

Clostridium tetani bacteremia without classical tetanus: A lesson from Malaysia

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SUMMARY

Clostridium tetani infection classically presents as a toxin-mediated neuromuscular disease, while invasive bloodstream infection is exceptionally rare. In regions with low tetanus incidence, atypical presentations may delay recognition and definitive management. We report a 61-year-old Malay man with long standing schizophrenia who presented with a two-week history of progressive right lower limb pain and blackish discoloration following a fall. Clinical examination and imaging were consistent with gas gangrene. Despite early broad-spectrum antibiotics, definitive surgical source control was delayed due to challenges in obtaining informed consent. The patient underwent right above knee amputation (AKA) but subsequently deteriorated with septic shock, respiratory failure, and pulmonary embolism. Blood cultures obtained during clinical deterioration yielded *Clostridium tetani*. He succumbed despite active resuscitation. This case illustrates a rare and fulminant presentation of *Clostridium tetani* bacteremia arising from necrotizing soft tissue infection without classical neurological features of tetanus. To our knowledge, this is the first case of *Clostridium tetani* bacteremia reported in Malaysia. Early recognition, prompt antimicrobial therapy, assessment of tetanus immunization status, timely administration of tetanus immunoglobulin, and urgent surgical source control remain critical to improve outcomes in similar cases.

INTRODUCTION

Tetanus came from a Greek word tetanos that was derived from teinein meaning to stretch. It was first described in Egypt over 3000 years ago. Clinical disease is typically mediated by neurotoxins responsible for the characteristic features of muscular rigidity and spasms. It is rare cases nowadays due to the effectiveness of vaccination program but potentially fatal if it is missed. Despite this success, low disease prevalence may reduce clinical vigilance and delay recognition, particularly when presentations deviate from classical neuromuscular manifestations. Although *Clostridium tetani* is well recognized as a toxin-producing pathogen, invasive infection with documented bacteremia is exceedingly rare and sparsely reported in the literature. Such presentations may mimic other necrotizing soft tissue infections and obscure the underlying diagnosis. Hereby, we describe a fatal case of *Clostridium tetani* bacteremia arising from necrotizing soft tissue infection in a patient without classical features of tetanus. This report highlights the diagnostic and management challenges posed by atypical

disease presentations in low-incidence settings and underscores the importance of early suspicion, prompt treatment with antibiotics, immunoglobulins, vaccinations and timely surgical source control.

CASE PRESENTATION

We reported a 61 years-old Malay gentleman who lived alone, with a known case of schizophrenia for the past 20 years under primary health care follow up, on Zuclopenthixol 10 drops twice daily. He was brought to emergency department by ambulance complaining of right lower limb pain and blackish discoloration for two weeks prior to admission. He had a preceding history of a fall, after which he experienced persistent right hip pain and inability to ambulate. He denied any history of insect bites, open wounds, or fever.

On initial assessment, he was alert and oriented but appeared unkempt. His blood pressure was 167/85 mmHg, heart rate of 92 beats per minute, and capillary blood glucose of 10.4 mmol/L. Examination of the right lower limb revealed extensive blackish discoloration involving the entire leg, extending until above the knee, with foul smelling and palpable crepitus. Distal pulses were not palpable, except for the right femoral pulse. There was marked tenderness over the right hip.

Plain radiography of the right lower limb demonstrated extensive gas shadows extending up to the upper thigh. Pelvic and right hip radiographs showed no evidence of fracture. Initial laboratory investigations revealed leukocytosis with a total white cell count of $16.2 \times 10^9/L$ and elevated C-reactive protein of 281 mg/L. The rest of blood investigations were unremarkable except hyponatremia with sodium of 126mmol/L with mild elevation of lactate of 2.4 mmol/L without any metabolic acidosis, as shown by Table 1.

A diagnosis of gas gangrene of the right lower limb was made. The patient was immediately commenced on intravenous piperacillin-tazobactam 4.5g stat followed by 6-hourly dosing. Urgent right AKA was planned. However, surgical intervention was delayed due to difficulty of obtaining informed consent because of his underlying psychiatric illness and difficulty to reach his family members. Right AKA with partial closure of the distal stump was performed three days after his admission. Intraoperatively, there was necrosis and edema of the medial compartment

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Table I: Table of serial blood investigations

