Infection and Drug Resistance

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

Necrotizing Myositis Caused by *Enterococcus faecium* with Concurrent *Klebsiella pneumoniae Bacteremia*: A Rare Case Report

Xilei Niu[1,*], Hairu Ji[2,*], Shuai Wang[1,*], Guoyu Wang[3], Yu Wang[1], Yanchao Liu[4], Lingwei Kong[1]

[1]Department of Orthopaedics, The Affiliated Hospital of Chengde Medical College, Chengde, Hebei, People's Republic of China; [2]Pathology Teaching and Research Section, Chengde Medical College, Chengde, Hebei, People's Republic of China; [3]Department of General Practice, The Affiliated Hospital of Chengde Medical College, Chengde, Hebei, People's Republic of China; [4]Department of Clinical Laboratory, The Affiliated Hospital of Chengde Medical College, Chengde, Hebei, People's Republic of China

*These authors contributed equally to this work

Correspondence: Lingwei Kong; Guoyu Wang, Email konglingwei0408@126.com; wgy031213@163.com

Abstract: We present a rare case of necrotizing myositis predominantly caused by *Enterococcus faecium* in a 68-year-old healthy man, with concurrent *Klebsiella pneumoniae bacteremia*. The patient rapidly progressed from diarrhea and fever to septic shock, multiple organ dysfunction, and compartment syndrome of the left leg. Blood cultures grew *K. pneumoniae*, while deep tissue cultures identified *E. faecium* as the main pathogen in necrotic muscle. Emergent fasciotomy, serial debridements, and targeted antibiotics (meropenem and linezolid/vancomycin) led to full recovery. This first reported case highlights the underappreciated virulence of *E. faecium* in soft tissue infections and underscores the need for prompt imaging, surgical intervention, and comprehensive microbiological workup in severe infections. The case also emphasizes the importance of considering polymicrobial etiology and monitoring for antibiotic adverse effects.

Keywords: Necrotizing myositis, *Enterococcus faecium*, *Klebsiella pneumoniae*, *Bacteremia*

Introduction

Enterococcus faecium (E. faecium) is a significant opportunistic pathogen involved in both healthcare-associated and community-acquired infections, especially in immunocompromised individuals or those with underlying comorbidities.[1] While its role in bacteremia, endocarditis, and urinary tract infections is well-documented, its capacity to induce severe monomicrobial or polymicrobial necrotizing soft tissue infections (NSTIs) remains less recognized and is likely underestimated.[2] Emerging evidence suggests that *enterococci*, particularly *E. faecium*, may play a dominant role in tissue necrosis, owing to virulence factors such as gelatinase, cytolysin, and biofilm-forming capacity, which facilitate tissue invasion and immune evasion.[3] The intrinsic and acquired multidrug resistance of *E. faecium*, including the increasing prevalence of vancomycin-resistant strains (VRE), poses considerable therapeutic challenges and is correlated with adverse clinical outcomes.[4]

Conversely, *Klebsiella pneumoniae (K. pneumoniae)* is a prevalent Gram-negative bacterium known to cause a diverse array of illnesses, including pneumonia, bloodstream infections, and hepatic abscesses. The recent emergence of *hypervirulent strains (hvKP)* has been associated with severe invasive disease, even in immunocompetent hosts, often marked by metastatic spread to sites such as the eyes, central nervous system, and soft tissues.[5,6] These hypervirulent clones commonly display enhanced capsular polysaccharide production, robust siderophore systems, and additional virulence factors that collectively heighten their pathogenic capacity.[7]

Necrotizing myositis, an aggressive and life-threatening variant of NSTIs characterized by extensive skeletal muscle necrosis, is uncommon yet progresses with alarming rapidity. It is most frequently triggered by pyogenic bacteria like Group A Streptococcus (GAS) or *Staphylococcus aureus*.[8] The pathogenic mechanism often entails bacterial exotoxin

https://doi.org/10.2147/IDR.S591080
Received: 21 January 2026
Accepted: 18 April 2026
Published: 27 April 2026

release and an exaggerated host inflammatory cascade, resulting in substantial tissue devastation, systemic toxicity, and considerable mortality if not addressed with prompt and aggressive intervention.[9] Diagnostic ambiguity is common in early stages; however, localized pain that is disproportionate to physical signs serves as a critical clinical hallmark.[10]

