and septic shock demands a sophisticated, resource-intensive, and multidisciplinary critical care approach. Effective management requires the coordinated efforts of various healthcare professionals, including intensivists, nurses, pharmacists, and other specialists, working together to address the complex needs of these patients.[8-10] This report presents the case of a 41-year-old male who developed severe tetanus complicated by septic shock. The report provides a detailed account of the diagnostic challenges encountered, the intensive care management strategies employed, and the patient's clinical course, highlighting the critical interventions necessary for survival in such complex and challenging cases.

2. Case Presentation

The patient, a 41-year-old male, was referred to our tertiary center's emergency department, presenting with a constellation of acute symptoms that indicated a critical underlying condition. His chief complaints at the time of arrival were acute shortness of breath and generalized body stiffness or rigidity, which collectively pointed towards a severe systemic disturbance. Delving into the patient's history, it was revealed that approximately ten days prior to his presentation, he had sustained a puncture wound on his right thumb. The injury was inflicted by a piece of bamboo, an important detail given the potential for such injuries to introduce Clostridium tetani spores, the causative agent of tetanus. Initial wound care had been administered at a local facility, but crucially, there was no documented evidence of any prior tetanus vaccination in this patient. This lack of immunization history is a significant factor in the development of tetanus, as vaccination is the primary preventive

measure against the disease. In the days following the injury, the patient's condition progressively deteriorated. Three days before his transfer to our center, he was admitted to the referring hospital due to increasing muscle stiffness, which had already led to a diagnosis of tetanus. Despite the initial diagnosis and presumably some level of supportive care, the patient's clinical course continued to decline. The severity of his condition escalated dramatically in the 24 hours preceding his transfer, marked by the onset of severe dyspnea and the occurrence of two episodes of generalized tonic spasms. These spasms are a hallmark of tetanus, resulting from the action of the tetanospasmin neurotoxin on the nervous system, disrupting inhibitory neurotransmission and leading to uncontrolled muscle contractions. It is noteworthy that the patient's past medical history was largely unremarkable. There was no indication of pre-existing conditions such as hypertension, diabetes mellitus, acute coronary syndrome, cardiac disease, liver disease, or gastric disease. This absence of significant comorbidities is an important consideration in the overall management of the patient, as it suggests that the acute illness was not complicated by any underlying chronic conditions. Upon arrival at our emergency department, the patient's presentation was concerning for a critical illness. He appeared acutely ill, was in severe distress, and exhibited agitation. A key finding was the compromise of his airway, characterized by significant trismus, which severely limited his ability to open his mouth. This trismus, or lockjaw, is a classic early symptom of tetanus, resulting from the involvement of the muscles of mastication. In addition to trismus, there was an increased amount of secretions in his airway, further compromising his respiratory status. The presence of laryngospasm risk was also noted which is a dangerous complication of tetanus. His respiratory status was further evaluated, revealing a respiratory rate of 34 breaths per minute, indicative of tachypnea. This elevated respiratory rate suggested significant respiratory distress and an attempt to compensate for inadequate oxygenation or ventilation. Despite the administration of high-flow oxygen via a non-rebreather mask at a rate of 15 liters per minute, his oxygen saturation as measured by pulse oximetry (SpO_2) was only 90%. This persistent hypoxemia, even with supplemental oxygen, pointed to a severe impairment of gas exchange within the lungs. Auscultation of the chest revealed vesicular breath sounds bilaterally, and initially, there were no adventitious sounds such as rhonchi or wheezes. This initial absence of abnormal lung sounds did not rule out underlying respiratory compromise but suggested that the primary issue at this stage was likely related to the effects of tetanus on respiratory muscle function rather than primary lung pathology. The cardiovascular examination revealed marked tachycardia, with a heart rate of 125 beats per minute and a regular rhythm. Tachycardia is a common finding in tetanus and can be attributed to autonomic nervous system dysfunction caused by the tetanospasmin toxin. Furthermore, the patient exhibited labile blood pressure, with an initial reading of 154/65 mmHg. However, it was noted that his blood pressure subsequently dropped despite initial fluid challenges, an important observation suggesting a developing hemodynamic instability. The patient's peripheries were cool to the touch, and his capillary refill time was less than 2 seconds. A capillary refill time of less than 2 seconds is generally considered normal, suggesting adequate peripheral perfusion at that specific moment. His temperature was recorded at 36.3°C. Neurological examination revealed that the patient was agitated and unable to follow commands. He only opened his eyes in response to painful stimuli, and based on these observations, his Glasgow Coma Scale (GCS) score was estimated to be E1 M1 V1. The GCS is a standardized tool used to assess a patient's level of consciousness, with scores ranging from 3 (deep coma) to 15 (fully alert). A GCS of E1 M1 V1 indicates severe impairment of consciousness. Further neurological assessment revealed generalized severe muscle hypertonicity and rigidity. This rigidity, affecting muscles throughout the body, is a hallmark of tetanus. Neck stiffness was also present, and the patient exhibited intermittent reflex spasms triggered by external stimuli. During the most severe spasms, he demonstrated opisthotonic posturing, characterized by severe hyperextension and arching of the back. Bowel sounds were present, indicating some level of gastrointestinal motility. A Foley catheter was in situ,

