Lactobacillus Necrotizing Soft Tissue Infection in the Lower Extremity

University of Cincinnati
Department of Orthopedics

Michael Liette DPM, Christopher Goddard CNP, Bryce Yamat DPM, Adena Mahadai DPM, Suhail Masadeh DPM
University of Cincinnati Medical Center, Cincinnati Ohio

Statement of Purpose

Necrotizing Fasciitis is a rapid progressive soft tissue infection characterized by extensive necrosis of fascial and subcutaneous tissue. Traditionally, this severe condition has been predominantly associated with well established pathogens, such as Streptococcus and Staphylococcus species. In this case report we aim to present a rare case of Lactobacillus-induced necrotizing fasciitis in the lower extremity, previously unreported in medical literature.

Literature Review

Lactobacillus species are a diverse group of gram-positive rod-shaped bacterium commonly considered a beneficial bacterium and an essential part of the human microbiota [1]. Once considered harmless commensal organisms certain strains of Lactobacillus have been implicated in an array of infectious diseases in various anatomic sites, most commonly reported in the genitourinary tract, where it can cause conditions like bacterial vaginosis, and the gastrointestinal tract, where it can cause invasive infections such as bacteremia or endocarditis [2]. The pathophysiology of Lactobacillus infections is not fully elucidated, especially in the context of rare and atypical infections. Lactobacillus infections can occur when certain strains of this bacterial genus become pathogenic and cause disease in susceptible individuals such as those with uncontrolled diabetes, individuals undergoing immunosuppressive therapies, or individuals suffering from chronic diseases [2]. In the context of uncontrolled diabetes the pathophysiological process of increased vascular permeability and microangiopathy, attributed to basement membrane glycosylation as well as metabolic alterations leading to endothelial cell injury could represent a potential mechanism for bacterial translocation to the bloodstream and dissemination to other locations within the body [3]. Certain strains of lactobacillus have exhibited varying expression levels of virulence factors including sex pheromones, gelatinase, cytolysin, hyaluronidase, aggregation substance, enterococcal surface protein, endocarditis antigen, and adhesion to collagen. Additionally, these strains have demonstrated diverse patterns of antibiotic resistance for vancomycin, tetracyclines, erythromycin, gentamicin, chloramphenicol, and bacitracin [4]. It has been suggested that adhesion to mucus surfaces, may contribute to the development of bacteremia by extending their persistence and promoting colonization. Apostolou et al. investigated a total of 55 strains encompassing isolates from bacteremia cases, fecal samples of healthy subjects, and dairy-derived strains. Notably, they observed significantly elevated adhesion levels to intestinal mucous in clinical isolates compared to nonclinical isolates and dairy strains. These findings suggest a plausible association between mucus adhesion and translocation of lactobacilli in patients harboring host risk factors [5]. Lactobacillus species are also known to form biofilms which are communities of bacteria encased in protective matrix. Biofilms can enhance bacterial persistence and resistance to host defenses and antimicrobial treatments [6]. The literature on the pathophysiology of lactobacillus is limited, and the mechanisms may vary depending on the specific site of infection and strain involved. Primarily, the majority of reported cases concerning lactobacillus infection involve bacteremia from localized infection and endocarditis. To our knowledge, there is an absence of documented cases associated with lactobacillus infections affecting the lower extremity.

Case Presentation

A 52 year-old male with complex medical history of uncontrolled type II diabetes mellitus (A1C 16.6), hyperlipemia, hypertension, peripheral neuropathy, and previous history of Fournier's gangrene was brought urgently to the emergency department due to concerns for sepsis, which arose in the context of a diabetic foot infection, with high suspicion of necrotizing skin involvement (LRINEC 10). Notably, radiographs and CT imaging did not reveal evidence of soft tissue emphysema. Clinical examination unveiled significant skin discoloration extending to midfoot region accompanied by malodor, purulence, erythema, calor, and hemorrhagic bullae to hallux and lesser digits. Given the clinical presentation and underlying risk factors the decision was made to proceed with emergent incision and drainage.

Figure 1: Clinical image of plantar foot

Figure 2: Clinical image of dorsal foot

Figure 3: Radiographs right foot

Surgical Management

The patient underwent emergency exploration, during which a transmetatarsal amputation was performed due to the extent of soft tissue compromise. Intraoperative findings revealed malodor, purulence, and central tracking of infection to the dorsal midfoot with extensive soft tissue necrosis. Deep specimens were obtained, and wide excision was performed until a physiologic wound bed was encountered, revealing blunt stops in separable fascial planes. Metatarsal heads were removed and irrigation was performed. Microbiological and pathological analyses of clean bone margins were undertaken.

