Shewanella putrefaciens Abscess of the Lower Extremity

Shewanella (Pseudomonas) putrefaciens is a ubiquitous organism that has been isolated from many foods, sewage, and both freshwater and salt water (9). Isolation of this organism from human specimens most commonly represents colonization. However, case reports describing this organism as a human pathogen exist in the literature. Descriptions of localized infections such as cellulitis or abscesses (3, 5, 6) and bacteremia (1, 2, 6–8) comprise the majority of these case reports. Patients who developed bacteremia had associated underlying illnesses: diabetes mellitus (2), burns (10), liver or biliary disease (2), or renal failure (1, 4). The pathogenesis of localized infections is thought to proceed from colonization to subsequent invasion (5). Herein we describe a case of S. putrefaciens abscess not associated with a preceding ulcer or evidence suggesting prior colonization.

A 61-year-old Caucasian male with chronic obstructive pulmonary disease and peripheral vascular disease was admitted for management of a foot abscess. His history was significant in that he had 2 years of bilateral lower-extremity discomfort from vascular insufficiency and increased pain in his right foot 10 days prior to admission. The dorsal aspect of his foot became red, swollen, and painful, and drainage of thick purulent material developed. He denied fever, chills, exposure to any freshwater or salt water (despite living on a boat), insect bites, trauma, or recent travel.

Examination revealed a nontoxic-appearing obese, afibrile male. His examination was only remarkable for a large, fluctuant, erythematous area on the dorsum of his foot. Grossly purulent material was draining from the dorsum of his foot. Admission laboratory findings were unremarkable. Foot radiography and bone scan results failed to show signs consistent with osteomyelitis. The abscess was incised and drained, and a Gram stain of the material demonstrated 1+ polymorphonuclear leukocytes, and 1+ gram-negative rods. Cultures grew S. putrefaciens, which was identified with 99% certainty by the VITEK System (bioMérieux Vitek, Inc., Hazelwood, Mo.) (positive for oxidase, H2S, urease, tryptophan, and ornithine). Cultures also revealed growth of coagulase-negative staphylococci, but this was not felt to be significant. Antimicrobial susceptibility was determined by the Kirby-Bauer technique and revealed organism susceptibility to trimethoprim-sulfamethoxazole, cefoxitin, ceftriaxone, aztreonam, imipenem, gentamicin, tobramycin, and amikacin (based on National Committee for Clinical Laboratory Standards performance standards [9a]). The patient was initially treated with ceftriaxone and clindamycin but was discharged while on therapy with trimethoprim-sulfamethoxazole. He responded well to treatment.

S. putrefaciens most likely represents an opportunistic infection given the rarity of disease, localization to devitalized or denuded skin areas, and infection relatively restricted to immunocompromised hosts. Our patient’s case represents only one of two reports clearly demonstrating true abscess formation. The case reported by Debois et al. was associated with an overlying ulcer (5). Our patient had no findings suggestive of predisposing ulceration. We speculate that colonization of the overlying skin and infection via some poorly defined portal of entry was the pathogenic mechanism of his illness. Certainly our patient had ample opportunity for water exposure. This patient responded extremely well to incision and drainage and appropriate antimicrobial therapy. This case report merely adds another organism to the differential diagnostic list of skin and soft tissue infections in immunocompromised hosts.

REFERENCES


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