draining dark yellow urine, with an output of 800 cc over the preceding 10 hours. The color and volume of urine are important parameters for assessing renal function and hydration status. Initially, there was no peripheral edema or cyanosis. The healed puncture wound on his right thumb was noted. Initial laboratory investigations provided further insights into the patient's physiological derangement. Hematological analysis revealed leukocytosis, with a white blood cell (WBC) count of 18.5 x 10^3/L. Leukocytosis, an elevated WBC count, is often indicative of an inflammatory response or infection. The patient's hemoglobin level was 14.3 g/dL, and his platelet count was 250 x 10^3/L. Inflammatory markers were notably elevated, with a C-reactive protein (CRP) level of 210 mg/L. CRP is an acute-phase protein that rises in response to inflammation. Biochemical analysis revealed evidence of mild acute kidney injury (AKI), with a creatinine level of 1.8 mg/dL and a blood urea nitrogen (BUN) level of 95 mg/dL. The normal range for creatinine can vary slightly between laboratories, but this level was above the typical range, suggesting impaired renal function. Similarly, the elevated BUN level is another indicator of kidney dysfunction. The patient's albumin level was low at 2.9 g/dL. Albumin is a protein in the blood, and low levels can indicate various issues such as malnutrition, liver disease, or inflammation. The lactate level was elevated at 4.8 mmol/L. Elevated lactate is a marker of tissue hypoperfusion and anaerobic metabolism, often seen in critical illness and shock states. Electrolyte levels (sodium, potassium, chloride) were initially within normal limits. Liver function tests showed mildly elevated transaminases (ALT, AST) but normal bilirubin levels. Creatine kinase (CK) was elevated at 1500 U/L. Elevated CK can indicate muscle damage, potentially related to severe muscle spasms or rhabdomyolysis. Arterial blood gas (ABG) analysis, performed while the patient was receiving high-flow oxygen via a non-rebreather mask, revealed hypoxemia, with a partial pressure of oxygen (PaO_2) of 58 mmHg. The normal range for PaO_2 is typically 80-100 mmHg, so this result indicates significant oxygenation impairment. The initial ABG also showed respiratory alkalosis, with a pH of 7.48, a partial pressure of carbon dioxide ($PaCO_2$) of 30 mmHg, and a bicarbonate (HCO_3) level of 23 mmol/L. The respiratory alkalosis was consistent with the patient's tachypnea, as hyperventilation leads to a decrease in $PaCO_2$. However, the elevated lactate level suggested an underlying metabolic acidosis or tissue hypoperfusion, which can be masked by the respiratory alkalosis. Imaging studies included a chest X-ray (CXR) and an electrocardiogram (ECG). The initial CXR showed clear lung fields bilaterally but suboptimal inflation. The ECG revealed sinus tachycardia without any acute ischemic changes. Sinus tachycardia, an elevated heart rate originating from the sinoatrial node, is a common finding in critically ill patients. Based on the synthesis of the patient's clinical presentation, including the history of a puncture wound, the progressive muscle rigidity, trismus, reflex spasms, respiratory compromise, and autonomic features such as tachycardia and labile blood pressure, a diagnosis of severe generalized tetanus, classified as Ablett Grade III, was confirmed. The Ablett grading system is used to classify the severity of tetanus, with Grade III indicating severe tetanus. Concurrently, the patient also met the clinical and laboratory criteria for septic shock. This diagnosis was based on the presence of a suspected infection (the wound), persistent hypotension requiring vasopressor support (noradrenaline was initiated in the emergency department), elevated lactate levels, and evidence of organ dysfunction, including respiratory failure, acute kidney injury, and altered mental status. The patient's Sequential Organ Failure Assessment (SOFA) score was calculated to be 7. The SOFA score is a scoring system used to quantify the degree of organ dysfunction in critically ill patients, and a higher score is associated with a greater risk of mortality (Table 1).