Microbiological Analysis

Intraoperative cultures from the initial debridement identified a mixed microbial infection, including heavy growth of Lactobacillus species, scant growth of Coagulase-negative Staphylococcus, and Candida Glabrata in dirty cultures.

Figure 4: Intra operative dirty culture results

Gram Stain Result	No Polymorphonuclear Leukocytes Seen
Gram Stain Result	No Organisms Seen
Culture Result	No Growth After 3 Days
Culture Result	No Fungus Isolated At 4 Weeks
Culture Result	No Anaerobes Isolated in 5 Days

Figure 5: Intra operative clean cultures results

Treatment and Clinical Course

The patient's initial antibiotic regimen of intravenous daptomycin, clindamycin, and Zosyn was modified for the following culture results. Clindamycin and daptomycin were discontinued, and fluconazole was added due to the presence of Candida Glabrata. Two subsequent wound debridements were performed to facilitate the grafting of large surgical wound with wound vac application for wound management. During the patient's hospitalization, Zosyn was de-escalated to Unasyn, which was continued through discharge. The patient was transitioned to oral Augmentin per infectious disease recommendations and Fluconazole was stopped at discharge as subsequent cultures remained negative for Candida growth. At the 6th month follow-up, the wound bed measures 2.3 cm x 2.2 cm x .5cm, indicating favorable progress in wound healing.

Figure 6: Post initial debridement

Figure 7: 6 month follow up

Analysis and Discussion

The presented case highlights a rare occurrence of a pathogen not known to cause necrotizing fasciitis. The findings in the presented case emphasize the importance of recognizing uncommon pathogens in patients with predisposing risk factors like uncontrolled diabetes and immunosuppression. Diagnosis of opportunistic infections like Lactobacillus can be challenging and typically involve isolating and identifying specific Lactobacillus strain from clinical samples. Due to the rarity of Lactobacillus infections, there is limited data on standardized treatment approaches. Many Lactobacilli case reports lack comprehensive identification methods, leading to common conclusion at the genus level "Lactobacillus spp." The management typically involves antimicrobial therapy and, in the present case, sequential debridements. Further research is needed to better understand factors that contribute to the transition of commensal organism to pathogen to develop effective prevention and tailored treatment strategies for these infections.

References

1. Goldstein EJ, Tyrrell KL, Citron DM. Lactobacillus species: taxonomic complexity and controversial susceptibilities. Clin Infect Dis. 2015 May 15;60 Suppl 2:S98-107. doi: 10.1093/cid/civ072. PMID: 25922408.
2. Rossi F, Amadoro C, Gasperi M, Colavita G. Lactobacilli Infection Case Reports in the Last Three Years and Safety Implications. Nutrients. 2022 Mar 11;14(6):1178. doi: 10.3390/nu14061178. PMID: 35334835; PMCID: PMC8954171.
3. Omar AM, Ahmadi N, Ombada M, Fuscaldo J, Siddiqui N, Safo M, Nalamalapu S. Breaking Bad: a case of Lactobacillus bacteremia and liver abscess. J Community Hosp Intern Med Perspect. 2019 Jun 19;9(3):235-239. doi: 10.1080/20009666.2019.1607704. PMID: 31258864; PMCID: PMC6586093.
4. Todorov SD, Perin LM, Carneiro BM, Rahal P, Holzapfel W, Nero LA. Safety of Lactobacillus plantarum ST8Sh and Its Bacteriocin. Probiotics Antimicrob Proteins. 2017 Sep;9(3):334-344. doi: 10.1007/s12602-017-9260-3. PMID: 28233282.
5. Apostolou E, Kirjavainen PV, Saxelin M, Rautelin H, Valtonen V, Salminen SJ, Ouwehand AC. Good adhesion properties of probiotics: a potential risk for bacteremia? FEMS Immunol Med Microbiol. 2001 Jul;31(1):35-9. doi: 10.1111/j.1574-695X.2001.tb01583.x. PMID: 11476979.
6. O'Callaghan J, O'Toole PW. Lactobacillus: host-microbe relationships. Curr Top Microbiol Immunol. 2013;358:119-54. doi: 10.1007/82_2011_187. PMID: 22102141.