The interplay between Gram-positive and Gram-negative pathogens in polymicrobial NSTIs is complex and can lead to synergistic tissue injury and amplified systemic inflammation.[11] Although in this case *K. pneumoniae* was isolated only from blood and *E. faecium* only from deep muscle tissue, the concurrent presence of both pathogens in the same patient—one driving systemic bacteremia and the other mediating local tissue destruction—constitutes a polymicrobial infectious process. Such anatomically distinct but temporally concurrent infections can exert synergistic effects on systemic inflammation and organ dysfunction, as supported by previous studies.[12,13] Presently, no cases of necrotizing myositis primarily caused by *E. faecium*, particularly in association with *K. pneumoniae bacteremia*, have been described in the medical literature. This case report seeks to fill this void by delineating a unique instance of lower extremity necrotizing myositis driven by *E. faecium*, complicated by co-existing *K. pneumoniae bacteremia*. We aim to augment clinical recognition of the potential involvement of atypical pathogens like *E. faecium* in serious soft tissue infections and to explore the attendant diagnostic and therapeutic complexities within polymicrobial frameworks.

Case Presentation

A 68-year-old man arrived at the emergency department reporting diarrhea and fever. His history indicated the onset of diarrhea, featuring multiple loose yellow stools, accompanied by abdominal discomfort and fever, roughly 8 hours before presentation, which he had initially ignored. Four hours later, he abruptly developed slurred speech and generalized malaise, leading him to seek emergency care. He had no known chronic illnesses and was previously in good health.

Upon admission, physical examination disclosed an alert patient with generally fair mental status and mild dysarthria. Inspection of the head, thorax, abdomen, and extremities revealed no obvious abnormalities. Vital signs were recorded as follows: temperature 39.0°C, heart rate 70 beats per minute, and blood pressure 107/65 mmHg. Initial laboratory evaluation demonstrated pronounced leukocytosis (white blood cell count 27.77×10^9/L) with neutrophilic predominance (91.8%), platelet count 153×10^9/L, and a markedly elevated procalcitonin level exceeding 100.00 ng/mL. Cardiac enzyme assays were strikingly abnormal: lactate dehydrogenase 646 U/L, creatine kinase (CK) >6400 U/L, and CK-MB activity 419 U/L. Troponin measurements indicated CK-MB mass >100.00 ng/mL and myoglobin >400.00 ng/mL. Serum lactate was raised at 5.86 mmol/L, indicative of tissue hypoperfusion. Hepatic and renal panels revealed: total bilirubin 30.4 μmol/L, indirect bilirubin 24.6 μmol/L, alanine aminotransferase 152.0 U/L, aspartate aminotransferase 538.0 U/L, gamma-glutamyl transferase 88.0 U/L; urea 7.63 mmol/L, creatinine 160 μmol/L, and bicarbonate 14.7 mmol/L.

A provisional diagnosis of sepsis complicated by acute kidney injury, hepatic dysfunction, and hyperlactatemia was made. The patient was immediately hospitalized and commenced on empirical intravenous meropenem (1g every 12 hours) for broad-spectrum antimicrobial protection, supplemented by supportive care including antidiarrheals, antipyretics, and hepatoprotective drugs. Two sets of blood cultures were procured from distinct sites before antibiotic initiation.

Within 1 hour of admission, the patient described sudden, intense pain in his left lower limb, mainly concentrated in the calf region. The overlying skin showed no erythema but was mildly warm on palpation. The pain escalated quickly, coupled with progressive swelling (Figure 1A). An urgent Magnetic Resonance Imaging (MRI) examination of the thigh and calf was conducted. MRI findings revealed swelling with heterogeneous increased signal intensity in the lateral muscles of the left thigh, as well as swelling and increased signal intensity within the tibialis posterior, soleus, and gastrocnemius muscles of the left calf, accompanied by obscuration of the intermuscular septa (Figure 1B).