The patient's management required a swift and multifaceted approach, initiated in the emergency department and continued in the intensive care unit (ICU). The immediate priorities upon the patient's arrival were stabilization and the institution of measures to address the life-threatening aspects of his condition. These initial steps were critical in providing the foundation for subsequent, more definitive treatments. The first crucial intervention was the immediate transfer of the patient to an ICU isolation room. This decision underscored the need for a controlled environment, minimizing external stimuli

such as light and noise, which are known to exacerbate the muscle spasms characteristic of tetanus. The rationale behind this environmental control is to mitigate any avoidable triggers that could precipitate or worsen the patient's symptoms. Given the patient's deteriorating respiratory status, severe trismus, and the imminent risk of laryngospasm, rapid sequence intubation (RSI) was performed either in the emergency department or immediately upon arrival in the ICU. Rapid sequence intubation is a critical procedure used to secure the airway in patients with conditions that pose a threat to their respiratory function. In this case, the combination of severe trismus, which hindered the patient's ability to open his mouth, and the high risk of laryngospasm, which could lead to complete airway obstruction, necessitated immediate intubation to establish a safe and patent airway. The procedure involved the administration of intravenous medications, specifically midazolam and vecuronium, to induce sedation and paralysis, respectively, facilitating the insertion of an endotracheal tube. A size 7.5 endotracheal tube was successfully secured, allowing for the initiation of mechanical ventilation. Simultaneously with airway management, hemodynamic support was initiated to counteract the septic shock. This involved the administration of intravenous fluids to address the fluid deficit and the use of vasopressors to maintain adequate blood pressure. In this particular case, noradrenaline infusion was required to support the patient's blood pressure. Following the initial stabilization, a series of interventions were implemented to address the various aspects of the patient's condition. Airway management remained a central focus of the patient's care. After the initial intubation with a 7.5 endotracheal tube, a surgical tracheostomy was performed on the third day of the patient's ICU stay. The decision to proceed with an early tracheostomy was based on the anticipation of a prolonged need for mechanical ventilation. Tracheostomy, an alternative airway management strategy, offers several advantages in patients requiring long-term ventilation, including improved patient comfort, reduced sedation needs, enhanced airway hygiene, and a decreased risk of laryngeal injury. Regular suctioning and monitoring of cuff pressure were integral components of the ongoing airway care to maintain the patency and integrity of the artificial airway and to prevent complications such as ventilator-associated pneumonia. To address the tetanus toxin itself, Human Tetanus Immunoglobulin (HTIG) was administered. Shortly after the patient's admission to the ICU, 5000 IU of HTIG was administered intramuscularly. Human Tetanus Immunoglobulin is a passive immunizing agent that provides immediate protection against tetanus toxin by neutralizing unbound toxin in the body. It is crucial to administer HTIG early in the course of the disease to prevent further toxin from binding to neural tissue. However, it is important to recognize that HTIG cannot reverse the effects of toxin that is already bound to the nervous system. Source control, aimed at eradicating the source of Clostridium tetani and preventing further toxin production, was another critical aspect of the treatment strategy. Intravenous metronidazole, an antibiotic effective against anaerobic bacteria like Clostridium tetani, was initiated at a dose of 500 mg every 6 hours. The typical duration of antibiotic therapy for tetanus is 7 to 10 days. In addition to antibiotic therapy, surgical debridement of the right thumb puncture wound was performed on the second day of the patient's admission. Even though the initial wound appeared relatively small and clean, debridement was undertaken to remove any potentially remaining necrotic tissue or foreign material that could harbor the bacteria. The management of the patient's severe muscle spasms and rigidity required a combination of pharmacological interventions. Continuous diazepam infusion was initiated and titrated aggressively to control these symptoms. Diazepam, a benzodiazepine, enhances GABAergic inhibition in the central nervous system, which helps to reduce muscle hyperactivity. The infusion rate was adjusted based on the patient's Richmond Agitation-Sedation Scale (RASS) score and a clinical assessment of his muscle tone. In this case, the patient required high doses of diazepam, with implied peak rates of up to 20 mg per hour and cumulative daily doses reaching 400 to 500 mg. Despite the high-dose diazepam, the patient continued to experience severe spasms, necessitating the addition of a continuous vecuronium infusion. Vecuronium is a neuromuscular blocking

