Staphylococcus lugdunensis Osteomyelitis: A Case Report

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Advanced techniques used to type coagulase-negative *Staphylococcus* have led to improved identification of pathogenic organisms. *Staphylococcus lugdunensis* has rarely been reported as an organism causing osteomyelitis. We report the first case of *S lugdunensis* osteomyelitis after a perforating injury.

Case Report

An otherwise healthy 8-year-old boy presented to the pediatric emergency department of our institution complaining of pain in his right foot. Approximately 16 weeks prior to presentation, the patient reported stepping on a nail while wearing sneakers. At the time of injury, the patient was on a beach in the country of Tobago.

The patient was evaluated and treated by a physician in Tobago, where local wound care and antibiotic therapy were instituted. At the time of injury, a radiograph was obtained and was reported as negative for fracture or foreign body. The patient completed a 3-week course of cefotaxime, after which the patient remained asymptomatic for 10 weeks. Over the course of the week prior to presentation, the patient reported increasing pain with ambulation over the site of the previous wound, causing him to seek our assistance. The patient denied a history of fever, chills, night sweats, or other constitutional symptoms. In addition, there was no history of erythema or drainage from the puncture site. The patient had a benign medical history and was taking no medications.

Initial evaluation found a healthy 8-year-old male child with a hyperpigmented 1-mm mark on the plantar surface of the foot directly under the cuboid. The patient was afebrile, and his vital signs were within normal limits. There was no evidence of erythema, increased calor, drainage, or any detectable sinus. The site was not tender to palpation, nor was there any fluctuance, and no palpable popliteal or inguinal lymphadenopathy was noted. The child ambulated with an antalgic gait, but he was able to bear full weight on the affected extremity.

Laboratory investigations performed on admission demonstrated a white blood cell count of 6.25 K/μL with a differential of 30 neutrophils, 44 lymphocytes, 6 macrophages, 2 basophils, 13 eosinophils, and 5 atypical lymphocytes. Erythrocyte sedimentation rate was 32 mm/hr. Blood cultures were negative. Radiographs were consistent with osteomyelitis of the cuboid (Figure).

Two doses of oxacillin were administered. This was discontinued after consultation with members of the pediatric orthopedic surgery and infectious diseases services; both services suggested surgical intervention. After discussion of the risks, benefits, and alternatives with the patient and his mother, the child was brought to the operating room to débride the cuboid and to obtain specimens for culture. An incision was made on the lateral aspect of the foot overlying the cuboid. The periosteum was incised with a scalpel. With fluoroscopic guidance, a curette was used to excise the central portion of the cuboid containing the osteolytic defect. The bone was sent as 4 specimens for aerobic, anaerobic, fungal, and acid-fast bacilli culture. A small drain was placed prior to skin closure and was discontinued on the first postoperative day because no drainage

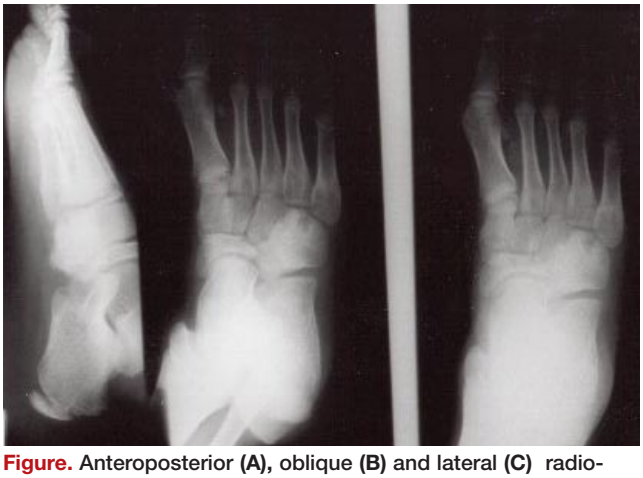

Figure. Anteroposterior **(A)**, oblique **(B)** and lateral **(C)** radiographs of the foot showing a lucency in the cuboid consistent with osteomyelitis.

