

A Rare Case of *Solobacterium moorei* Bacteraemia Secondary to Necrotising Fasciitis in a Patient with Chronic Elephantiasis.

Mohd Shah NA^{a,b}, Mohamed NN^{a,b}, Awad SN^{a,b}, Noorman MF^{b,c}

^aMicrobiology and Parasitology Unit, Department of Clinical Diagnostic Laboratories, Hospital Al-Sultan Abdullah (HASA), Universiti Teknologi MARA, Selangor

^bFaculty of Medicine, Universiti Teknologi MARA, Selangor

^cDepartment of Orthopaedic & Traumatology, Hospital Al-Sultan Abdullah (HASA), Universiti Teknologi MARA, Selangor

ABSTRACT

Keywords

Solobacterium moorei, bacteraemia, necrotising fasciitis, immunocompromised

Corresponding Author

Dr Nurul Azira Mohd Shah
Microbiology and Parasitology Unit,
Department of Clinical Diagnostic
Laboratories, Hospital Al-Sultan Abdullah
(HASA), Universiti Teknologi MARA,
Selangor
E-mail: nurul890@uitm.edu.my

Received: 16th May 2025; Accepted: 25th
September 2026

Doi:
<https://doi.org/10.31436/10.31436/imjm.v25i04/2964>

Solobacterium moorei is an anaerobic Gram-positive bacillus, part of the normal oral and intestinal microbiota, and is infrequently implicated in systemic infections. We report a rare case of *S. moorei* bacteraemia in a 64-year-old man with chronic right lower limb elephantiasis and multiple metabolic comorbidities. The patient presented with necrotising fasciitis of the right foot, necessitating urgent surgical intervention. Blood culture yielded *S. moorei*, while other significant cultures demonstrated mixed growth of aerobic and anaerobic organisms. The patient responded well to intravenous antibiotic therapy and was discharged in stable condition. This case underscores the potential pathogenicity of *S. moorei* in immunocompromised individuals and highlights the importance of considering rare anaerobic organisms in severe soft tissue infections.

INTRODUCTION

Solobacterium moorei is a non-spore-forming, gram-positive and an obligate anaerobic bacillus. Although part of the normal flora of the oral cavity and gut, it is rarely isolated in clinical settings and has been historically linked to halitosis.¹ Recent evidence has implicated *S. moorei* in a variety of opportunistic and invasive infections, including bacteremia, liver abscesses, oral infection including skin and soft tissue infections particularly in immunocompromised individuals.¹⁻⁴

The identification of *S. moorei* in blood cultures remains rare, and it is often underreported due to limitations in anaerobic culture methods and challenges with conventional identification systems. Newer diagnostic technologies such as MALDI-TOF MS and 16S rRNA gene sequencing have enhanced recognition of this organism in clinical practice.³

Necrotising fasciitis is a rapidly progressing, life-threatening soft tissue infection that is frequently polymicrobial and commonly affects individuals with predisposing factors such as diabetes, obesity, or chronic lymphoedema. The association between *S. moorei* and necrotising fasciitis has not been well established, and there are only a few published reports describing its presence in systemic infections of this nature.⁴

We present a case of *S. moorei* bacteremia secondary to necrotising fasciitis in a patient with chronic right leg elephantiasis and multiple metabolic comorbidities. This case adds to the growing recognition of *S. moorei* as a potentially invasive pathogen in vulnerable hosts.

CASE REPORT

A 64-year-old male presented to the emergency department with a 2-week history of right foot pain and swelling. The condition acutely worsened on the day of presentation with new-onset fever and foul-smelling purulent discharge from the plantar aspect of the right foot. He was referred by a general practitioner who had noted signs suggestive of severe infection during a routine wound dressing visit after a progressive, painful swelling of the plantar aspect of the foot following a puncture wound which subsequently ruptured with purulent discharge.