agent (NMB) that induces paralysis by blocking the transmission of nerve impulses to muscles. The vecuronium infusion was carefully titrated using train-of-four (TOF) monitoring to achieve a target of 1 to 2 twitches. Train-of-four monitoring is a technique used to assess the degree of neuromuscular blockade. The use of neuromuscular blockade was limited to approximately 7 days, balancing the need for spasm control with the potential risks associated with prolonged paralysis. Continuous morphine infusion was administered for analgesia, providing pain relief during the period of deep sedation and neuromuscular blockade. Alongside pharmacological management, environmental control was maintained by ensuring a quiet and low-stimulus environment to minimize external triggers of spasms. Mechanical ventilation was a critical supportive measure for this patient, addressing the respiratory failure caused by tetanus-induced muscle rigidity and spasms. Initially, ventilation was provided using a volume-controlled mode. The ventilation strategy employed lung-protective settings, with a tidal volume of approximately 6 mL/kg of ideal body weight, a positive end-expiratory pressure (PEEP) of 5 to 8 cmH_2O, and the fraction of inspired oxygen (FiO_2) titrated to maintain an oxygen saturation (SpO_2) greater than 92%. The patient required mechanical ventilation for a total of 21 days. Weaning from mechanical ventilation was commenced around day 14, utilizing pressure support ventilation as a transitional mode. The patient was successfully liberated from mechanical ventilation on day 21. The management of septic shock was a parallel and crucial aspect of the patient's care, guided by established sepsis protocols. Initial fluid resuscitation was guided by dynamic assessments of fluid responsiveness. Noradrenaline infusion was required for 8 days to maintain a mean arterial pressure (MAP) greater than 65 mmHg. The dose of noradrenaline fluctuated significantly, reflecting the underlying autonomic instability characteristic of severe tetanus. In cases of refractory shock, hydrocortisone was administered intravenously. Serial lactate monitoring was performed, and the lactate levels normalized by day 4, indicating improving tissue perfusion and resolution of shock. Given the autonomic dysfunction that frequently accompanies severe tetanus, specific management strategies were implemented to address this aspect of the patient's condition. Close observation for fluctuations in heart rate and blood pressure was essential. Magnesium sulfate infusion was initiated on day 2, with a loading dose followed by a continuous infusion, titrated to maintain serum magnesium levels between 2.0 and 3.0 mmol/L. Magnesium sulfate has been used to help control cardiovascular instability and reduce the need for other sedatives or neuromuscular blocking agents. Intermittent labetalol infusion was used to manage episodes of paroxysmal hypertension. Throughout the patient's care, meticulous attention was paid to minimizing unnecessary stimuli. In addition to the treatments directly targeting tetanus and its complications, several supportive measures were implemented. Early enteral nutrition via a nasogastric tube (NGT) was initiated to meet the patient's high caloric needs, driven by the increased metabolic demands associated with muscle hyperactivity. A high caloric goal of 2400 kcal per day was established. Stress ulcer prophylaxis with a proton pump inhibitor and deep vein thrombosis (DVT) prophylaxis with low molecular weight heparin (LMWH) were administered to prevent common complications in critically ill patients. Regular turning, meticulous skin care, and passive physiotherapy were implemented to prevent pressure sores and contractures, which can occur during prolonged immobility. Fluid balance and renal function were closely monitored, and the acute kidney injury resolved by day 5. Despite the preventive measures, the patient developed a complication during his ICU stay. On day 9 of ventilation, he developed ventilator-associated pneumonia (VAP). The diagnosis of VAP was based on clinical signs, including new purulent secretions, fever, and increased oxygen requirements, as well as radiographic evidence of a new infiltrate on the chest X-ray. Cultures from the endotracheal aspirate grew Pseudomonas aeruginosa. Targeted antibiotic therapy with piperacillin-tazobactam, adjusted based on the culture sensitivities, was administered for 10 days, leading to the resolution of the VAP. As the patient's condition improved, weaning from mechanical ventilation and sedation was initiated. After approximately 14 days, the muscle