The patient's status rapidly declined into septic shock, mandating the commencement of a continuous norepinephrine infusion for hemodynamic stabilization. Simultaneously, tension in the thigh and calf musculature increased steadily, more pronounced in the calf, accompanied by indicators of compromised circulation and sensation in the foot, fitting with compartment syndrome. An urgent surgical procedure was undertaken, involving exploratory incisions, fasciotomy, and debridement of the left thigh and calf. Intraoperative assessment revealed elevated compartment pressures in the calf. Following medial and lateral fasciotomies, the gastrocnemius, soleus, and tibialis posterior muscles displayed edema with patches of necrotic and non-viable tissue (Figure 1C). In the thigh, a lateral incision showed subcutaneous edema, and

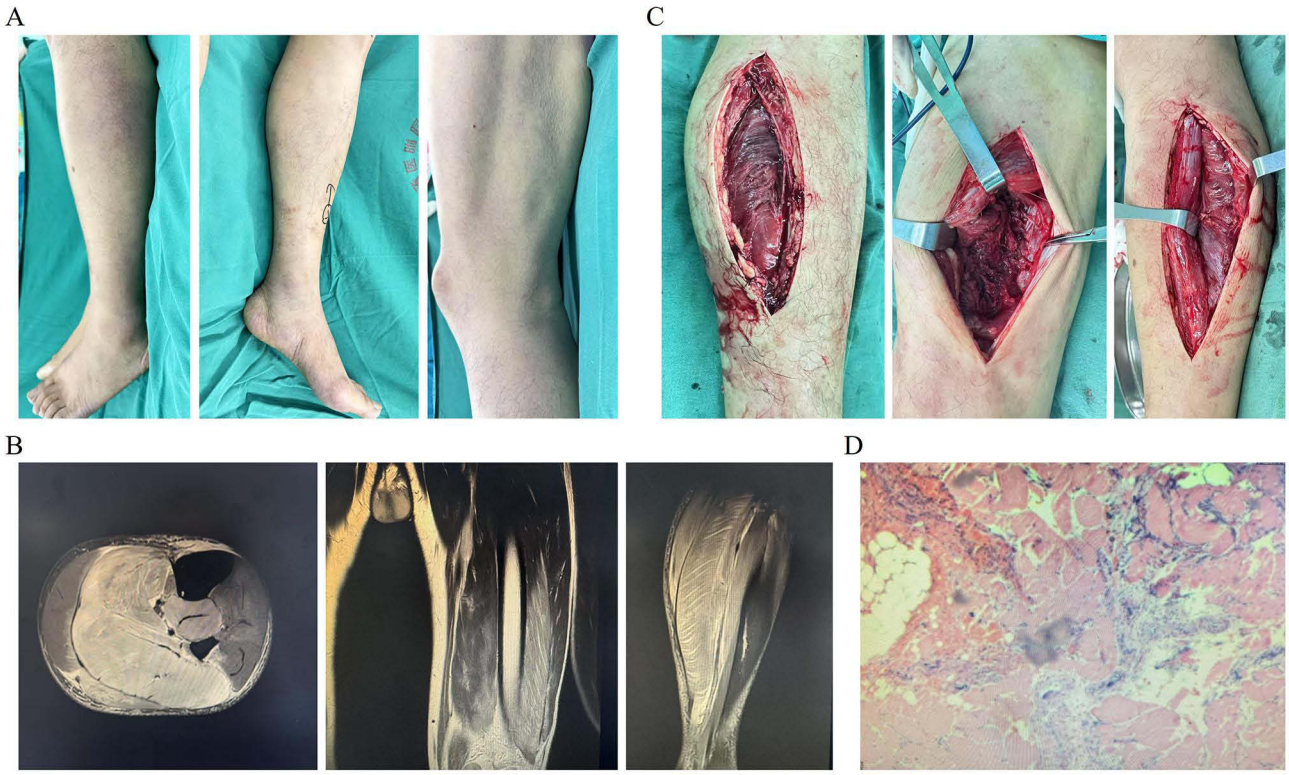

Figure 1 (**A**) The skin over the affected limb was intact and swollen; (**B**) MRI of the left lower extremity showed heterogeneous increased signal intensity and swelling in the lateral thigh muscles, as well as in the posterior tibialis, soleus, and gastrocnemius muscles of the calf. These findings were accompanied by obscuration of the intermuscular septa; (**C**) Intraoperatively, edema and partial tissue necrosis were observed in the gastrocnemius, soleus, and posterior tibialis muscles; (**D**) Hematoxylin and eosin (H&E) staining of the biopsied tissue revealed inflammatory cell infiltration.

deep fascial release uncovered significant edema of the vastus lateralis. During the surgery, purulent material and tissue specimens were collected for both microbiological and pathological evaluation. The pathological findings confirmed the diagnosis of necrotizing myositis (Figure 1D). After initial debridement, the patient was moved to the Intensive Care Unit (ICU) for continued intensive support. The patient necessitated mechanical ventilation, aggressive fluid resuscitation, blood product transfusion, and sustained norepinephrine infusion. He developed persistent anuria and escalating creatinine levels, prompting the initiation of renal replacement therapy.