He had multiple significant medical histories of Type 2 diabetes mellitus, hypertension, chronic kidney disease (CKD Stage 3), dyslipidaemia, ischemic heart disease, and obstructive sleep apnoea (OSA). He also had chronic dermatologic conditions, including bilateral stasis eczema, tinea pedis, and lymphostasis verrucosa cutis or elephantiasis nostras verrucosa (ENV), with a history of leg reduction surgery in 2011.

Clinical examination revealed that he was obese with a BMI of 52.6 kg/m². On admission, he was febrile with temperature of 38.0°C and the vital signs were stable. There were chronic skin changes consistent with elephantiasis, which were thickened and cracked skin over the calf, sloughy and swelling of the dorsum and sole of the right lower limb.

Physical examination of the right lower limb revealed massive, brawny, non-pitting oedema extending from the mid-thigh to the toes, with complete obliteration of the malleoli and normal anatomical contours. These findings are diagnostic of International Society of Lymphology (ISL) Stage III lymphoedema (lymphostatic elephantiasis). The overlying skin exhibited pathognomonic features including severe pachydermia, diffuse hyperkeratosis, and verrucous papillomatosis prominent over the distal leg and dorsum of the foot, alongside a positive Stemmer's sign. Furthermore, deep transverse skin folds

at the ankle and interdigital maceration with superficial fissures were present. Wet gangrene of the right fifth toe with surrounding oedema and slough was evident. The dorsalis pedis and posterior tibial pulses were not appreciable

An X-ray of the right foot showed gas shadows over the fourth and fifth toes, suggestive of soft tissue gas-forming infection. Plain radiographs of the right lower extremity demonstrated features classic for severe chronic lymphostasis. There was massive expansion of the soft tissue compartment exhibiting a prominent reticular 'honeycomb' pattern, consistent with extensive fibrous septal thickening and oedema. The cutaneous silhouette was markedly thickened and lobulated. Superimposed on these changes, tracking gas dissecting along the deep fascial planes of the forefoot was observed, highly suspicious for an acute necrotising infection. No MRI or ultrasound was performed for lymphatic assessment in this acute setting.

He was immediately started on empirical intravenous piperacillin-tazobactam plus clindamycin, and underwent emergency debridement and Ray's amputation of the fourth and fifth toes. Blood culture was taken prior to antibiotic initiation. Intraoperative samples from bone, tissue, and pus were sent for culture and sensitivity.

The patient presented with a baseline haemoglobin of 11.3 g/dL. The postoperative drop to 9.0 g/dL was primarily attributed to surgical blood loss following extensive wound debridement and Ray's amputation. The team managed subsequent decreases in haemoglobin with appropriate blood product transfusions (fresh frozen plasma and packed cells). On Day 13, haemoglobin levels decreased to 6.9 g/dL; notably, this occurred in the absence of active surgical bleeding or clinical signs of sepsis. Given the clinical stability, occult gastrointestinal blood loss was suspected; consequently, the patient has been scheduled for outpatient endoscopic investigation to determine the definitive aetiology.

Blood culture yielded growth after 24 hours of incubation in an anaerobic bottle. Direct Gram staining from the anaerobic bottle revealed gram-positive bacilli. Subcultures were performed on MacConkey agar, and chocolate agar; however, no growth was observed after 48 hours under standard incubation conditions. Pure growth of tiny grey colonies was only seen on blood agar incubated under strict anaerobic conditions anaerobically after 48 hours (see figure 1). These colonies were sensitive to metronidazole. Identification was initially done by VITEK®system which was identified as 99% *Terrisporobacter glycolicus*, However, in view of the microbiology characteristics discrepancy, further identification was done via MALDI-TOF revealed *Solobacterium moorei*. Antibiotic susceptibility was evaluated by the E-test method in accordance with CLSI guidelines demonstrated sensitivity to ceftriaxone (MIC: 0.125 µg/mL), penicillin (MIC: 0.016 µg/mL), and piperacillin-tazobactam (MIC: 2 µg/mL).