rigidity and the frequency of spasms had significantly decreased, allowing for the gradual weaning of neuromuscular blockade and then sedation. Ventilator weaning commenced using pressure support ventilation. The patient was successfully liberated from mechanical ventilation on ICU day 21. Following successful ventilator weaning and airway assessment, the tracheostomy tube was downsized and eventually decannulated on ICU day 28. The patient required ongoing physiotherapy for generalized weakness and rehabilitation to regain functional independence. Prior to discharge from the hospital, the patient received the first dose of the tetanus toxoid vaccine. Furthermore, arrangements were made for him to complete the primary immunization series and receive booster doses, as natural infection with tetanus does not confer immunity. The patient's clinical course involved a prolonged ICU stay and hospitalization. His total ICU length of stay was 30 days, and his total hospital length of stay was 45 days. At the time of discharge, the patient was transferred to a rehabilitation facility. He had achieved significant functional recovery but still exhibited residual muscle stiffness and fatigue, necessitating ongoing rehabilitation efforts (Table 2).

Table 1. Summary of patient's clinical findings on admission.

Parameter	Finding
Demographics	• **Age:** 41 years • **Gender:** Male • **Weight:** 60 kg • **Height:** 160 cm • **Occupation:** Assumed Farmer (based on injury mechanism/context) • **Residence:** Rural area (inferred from referral pattern)
Anamnesis (History)	• **Chief Complaints:** Acute shortness of breath, generalized body stiffness/rigidity • **History of Present Illness:** - Puncture wound (right thumb) from bamboo, 10 days prior - Onset of muscle stiffness 3 days prior to transfer - Progressive symptoms leading to admission at referring hospital - Acute worsening with severe dyspnea and generalized spasms (2 episodes) starting evening before transfer • **Past Medical History:** No history of Hypertension, Diabetes Mellitus, Acute Coronary Syndrome, Cardiac Disease, Liver Disease, or Gastric Disease • **Vaccination History:** No documented history of tetanus vaccination • **Allergies:** None known • **Last Meal/Intake:** Milk intake noted hours prior to ED arrival
Physical examination	• **General:** Acutely ill, severe distress, agitated • **Vital Signs:** - Respiratory Rate: 34 breaths/min - SpO_2: 90% on 15L Non-Rebreather Mask - Heart Rate: 125 beats/min, regular - Blood Pressure: 154/65 mmHg (initially, labile) - Temperature: 36.3 °C • **Airway:** Compromised, significant trismus (unable to open mouth > 1 finger breadth), increased secretions • **Breathing:** Tachypnea, use of accessory muscles, bilateral vesicular breath sounds, no rhonchi/wheezes initially • **Circulation:** Tachycardia, cool peripheries, Capillary Refill Time < 2 seconds • **Neurology:** - GCS: E1 M1 V1 (estimated due to distress/trismus) - Generalized severe muscle hypertonicity/rigidity - Neck stiffness - Intermittent reflex spasms triggered by stimuli - Opisthotonic posturing during severe spasms • **Abdomen:** Board-like rigidity (defans muscular), non-distended, bowel sounds present • **Extremities:** No peripheral edema or cyanosis initially. Healed puncture wound noted on right thumb.
Laboratory findings	• **Hematology:** - White Blood Cell (WBC): 18.5 x 10^3/L (Leukocytosis) - Hemoglobin: 14.3 g/dL - Platelets: 250 x 10^3/L • **Inflammatory Markers:** - C-Reactive Protein (CRP): 210 mg/L (Elevated) • **Biochemistry:** - Creatinine: 1.8 mg/dL (Elevated - AKI) - Blood Urea Nitrogen (BUN): 95 mg/dL (Elevated) - Albumin: 2.9 g/dL (Low) - Lactate: 4.8 mmol/L (Elevated) - Electrolytes (Na, K, Cl): Within normal limits - Liver Function Tests (ALT, AST, Bilirubin): Mildly elevated transaminases, normal bilirubin - Creatine Kinase (CK): 1500 U/L (Elevated - Rhabdomyolysis) • **Arterial Blood Gas (on NRB):** - pH: 7.48 (Alkalosis) - $PaCO_2$: 30 mmHg (Low) - PaO_2: 58 mmHg (Hypoxemia) - HCO_3: 23 mmol/L
Imaging findings	• **Chest X-Ray (CXR):** Clear lung fields bilaterally, suboptimal inflation initially. • **Electrocardiogram (ECG):** Sinus tachycardia, no acute ischemic changes.
Clinical diagnosis	• Severe Generalized Tetanus (Ablett Grade III) • Respiratory Failure secondary to tetanus (requiring mechanical ventilation) • Septic Shock (Suspected source: Wound infection/Tetanus-related processes; SOFA Score = 7)

Table 2. Summary of treatment procedures and follow-up.