Gram-negative bacilli were detected in the blood culture sample (Figure 2A). The specimen was inoculated onto blood agar and MacConkey agar, and after 18–24 hours of incubation at 36°C, the colonies exhibited a hyperviscous morphology (Figure 2B). The isolate was identified as *Klebsiella pneumoniae* by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS; bioMerieux, France) (Figure 2C). Antimicrobial susceptibility testing (AST) using the Vitek 2 Compact system with the GN-09 card (bioMerieux, France) revealed susceptibility to aminoglycosides (eg, gentamicin, tobramycin, and amikacin), β-lactams (eg, meropenem), fluoroquinolones (eg, levofloxacin), and tetracyclines (eg, tigecycline). Concurrently, Gram-positive cocci were observed in a direct Gram stain of the patient's pus specimen (Figure 2D). The specimen was inoculated onto blood agar, MacConkey agar, and anaerobic blood agar; following incubation at 36°C for 18–24 hours (with the anaerobic agar incubated in an anaerobic jar for at least 48 hours), the colonies displayed a buttery consistency (Figure 2E). This isolate was identified as *Enterococcus faecium* by MALDI-TOF-MS (bioMerieux, France) (Figure 2F), and AST performed with the GP-67 card (Vitek 2 Compact; bioMerieux, France) demonstrated susceptibility to aminoglycosides (high-level gentamicin and high-level streptomycin), fluoroquinolones (levofloxacin and ciprofloxacin), glycopeptides (vancomycin and teicoplanin), tetracyclines (tetracycline and tigecycline), and an oxazolidinone (linezolid). Guided by these antimicrobial susceptibility profiles, the antibiotic regimen was modified to linezolid in conjunction with meropenem.