Laboratory test	Reference Range (Units)	20/4/24 (Admission)	27/4/24 Day 7 (deterioration)	1/5/24 Day 11
Hemoglobin	13.0 – 17.0 (g/dL)	16.1	14.4	13.4
White cell count	4.0 - 10.0 (10 ⁹ /L)	16.2	22.7	21.1
Platelet	150 – 410 (10 ⁹ /L)	187	296	220
Urea	3.2 – 8.2 (mmol/L)	5.2	5.3	14.6
Creatinine	62 – 115 (umol/L)	64	60	105
Sodium	136 – 145 (mmol/L)	126	129	161
Potassium	3.5 – 5.1 (mmol/L)	4.9	3.5	4.2
Chloride	98 – 107 (mmol/L)	91	98	121
Total protein	57 – 82 (g/L)	61	60	
Albumin	32 – 48 (g/L)	32	29	
Total Bilirubin	<21 (umol/L)	16.4	8.8	
Alanine Transferase (ALT)	10 – 49 (U/L)	118	139	
Alkaline Phosphate (ALP)	46 – 116 (U/L)	144	205	
Aspartate Transaminase (AST)	<34 (U/L)	113		
Calcium	2.18-2.60 (mmol/L)		2.23	1.88
Phosphate	0.78-1.65 (mmol/L)		1.53	1.26
Magnesium	0.53-1.11 (mmol/L)		0.85	0.93
C-Related Peptide, CRP	<10 (mg/L)	281	253	268
PT	9.4 - 11.0 (sec)	11.8	16.3	13.0
INR	0.87 - 1.03	1.14	1.61	1.27
aPTT	22.2 - 31.0 (sec)	32.1	39.1	32.4
pH	7.35 – 7.45 (mmHg)		7.26	7.62
partial oxygen	83 – 108 (mmHg)		136	140
partial carbon dioxide	35 – 48 (mmol/L)		34	25
bicarbonate	18 – 23 (mmol/L)		13.9	25.7
Base excess			-12	5.5
oxygen saturation	95 – 98 (%)		99	100
lactate	0.5 – 2.2 (mmol/L)	2.4	8.0	
D-dimer	<0/5 (mg/L)		2.31	

Cultures Summary

Type of cultures	Date	Result
Tissue	23/4/24	No Growth
Blood	27/4/24	Clostridium tetani

muscles with sloughy tissue, while the anterior and posterior compartments appeared viable. The patient was transferred back to the general orthopedic ward with a plan for secondary wound closure later. Intraoperative tissue cultures showed no microbial growth. The patient was successfully extubated postoperatively and demonstrated initial clinical improvement. However, the surgical wound remained unsatisfactory, with persistent slough, non-viable muscle tissue, macerated surrounding skin, and pale bluish discoloration despite regular wound care.

On post-operative day four, he was found gasping with a sudden drop in Glasgow Coma Scale (GCS) to E4V1M1 and hypoglycemia (capillary blood glucose 3.3 mmol/L). His blood pressure was 128/77 mmHg and tachycardia at 120 beats per minute. Oxygen saturation was 80% under room air and remained critically low (70–80%) despite on high flow mask oxygen therapy. He was intubated for respiratory distress and reduced consciousness, subsequently requiring inotropic support. A computed tomography pulmonary angiogram (CTPA) revealed pulmonary embolism involving the distal right pulmonary artery with extension into the right ascending and descending pulmonary arterial branches. Computed tomography (CT) brain showed no intracranial haemorrhage.

Wound inspection during his deterioration revealed macerated skin, foul odor, and necrotic tissue extending proximally toward the right hip. A plan for further surgical debridement and possible hip disarticulation was made. Unfortunately, the patient experienced cardiac arrest and passed away after 11 days of hospitalization despite active resuscitation. Blood culture obtained during his clinical deterioration on day seven of admission yielded *Clostridium tetani*.

DISCUSSION

Tetanus is a toxin-mediated, non-communicable, potentially fatal disease caused by *Clostridium tetani*, an obligate anaerobic, gram-positive, spore-forming bacillus that is ubiquitous in the environment. In Malaysia, tetanus remains uncommon, with an incidence of 0.09 per 100,000 population according to Health Facts 2024 published by the Ministry of Health Malaysia.¹ While this reflects successful national immunization efforts, the rarity of the disease may contribute to reduced clinical familiarity and delayed recognition, particularly in atypical presentations. Despite advances in critical care, generalized tetanus continues to be associated with a mortality rate of 10–20%.