Procedure / Aspect	Details
Initial stabilization (ED/ICU)	• Immediate transfer to ICU isolation room (quiet, dark environment). • Rapid Sequence Intubation (RSI) in ED/upon ICU arrival. • Initiation of hemodynamic support (fluids, vasopressors).
Airway management	• **Intubation:** RSI with Midazolam and Vecuronium; Size 7.5 Endotracheal Tube (ETT) secured. • **Tracheostomy:** Surgical tracheostomy performed on ICU Day 3 due to anticipated prolonged ventilation needs. • **Airway Care:** Regular suctioning, cuff pressure monitoring.
Toxin neutralization	• **HTIG:** Human Tetanus Immunoglobulin 5000 IU administered intramuscularly soon after admission.
Source control	• **Antibiotics (Tetanus):** Intravenous Metronidazole 500mg every 6 hours initiated (Duration typically 7-10 days). • **Wound Care:** Surgical debridement of the right thumb puncture wound performed on Day 2.
Sedation & spasm control	• **Benzodiazepines:** Continuous Diazepam infusion, titrated aggressively (peak rates implied up to 20 mg/hr; cumulative daily doses 400-500mg) based on RASS and spasm control. • **Neuromuscular Blockade (NMB):** Continuous Vecuronium infusion initiated due to severe spasms refractory to high-dose Diazepam; titrated to Train-of-Four (TOF) monitoring (target 1-2 twitches); duration approximately 7 days. • **Analgesia:** Continuous Morphine infusion administered during deep sedation/NMB. • **Environment:** Maintained quiet, low-stimulus environment.
Mechanical ventilation	• **Mode:** Volume-Controlled Ventilation initially. • **Strategy:** Lung-protective ventilation (Tidal Volume ~6 mL/kg IBW, PEEP 5-8 cmH_2O, FiO_2 titrated for SpO_2 >92%). • **Duration:** Total 21 days. • **Weaning:** Commenced around Day 14 via Pressure Support Ventilation; successfully liberated on Day 21.
Hemodynamic support (Septic Shock)	• **Fluids:** Initial resuscitation guided by dynamic assessments. • **Vasopressors:** Noradrenaline infusion required for 8 days to maintain MAP >65 mmHg (peak dose 0.5 mcg/kg/min). • **Adjunctive:**Hydrocortisone 50mg IV Q6H administered for refractory shock. • **Monitoring:** Continuous arterial blood pressure, central venous pressure (implied), serial lactate monitoring (normalized by Day 4).
Autonomic dysfunction management	• **Monitoring:** Close observation for fluctuations in heart rate and blood pressure. • **Magnesium Sulfate:** Infusion initiated on Day 2 (loading dose + continuous infusion), titrated to target serum levels (2.0-3.0 mmol/L). • **Antihypertensives:** Intermittent Labetalol infusion used for paroxysmal hypertension. • **Stimulus Control:** Minimized unnecessary stimuli during care.
Antibiotic therapy (Specific)	• **Tetanus:** Metronidazole 500mg IV Q6H. • **VAP:** Piperacillin-tazobactam initiated on Day 9 based on clinical signs and *Pseudomonas aeruginosa* growth in aspirate; duration 10 days.
Nutritional Support	• Early initiation of Enteral Nutrition via Nasogastric Tube (NGT). • High caloric goal (target 2400 kcal/day) established.
Prophylaxis	• **Stress Ulcer:** Proton Pump Inhibitor administered. • **Deep Vein Thrombosis (DVT):** Low Molecular Weight Heparin (LMWH) administered subcutaneously.
Complication management	• **Ventilator-Associated Pneumonia (VAP):** Diagnosed on Day 9; treated with targeted antibiotics based on culture results for 10 days.
Weaning & rehabilitation	• **Sedation Weaning:** Gradual reduction of Diazepam and Morphine after Day 7, guided by clinical assessment. • **Ventilator Weaning:** Started around Day 14, progressed through pressure support, liberation on Day 21. • **Tracheostomy Decannulation:** Performed on Day 28 after successful ventilator liberation and airway assessment. • **Physiotherapy:** Initiated early (passive ROM), progressed to active mobilization as tolerated.
Immunization	• First dose of Tetanus Toxoid vaccine administered prior to hospital discharge. • Plan established for completion of primary immunization series post-discharge.
Follow-up & outcome	• **ICU Length of Stay:** 30 days. • **Hospital Length of Stay:** 45 days. • **Discharge Disposition:** Transferred to a rehabilitation facility. • **Condition at Discharge:** Significant functional recovery but with residual muscle stiffness and fatigue requiring ongoing rehabilitation.