https://doi.org/10.2147/IDR.S591080

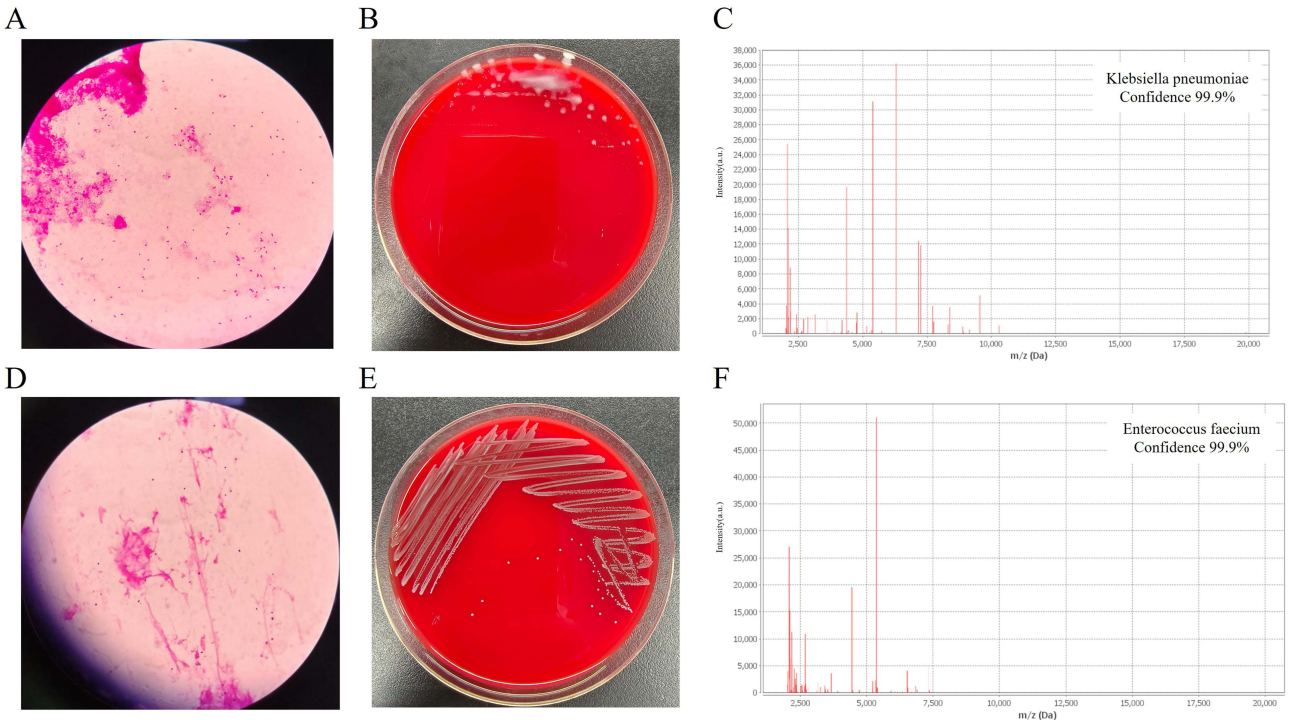

Figure 2 (**A**–**C**) *Klebsiella pneumoniae* isolated from blood culture. (**A**) Gram staining of the positive blood culture showing Gram-negative rods arranged singly and in pairs. (**B**) Colony morphology on blood agar after 18–24 h of incubation, demonstrating a hyperviscous appearance. (**C**) Identification of the isolate as *K. pneumoniae* with a confidence level of 99.9% using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; bioMérieux, France). (**D**–**F**) *Enterococcus faecium* isolated from intraoperative deep tissue culture. (**D**) Gram staining of the wound specimen revealing Gram-positive cocci in pairs and chains. (**E**) Colony morphology on blood agar showing a buttery consistency. (**F**) Identification of the isolate as *E. faecium* with a confidence level of 99.9% using MALDI-TOF MS (bioMérieux, France).

The patient's status slowly stabilized with directed antibiotics and several additional debridements, and renal function eventually recuperated. During the ensuing clinical trajectory, the patient manifested persistent high-grade fever (peaking at 39.1°C) and a widespread non-pruritic rash. Laboratory analyses showed a substantial drop in white blood cell and neutrophil counts. After multidisciplinary assessment, a diagnosis of severe drug-induced dermatitis with associated neutropenia was established. Linezolid was halted and substituted with vancomycin, leading to abatement of fever, rash improvement, and neutrophil count recovery. He was later discharged. At the three-month follow-up, there was no evidence of soft tissue infection recurrence, and renal function along with leukocyte/neutrophil counts were maintained within normal limits.