Table I: Table of serial blood investigations

Case number	Author, Country	Year	Age, Gender	Clinical presentation	Investigation	Source	Treatment	Outcome
1	Rabih Riad Hallit et al, USA	2012	87 years old, Female	Generalized weakness and diarrhea for 3 days	Blood culture: <i>C. tetani</i> , colonoscopy : moderately severe colitis, diverticulosis, internal haemorrhoids	Gastrointestinal	Metronidazole, booster dose of the tetanus and diphtheria (Td) vaccine	Alive
2	Chih-Cheng Lai et al, Taiwan	2018	73 years old, Male	fever, chills, vomiting and general weakness for 1 day, 4 days after TAE for HCC	Blood culture: <i>C. tetani</i>	Gastrointestinal	Metronidazole	Alive
3	Kazadi D et al, USA	2022	86 years old, Male	Found on ground after unwitnessed fall with history of left leg pain prior	Blood culture: non perferingens <i>C. spp</i> , sacral ulcer cultures: <i>Clostridium inoum</i> and <i>Clostridium sporogenes</i>	Cutaneous (blistering ulceration left leg and sacral ulcer)	IV Piperacillin-Tazobactam, IV Vancomycin, debridement of sacral and left lower extremities ulcer	Alive
4	Our patient, Malaysia	2024	61 years old, Male	Right lower limb pain for 2 weeks	Blood culture: <i>C. tetani</i>	Cutaneous	IV Piperacillin-Tazobactam, right AKA	Deceased

The clinical manifestations of tetanus are primarily mediated by tetanospasmin, a potent neurotoxin that inhibits the release of inhibitory neurotransmitters, resulting in sustained muscle rigidity and painful spasms as shown in Figure 1. Classical features include trismus, neck stiffness, and generalized muscle rigidity, which may progress to respiratory failure and autonomic dysfunction. However, not all *Clostridium tetani* infections present with the characteristic neurological syndrome, creating diagnostic challenges and potentially delaying appropriate prophylactic measures.

As mentioned, *Clostridium tetani* is classically associated with toxin-mediated neuromuscular disease however, invasive bloodstream infection can occur. A limited number of cases of *Clostridium tetani* bacteremia have been reported in the literature, as summarized by Boualam et al.² To our knowledge, this case represents the second reported case of confirmed *Clostridium tetani* bacteremia arising from a cutaneous source following the report by Kazadi et al.³, and the first documented case from Malaysia. The present case highlights the capacity of *Clostridium tetani* to cause severe invasive disease with a fulminant clinical course even in the absence of classical neurological manifestations.

The diagnosis of *Clostridium tetani* bacteremia was established through blood culture. A paired blood culture set became positive after 46 hours of incubation, with growth detected in the anaerobic bottle. Identification of the isolate was identified by MALDI-TOF mass spectrometry and was verified by microbiologist. Antimicrobial susceptibility testing

was not done, as there are currently no guideline recommendations for routine susceptibility testing of *Clostridium tetani*. The isolation of *Clostridium tetani* from blood culture in the context of cutaneous and soft tissue source strongly supports its pathogenic role and against specimen contamination.

At present, there is also no definitive consensus guideline for the management of *Clostridium tetani* bloodstream infection. Consequently, treatment strategies are largely guided by expert opinion and source control principles. Review of previously reported cases as shown in Table II demonstrates that empiric antimicrobial therapy was directed according to the suspected source of infection. Intravenous metronidazole was administered in cases with presumed gastrointestinal sources, whereas piperacillin-tazobactam was used in cases involving skin and soft tissue infection, including our patient. Surgical debridement remains a cornerstone of management, as it removes necrotic tissue, reduces bacterial burden, eradicates spores, and limits ongoing toxin production. Delayed source control in our patient may have contributed to persistent infection and clinical deterioration.

An additional consideration in this case was the presence of pulmonary embolism, which may have contributed to the patient's worsening clinical condition and eventual death. Although severe soft tissue infection and septic complications likely represented the primary drivers of clinical decline, pulmonary embolism may have further compromised cardiopulmonary reserve and exacerbated tissue hypoxia.