3. Discussion

The patient's initial presentation was characterized by a constellation of signs and symptoms that are pathognomonic for severe tetanus. These included trismus, generalized muscle rigidity, and reflex spasms, all following a puncture wound. The temporal relationship between the injury and the onset of these symptoms is crucial in establishing the diagnosis. In this case, the patient developed symptoms approximately ten days after sustaining a puncture wound from a piece of bamboo. This aligns with the typical incubation period for tetanus, which can range from a few days to several weeks, although it commonly falls within the range of 3 to 21 days. The variability in the incubation period is influenced by factors such as the dose of the toxin, the distance of the wound from the central nervous system, and the patient's immune status. Trismus, often referred to as "lockjaw," is one of the earliest and most characteristic features of tetanus. It results from the involvement of the muscles of mastication, particularly the masseter muscles, and reflects the generalized increase in muscle tone and spasms caused by the tetanus toxin. The patient's inability to open his mouth more than the width of one finger was a clear indication of the severity of his trismus. Generalized muscle rigidity is another hallmark of tetanus. In this patient, it manifested as a board-like rigidity of the abdomen and stiffness in other muscle groups. This rigidity is a consequence of the toxin's action on the inhibitory neurons in the spinal cord, leading to the disinhibition of motor neurons and subsequent muscle hyperactivity. The reflex spasms observed in this case are also a direct result of this disinhibition. These spasms are typically painful and can be triggered by even minor external stimuli, such as touch, noise, or light. The severity of the spasms can vary, and in severe cases, they can lead to complications such as respiratory compromise and rhabdomyolysis. The patient's presentation was classified as Ablett Grade III tetanus. The Ablett classification system is a clinical grading scale used to assess the severity of tetanus. It takes into account factors such as the extent of muscle involvement, the presence of respiratory complications, and the occurrence of autonomic dysfunction. Grade III tetanus is characterized by generalized tetanus with respiratory complications, indicating a severe form of the disease. The patient's respiratory distress, as evidenced by tachypnea and hypoxemia, along with the generalized muscle rigidity and spasms, supported this classification. It is important to emphasize that the diagnosis of tetanus is primarily clinical. While laboratory tests may be helpful in ruling out other conditions, there are no specific laboratory tests that can definitively confirm the diagnosis of tetanus. Blood cultures are typically negative, and the toxin itself is rarely detectable in blood or other bodily fluids. Wound cultures may grow Clostridium tetani, but this is not always the case, and the presence of the bacteria does not necessarily equate to a diagnosis of tetanus, as the bacteria can sometimes be found in wounds without causing disease. In this case, the diagnosis was based on the patient's history of a puncture wound, the characteristic clinical features, and the exclusion of other possible causes.[11-14]

The development of septic shock in this patient significantly complicated his clinical course and added another layer of complexity to his management. Septic shock is a life-threatening condition that results from a dysregulated host response to infection. It is characterized by persistent hypotension requiring vasopressors to maintain adequate perfusion despite adequate fluid resuscitation, along with evidence of organ dysfunction. In this patient, the septic shock likely stemmed from a combination of factors. While tetanus itself is a toxemia caused by the effects of the bacterial toxin, the initial wound can serve as a portal of entry for other bacteria, leading to a localized infection that can progress to sepsis. Although wound cultures were negative in this case, it does not entirely rule out the possibility of a localized infection contributing to the septic process. Furthermore, the prolonged ICU stay and the invasive procedures required for the management of severe tetanus increase the risk of nosocomial infections. Ventilator-associated pneumonia (VAP), which the patient developed later in his ICU stay, is a common complication in patients requiring prolonged mechanical ventilation and can itself be a source of sepsis. The presence of septic shock in a patient with severe tetanus has profound