Discussion

We delineate a highly uncommon and serious case of necrotizing myositis principally instigated by *Enterococcus faecium*, accompanied by concurrent *Klebsiella pneumoniae bacteremia*. The swift clinical transition from non-specific gastroenteritis-like manifestations to septic shock, multi-organ dysfunction syndrome (MODS), and compartment syndrome within a narrow temporal window underscores the highly aggressive behavior of this polymicrobial infection. This case not only extends the acknowledged pathogenic range of *E. faecium* but also sheds light on pivotal elements in the diagnosis and treatment of severe soft tissue infections.

Although *E. faecium* is frequently perceived as a commensal with limited innate virulence, it can emerge as a potent pathogen, especially in vulnerable hosts or particular clinical settings. Virulence determinants such as gelatinase (which degrades extracellular matrix constituents), cytolysin (a pore-forming exotoxin), and assorted surface adhesins encourage tissue penetration, biofilm development, and circumvention of host immune mechanisms.[4] In this instance, the initial bout of acute gastroenteritis plausibly impaired intestinal mucosal barrier function, potentially permitting bacterial translocation of gut microbiota, including *E. faecium*, into the circulation.[14] Subsequent hematogenous spread to soft tissues, possibly colonizing a locus of minor strain or microtrauma, might have provided the foundation for initiating necrotizing myositis. The profoundly

elevated creatine kinase level (>6400 U/L) acted as a direct laboratory indicator of widespread muscle necrosis, aligning with surgical observations.

The co-existence of *K. pneumoniae bacteremia* probably contributed a synergistic influence, intensifying the systemic inflammatory reaction and hastening the descent into septic shock. *K. pneumoniae* harbors potent virulence attributes, including capsular polysaccharides that resist phagocytosis and lipopolysaccharides (LPS) that potently stimulate pro-inflammatory cytokines like TNF-α and IL-6, thus propelling the pathogenesis of septic shock and organ failure.[5,7] Although the hypervirulent nature of this specific isolate was not genetically verified, the clinical picture featuring rapid hematogenous dissemination and profound systemic illness is highly indicative. The polymicrobial interplay between *E. faecium* and *K. pneumoniae* may have cultivated a more damaging microenvironment. Published evidence indicates that co-infecting bacteria can act synergistically to worsen tissue injury via combined toxin excretion, metabolic collaboration, or alteration of host immune reactions, though the exact mechanisms for this specific pathogen combination demand deeper exploration.[11,15]

This case strongly reaffirms several vital diagnostic and management tenets. Firstly, identifying severe, disproportionate pain as a fundamental characteristic of necrotizing soft tissue infections is essential.[10] In our patient, the severe limb pain and subsequent neurological deficits were crucial warning signs that antedated visible skin alterations. Secondly, MRI was invaluable for early identification, precisely mapping the degree of muscle edema, necrosis, and fascial engagement, thus directly shaping the timing and magnitude of surgical action.[16] Thirdly, the fast development of compartment syndrome required immediate surgical release via fasciotomy. Expedient and thorough excision of all non-viable tissue constitutes a life-preserving mainstay in managing necrotizing myositis.[9,17] Lastly, this case accentuates the critical need to procure deep tissue samples during surgery for exhaustive microbiological culture and sensitivity analysis. Dependence on blood cultures alone would have detected *K. pneumoniae* but overlooked the primary muscle pathogen, *E. faecium*, possibly leading to inadequate antimicrobial coverage. Successful antibiotic strategy for such polymicrobial infections demands a customized approach: anti-enterococcal agents like linezolid or daptomycin (especially given potential VRE), merged with anti-pseudomonal carbapenems or other broad-spectrum options effective against Gram-negative organisms, including *K. pneumoniae*.[18]

The patient's subsequent acute renal failure requiring renal replacement therapy exemplifies the serious end-organ impairment that can accompany the systemic inflammatory reaction in such infections. His eventual recuperation following vigorous organ support and repeated debridements resonates with published reports stressing the value of a multidisciplinary strategy incorporating intensivists, surgeons, and infectious disease experts.[17]

Another significant feature of this case was the emergence of notable adverse effects—specifically, severe drug-induced dermatitis and neutropenia—ascribed to linezolid treatment. This complication highlights the difficulties in antimicrobial stewardship in critically ill patients and the need for careful surveillance for drug-related toxicities, even when employing targeted medications. The successful reversal of these side effects after switching to vancomycin further underscores the significance of therapeutic flexibility.