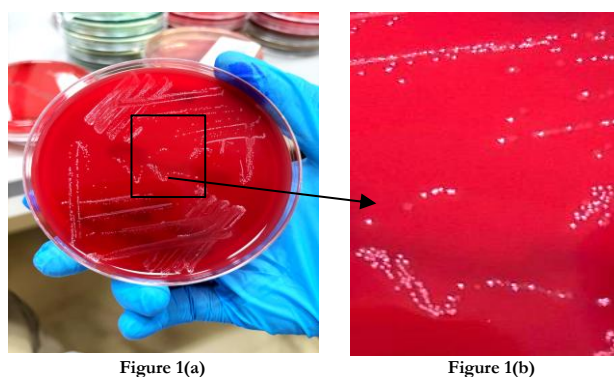


Figure 1: (a) Anaerobic blood agar plate showing pure growth of tiny grey colonies of *Solobacterium moorei* after 48 hours of incubation. (b) Higher magnification of the colonies.

There was no growth detected from the aerobic blood culture bottle. Mixed growth of more than three types of organisms which include three types of gram positive cocci and two types of gram negative bacilli were isolated from pus swabs, bone, and tissue samples.

Throughout the admission, C-reactive protein (CRP) levels demonstrated a steady downward trajectory, falling from 233.2 mg/L at presentation to 203.0 mg/L on Day 2, 124.5 mg/L on Day 4, 55.1 mg/L on Day 6,

and 20.1 mg/L on Day 11, eventually normalizing to 11.4 mg/L at discharge on Day 13. Total White Cells (TWC) trends normalized (Admission: $17.1 \times 10^9/L$ to $8.7 \times 10^9/L$ at discharge).

Empirical antibiotic therapy was initiated with intravenous piperacillin-tazobactam (4.5 g QID) and clindamycin (600 mg TDS), with clindamycin subsequently discontinued after a 10-day course. Following an Infectious Disease consultation, intravenous piperacillin-tazobactam was continued to complete a total 14-day course. Surveillance blood cultures were deemed unnecessary by the Infectious Disease team in light of the patient's rapid clinical improvement and significant inflammatory marker clearance. Throughout the hospital stay, the patient maintained haemodynamic stability, with the exception of an isolated febrile episode ($38.0^\circ C$) on admission that resolved within 24 hours of initiating targeted antimicrobial therapy. Leukocytosis showed marked improvement, decreasing from $17.1 \times 10^9/L$ at presentation to $8.7 \times 10^9/L$ at discharge, while the surgical site consistently demonstrated healthy granulation tissue.

Following that, antibiotics were continued for 15 days, and the infection resolved without further complications. Postoperative recovery was uneventful. By day 15 post surgery, the patient was afebrile, hemodynamically stable, and his surgical site demonstrated healthy granulation tissue with no residual pus. The vital signs remained stable throughout the admission. Inflammatory markers and haematological trends showed significant improvement during the course of hospitalization: the C-reactive protein (CRP) level decreased from 233.2 mg/L on admission to 11.4 mg/L at discharge, while the total white cell count (TWC) normalized from $17.1 \times 10^9/L$ to $8.7 \times 10^9/L$. He was mobilizing with a wheelchair and was deemed fit for discharge

DISCUSSION

Although traditionally associated with oral microbiota, *Solobacterium moorei* has increasingly been reported in invasive infections, including bacteraemia (Table I).² In a recent review of clinical cases, *S. moorei* was isolated from blood cultures, abscesses, and surgical wounds in both immunocompromised and critically ill patients.¹⁻⁴ However, its involvement in necrotising fasciitis remains exceedingly rare and poorly documented in the literature.