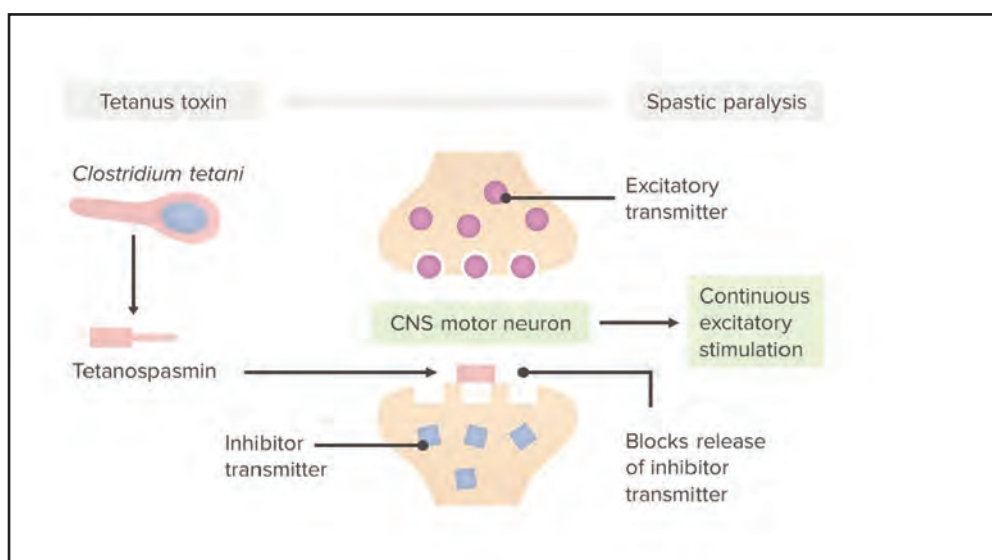


Fig. 1: Schematic illustration of pathophysiology in tetanus induced spastic paralysis: in an anaerobic environment (e.g. during active inflammation from the contaminated tissue) *C. tetani* spores germinate and produce the tetanospasmin toxin. Tetanospasmin binds to the presynaptic neuron, eventually allowing the light chain part of the protein to reach the spinal cord. The toxin specifically enters the central inhibitory neurons, prohibiting the release of GABA- and glycine containing vesicles from the cell membrane. This results in a loss of inhibition and subsequently continuous excitatory stimulation in motor neurons and the autonomic nervous system, causing uncontrolled motoric contractions⁴

Given the multifactorial nature of the patient's deterioration, it is difficult to determine the relative contribution of each process however, pulmonary embolism should be recognized as a potentially important coexisting factor influencing the fatal outcome.

Several important clinical challenges were illustrated by this case. First, delayed presentation to healthcare services, likely influenced by underlying psychosocial factors, resulted in extensive soft tissue involvement at the time of diagnosis. Second, definitive surgical source control was delayed because the patient lacked the capacity to provide informed consent, creating ethical and legal complexities that hindered timely intervention. Third, the atypical presentation, which was more consistent with an invasive soft tissue infection than classical tetanus, contributed to the omission of early tetanus vaccination and tetanus immunoglobulin administration. Collectively, these factors likely contributed to disease progression and limited opportunities for effective source control.

This case highlights the importance of assessing tetanus immunization status in all patients with tetanus prone wounds requiring post-exposure prophylaxis. Among previously reported cases in Table II, only one patient received a tetanus booster vaccination, while the remainder did not receive either a tetanus vaccine or human tetanus immunoglobulin. Current national recommendations advise administration of a tetanus vaccine and human tetanus immunoglobulin when vaccination history is uncertain or when wounds are considered high risk.⁵ Unfortunately, our patient's immunization status could not be verified, and neither a tetanus vaccine nor tetanus immunoglobulin was administered during hospitalization. From a broader public

health perspective, this case reinforces the importance of maintaining adequate tetanus immunity throughout adulthood. Routine tetanus booster vaccination is generally recommended every 10 years as per the National Vaccine-Preventable Diseases Recommendation for Older Adults published in 2025.⁵ Although widespread childhood immunization has markedly reduced the incidence of tetanus, waning immunity remains a concern in adults. Greater awareness among healthcare professionals and the public regarding routine booster vaccination and appropriate post-exposure prophylaxis may help prevent both classical tetanus and invasive *Clostridium tetani* infections.

CONCLUSION

This case demonstrates that *Clostridium tetani* can present as a fulminant invasive bloodstream infection without classical neurological manifestations of tetanus. Failure to recognize atypical presentations may result in delayed prophylaxis, postponed surgical intervention, and missed opportunities for escalation of care. Clinicians should maintain a high index of suspicion for tetanus related infection in patients presenting with necrotizing soft tissue infection of unclear aetiology, even in the absence of trismus or muscle spasms. Early recognition, prompt antimicrobial therapy, timely surgical source control, and appropriate administration of tetanus vaccine and immunoglobulin remain essential components of management and may improve outcomes in similar cases.

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CONFLICT OF INTEREST STATEMENT

The authors declare that there are no financial, personal, or professional relationships that could have influenced the work reported in this case report.

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