Several limitations should be acknowledged. First, genetic confirmation of *hypervirulent K. pneumoniae (hvKP)* and virulence determinants of *E. faecium* (eg, gelE and cylA) were not performed due to the unavailability of whole-genome sequencing Second, fecal carriage of *E faecium* was not assessed; therefore, whether the infecting strain originated from the patient's gut microbiota remains speculative. Third, the absence of *E. faecium* in blood cultures limits the certainty of hematogenous seeding, although transient bacteremia prior to antibiotic administration cannot be excluded.

To our knowledge, only a few cases of necrotizing soft tissue infections attributable to Enterococcus species have been reported, predominantly in immunocompromised hosts.[19] This report adds to the mounting proof that *E. faecium* should not be routinely discounted as a simple contaminant or insignificant colonizer in deep tissue infections, particularly in the milieu of severe sepsis. It also acts as an important alert that even after one pathogen is detected, clinicians should preserve a high suspicion for polymicrobial causation in critically ill patients with rapidly advancing soft tissue involvement. Continuous and detailed microbiological inquiry is vital.

Conclusion

We document the first reported case of necrotizing myositis mainly caused by *Enterococcus faecium*, coinciding with *Klebsiella pneumoniae bacteremia*. This report considerably broadens the recognized scope of severe soft tissue infections

linked to *E. faecium* and demonstrates a potentially fatal synergistic relationship with a Gram-negative pathogen. It stresses the crucial importance of early clinical detection, the value of sophisticated imaging, the necessity of immediate and decisive surgical care, and the need for exhaustive microbiological analysis from deep tissue samples to inform targeted antimicrobial treatment. Additionally, it points out the requirement for attentive monitoring of treatment-associated adverse events. Increased clinical alertness and further investigation into the virulence pathways and polymicrobial dynamics of enterococci in soft tissue milieus are strongly advocated.

Abbreviations

hvKP, Hypervirulent Klebsiella pneumoniae; E. faecium, Enterococcus faecium; NSTIs, necrotizing soft tissue infections.

Ethics Approval and Informed Consent

This study was reviewed and approved by the Ethics Committee of the Affiliated Hospital of Chengde Medical College University (No. CYFYLL2026001) and was conducted in accordance with the principles of the Declaration of Helsinki. Institutional approval was not required for publication of the case details as it does not involve sensitive patient information. Written informed consent to have the case details and any accompanying potentially identifiable images or data published has been obtained from the patient and their family.

Consent for Publication

Written informed consent was obtained from the patient and their family for publication of this report and any accompanying images.