implications for management. The hemodynamic instability associated with septic shock can exacerbate the autonomic dysfunction caused by tetanus, making blood pressure control particularly challenging. Both conditions can lead to end-organ damage, increasing the risk of complications such as acute kidney injury, respiratory failure, and cardiovascular dysfunction. The mortality rate in patients with severe tetanus is already significant, and the co-occurrence of septic shock further increases the risk of a fatal outcome. The diagnosis of septic shock in this patient was based on the clinical criteria defined by the Surviving Sepsis Campaign. These criteria include the presence of a suspected infection, persistent hypotension requiring vasopressor support, elevated lactate levels, and evidence of organ dysfunction. In this case, the patient had a suspected infection related to the wound, required noradrenaline to maintain his blood pressure, had an elevated lactate level, and exhibited organ dysfunction in the form of respiratory failure and acute kidney injury. The patient's SOFA score of 7 also supported the diagnosis of septic shock, as higher SOFA scores are indicative of more severe organ dysfunction and a worse prognosis.[15-17]

Immediate airway management is a cornerstone of tetanus treatment, particularly in severe cases like the one presented here. The patient's presentation with respiratory distress, severe trismus, and the risk of laryngospasm necessitated prompt intervention to secure his airway. The severe trismus associated with tetanus can significantly impair the patient's ability to maintain a patent airway. The rigidity of the muscles of mastication makes it difficult or impossible for the patient to open their mouth, hindering effective ventilation and increasing the risk of aspiration. Laryngospasm, a sudden and forceful contraction of the laryngeal muscles, is another potentially life-threatening complication of tetanus. It can be triggered by stimuli such as suctioning or even the presence of secretions in the airway. Laryngospasm can lead to complete airway obstruction and respiratory failure. In this patient, rapid sequence intubation (RSI) was performed to establish a secure airway. RSI is a standardized procedure that involves the simultaneous administration of a potent sedative and a neuromuscular blocking agent to facilitate endotracheal intubation. The sedative induces unconsciousness, while the neuromuscular blocking agent paralyzes the muscles, including the respiratory muscles, allowing for the insertion of an endotracheal tube into the trachea. This procedure is crucial in patients with tetanus to prevent aspiration, ensure adequate ventilation, and protect against laryngospasm. Following the initial intubation, the patient underwent a surgical tracheostomy on the third day of his ICU stay. Tracheostomy is a surgical procedure that creates an opening in the trachea, into which a tube is inserted to provide an alternative airway. The decision to perform an early tracheostomy in this patient was based on the anticipation of a prolonged need for mechanical ventilation. There are several advantages to tracheostomy in patients requiring prolonged ventilation. It can reduce the risk of laryngeal injury and subglottic stenosis, which are potential complications of prolonged endotracheal intubation. Tracheostomy can also improve patient comfort and facilitate airway suctioning, making it easier to manage secretions. In patients with tetanus, where prolonged ventilation is often necessary due to the duration of muscle spasms and the need for sedation, early tracheostomy can be a valuable intervention.[18-20]

4. Conclusion

The case report highlights the complexities involved in the management of severe tetanus complicated by septic shock. The successful treatment of this patient underscores the importance of a prompt, multidisciplinary, and aggressive critical care approach. Key to the patient's survival was the early recognition of tetanus and septic shock, followed by immediate airway management, aggressive control of muscle spasms and rigidity, hemodynamic support, and targeted antimicrobial therapy. The utilization of human tetanus immunoglobulin and metronidazole aimed at neutralizing the toxin and eradicating the source of infection, respectively, while continuous infusions of diazepam and vecuronium effectively managed the severe muscle hyperactivity. Furthermore, the case emphasizes the significance of meticulous

supportive care, including mechanical ventilation, nutritional support, VTE prophylaxis, and management of complications such as ventilator-associated pneumonia. The management of autonomic instability, a frequent and life-threatening complication of severe tetanus, required continuous monitoring and specific interventions to control fluctuations in heart rate and blood pressure. Despite the challenges posed by the severity of the patient's condition and the development of a nosocomial infection, the patient achieved significant recovery, highlighting that a comprehensive and intensive critical care approach can lead to a favorable outcome in such complex cases.

5. References

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