Table I: Summary of Previously Reported *S. moorei* Infection Cases²

Study Focus	Reported Cases	Key Demographics	Primary Infection Source
Retrospective Analysis	~27	Immunocompromised and critical care patients	Bacteraemia, Abscesses, Surgical Wounds

This case highlights the potential for *S. moorei* to contribute to polymicrobial infections in high-risk patients with chronic limb disease, poor skin integrity, and metabolic diseases. Isolation of this organism in blood cultures indicates haematogenous spread, likely originating from infected necrotic foot tissue. Chronic tinea pedis, elephantiasis (ENV), and diabetes further compromised the skin barrier, predisposing the patient to rapid progression of soft tissue infection. In addition, chronic dermatological breaches, specifically deep transverse skin folds at the ankle and interdigital maceration with superficial fissures combined with local lymphostasis in this patient, served as the primary predisposing portal of entry facilitated the translocation of this oral or intestinal commensal into the systemic circulation.⁵ Consequently, these factors facilitated the subsequent development of the necrotising fasciitis. Elephantiasis is a rare disease which leads to chronic lymphoedema in these patients. It has been documented to increase susceptibility to cellulitis or recurrent skin infections significantly.⁶

Identification was initially attempted using the VITEK® 2 system, which misidentified the isolate as *Terrisporobacter glycolicus* (99% probability). However, in view of the microbiological characteristics, which were

non-spore-forming Gram-positive bacilli, further identification was performed via matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS), which definitively identified the isolate as *S. moorei*. While 16S rRNA gene sequencing was not performed in this case, the MALDI-TOF MS identification was consistent with the clinical presentation and morphological findings. Misidentification of *S. moorei* as *T. glycolicus* (formerly known as *Clostridium glycolicum*) occurred because *S. moorei* was not present in the VITEK® 2 database, highlighting the limitations of automated systems and the need for advanced techniques like MALDI-TOF MS. Thus, *S. moorei* infection is likely underreported due to previous identification limitations before the widespread use of MALDI-TOF. As a fastidious obligate anaerobe, *S. moorei* may present with a metabolic profile that overlaps sufficiently with *T. glycolicus* in the limited biochemical tests performed by the VITEK® 2 system. A study has documented that commercially available systems such as the VITEK® 2 system have failed to identify *S. moorei*.⁵

Anaerobic bacteria are often underdiagnosed in necrotizing soft tissue infections because of the inherent limitations of culture-based methods, particularly in polymicrobial infections.⁷ *S. moorei* may be overlooked in mixed cultures if colony isolation on primary plates is suboptimal. The isolation of *S. moorei* as a pure isolate from the anaerobic blood culture, distinct from the polymicrobial wound environment, strongly supports its role as the causative agent of the patient's bacteraemia.^{1,2} Diagnostic confirmation proved challenging, as initial testing via the VITEK®2 system misidentified the organism, a known limitation documented in previous studies.⁷ Consequently, reliance on advanced diagnostic techniques like MALDI-TOF MS was essential to identify *S. moorei* and guide targeted therapy.³

This highlights the value of advanced diagnostic techniques for the accurate detection of fastidious anaerobes, which can ultimately form targeted antimicrobial therapy.³

Despite his high-risk clinical profile, the patient responded favourably to prompt surgical debridement, culture-guided antimicrobial therapy, and supportive care. This outcome underscores the imperative for early surgical intervention and broad-spectrum empirical coverage in severe necrotizing soft tissue infections.

CONCLUSION

This report emphasizes the emerging clinical significance of *Solobacterium moorei* as a potential opportunistic pathogen in necrotising soft tissue infections. Clinicians should be aware of its ability to cause systemic infections, particularly in immunocompromised individuals or those with compromised skin barriers. Appropriate anaerobic cultures and multidisciplinary care remain essential for optimizing outcomes in complex infections such as necrotising fasciitis.

ACKNOWLEDGMENTS

The authors would like to thank the multidisciplinary care team involved in the management of this patient.

CONFLICT OF INTEREST

The authors declare no conflict of interest related to this case report. All authors have read and approved the final manuscript.

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