Acknowledgments

The authors would like to thank all participants in this study.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Garcia-Solache M, Rice LB. The Enterococcus: a model of adaptability to its environment. *Clin Microbiol Rev*. 2019;32(2):e00058–18. doi:10.1128/CMR.00058-18
2. Soro O, Kigen C, Nyerere A, et al. Characterization and anti-biofilm activity of lytic enterococcus phage vB_Efs8_KEN04 against clinical isolates of multidrug-resistant *Enterococcus faecalis* in Kenya. *Viruses*. 2024;16(8):1275–1301. doi:10.3390/v16081275
3. Schiopu P, Toc DA, Colosi IA, et al. An overview of the factors involved in biofilm production by the *Enterococcus genus*. *Int J Mol Sci*. 2023;24 (14):11577. doi:10.3390/ijms241411577
4. Arias CA, Murray BE. The rise of the Enterococcus: beyond vancomycin resistance. *Nat Rev Microbiol*. 2012;10(4):266–278. doi:10.1038/nrmicro2761
5. Russo TA, Marr CM. Hypervirulent *Klebsiella pneumoniae*. *Clin Microbiol Rev*. 2019;32(3):e00001–19. doi:10.1128/CMR.00001-19
6. Kong L, Wang Y, Ji H, et al. A case of forearm soft tissue infection caused by hypervirulent *K. pneumoniae* in an otherwise healthy 24-year-old woman. *Infect Drug Resist*. 2022;15:63–68. doi:10.2147/IDR.S342019
7. Guo MY, Liu Y, Fei B, et al. Research progress on virulence factors of hypervirulent *Klebsiella pneumoniae*. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2021;55(11):1357–1363. doi:10.3760/cma.j.cn112150-20210730-00732
8. Stevens DL, Bryant AE. Necrotizing soft-tissue infections. *N Engl J Med*. 2017;377(23):2253–2265. doi:10.1056/NEJMra1600673
9. Shivalingappa S, Manjunath KN, Waiker V, et al. Necrotising fasciitis: appearances can be deceptive. *World J Plast Surg*. 2021;10(1):43–52. doi:10.29252/wjps.10.1.43
10. Fernando SM, Tran A, Cheng W, et al. Necrotizing soft tissue infection: diagnostic accuracy of physical examination, imaging, and LRINEC score: a systematic review and meta-analysis. *Ann Surg*. 2019;269(1):58–65. doi:10.1097/SLA.0000000000002774
11. Jahagirdar S, Morris L, Benis N, et al. Analysis of host-pathogen gene association networks reveals patient-specific response to streptococcal and polymicrobial necrotising soft tissue infections. *BMC Med*. 2022;20(1):173–191. doi:10.1186/s12916-022-02355-8
12. Thänert R, Itzek A, Hoßmann J, et al. Molecular profiling of tissue biopsies reveals unique signatures associated with streptococcal necrotizing soft tissue infections. *Nat Commun*. 2019;10(1):3846–3861. doi:10.1038/s41467-019-11722-8
13. Khatib R, Sharma M, Iyer S, Fakih MG, Opara KN, Riederer K. Polymicrobial *Staphylococcus aureus* bacteremia: frequency, distinguishing characteristics and outcome. *Diagn Microbiol Infect Dis*. 2016;86(3):311–315. doi:10.1016/j.diagmicrobio.2016.08.010
14. Moura FA, Siqueira AIAN. Gut-liver axis in sepsis-associated liver injury: epidemiology, challenges and clinical practice. *World J Gastroenterol*. 2025;31(1):99987. doi:10.3748/wjg.v31.i1.99987

15. Madsen MB, Skrede S, Perner A, et al. Patient's characteristics and outcomes in necrotizing soft-tissue infections: results from a Scandinavian, multicenter, prospective cohort study. *Intensive Care Med*. 2019;45(9):1241–1251. doi:10.1007/s00134-019-05730-x
16. Weaver JS, Omar IM, Mar WA, et al. Magnetic resonance imaging of musculoskeletal infections. *Pol J Radiol*. 2022;87:e141–e162. doi:10.5114/pjr.2022.113825
17. Misiakos EP, Bagias G, Papadopoulos I, et al. Early diagnosis and surgical treatment for necrotizing fasciitis: a multicenter study. *Front Surg*. 2017;4:5. doi:10.3389/fsurg.2017.00005
18. Tamma PD, Aitken SL, Bonomo RA, Mathers AJ, van Duin D, Clancy CJ. Infectious Diseases Society of America Guidance on the Treatment of Extended-Spectrum β-lactamase Producing Enterobacterales (ESBL-E), Carbapenem-Resistant Enterobacterales (CRE), and Pseudomonas aeruginosa with Difficult-to-Treat Resistance (DTR-P. aeruginosa). *Clin Infect Dis*. 2021;72(7):e169–e183. doi:10.1093/cid/ciaa1478
19. Kohli P, Ichhpujani P, Bansal RK, Kumar S. A rare case of *Enterococcus faecalis*-induced orbital cellulitis and myositis. *Indian J Ophthalmol*. 2016;64(8):609–611. doi:10.4103/0301-4738.191513