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was noted. Empiric therapy was initiated by the infectious disease service using gentamicin and ticarcillin combined with clavulanic acid for 5 days. Aminoglycoside troughs were noted to be therapeutic. At 3 days, cultures revealed *Staphylococcus lugdunensis* in the aerobic specimen, and treatment was changed to orally administered levofloxacin.

After 2 months of close follow-up, the patient's wound had healed and there was no clinical or radiographic evidence of infection, with complete resolution of the patient's pain.

Discussion

Ninety-eight percent of all puncture wounds to the foot result from stepping on a nail.[1] Osteomyelitis is a relatively rare, but well-described sequela of nail injuries to the foot. Although most nail injuries heal completely, up to 10% may become infected and produce late complications,[2] and about 0.6% to 1.8% will develop osteomyelitis.[1] In 10% of children with osteomyelitis, the primary site of involvement is one of the bones of the foot, with the calcaneus being the most common, followed by, in decreasing order of frequency, the metatarsals, cuboid, talus, phalanges, and cuneiforms.[3] Common causative organisms are *Staphylococcus aureus* and *Pseudomonas*.[1] We believe that this represents the first case of *S lugdunensis* osteomyelitis in an otherwise healthy individual secondary to a puncture wound.

Staphylococcus lugdunensis was first described in 1989 by Freney and colleagues.[4] *Staphylococcus lugdunensis* is a coagulase-negative staphylococcus; it has been recognized as an important cause of endocarditis with the ability to involve native heart valves, resulting in an aggressive, often fatal illness. *Staphylococcus lugdunensis* can produce a wide range of infections that vary from life-threatening to benign, because of its ability to produce toxins and cell wall proteins. Most strains (95%) of *S lugdunensis* produce a delta-hemolysin that shares homology with the delta-toxin found in *S aureus*.[5] About 12% of *S lugdunensis* strains are thought to produce adherins.[6]

Staphylococcus lugdunensis is a normal inhabitant of the skin flora that more commonly serves as a blood-borne microbe or in skin and soft-tissue infections after a traumatic event. Up to 80% of its isolates have been obtained from skin surface sites, with blood and blood-related devices the only other prominent sources (9.6%).[7] The bacterium has been implicated in bacteremia, endocarditis, peritonitis associated with peritoneal dialysis, and vascular prosthesis infections.

Unlike other coagulase-negative staphylococcal species, *S lugdunensis* is often methicillin sensitive. Herchline and colleagues[8] examined 59 clinical isolates of *S lugdunensis* and found that 76% were beta-lactamase negative. Those that were beta-lactamase negative had penicillin G minimum inhibitory concentrations (MICs) of less than or equal to 0.13 μg/mL, and the remaining 24% were beta-lactamase positive with penicillin MICs of greater than or equal to 0.5 μg/mL. In Europe, however, the incidence of beta-lactamase production has been demonstrated to be lower.[9]

Although described as a causative agent in soft-tissue infections, *S lugdunensis* has been infrequently implicated in cases of osteomyelitis or joint space infections. One report describes *S lugdunensis* causing vertebral osteomyelitis[10] in an immunocompromised patient. *Staphylococcus lugdunensis* has also been reported to have seeded a prosthetic hip implant and to have caused septic arthritis after a knee arthroscopy.[9]

Herchline and Ayers[7] reported a series of patients in whom *S lugdunensis* accounted for 10.1% of staphylococcal species not classified as *S aureus* or *S epidermidis*. In their series, most patients had at least 1 predisposing illness, and half were diabetic or had previous surgery. Most of the patients had low-grade fevers, but systemic signs of infection were generally absent. Of their patients with *S lugdunensis* isolates, in 40% of the specimens *S lugdunensis* was the sole organism isolated, and in the remaining 60% it was part of the mixed flora. Our patient differs in that he had no predisposing illness or surgery and received the inoculum through penetrating trauma.

Conclusions

Staphylococcus lugdunensis is a recently described organism.[4] Advances in typing of bacteria have led to its identification, and it is possible that *S epidermidis* species previously implicated as causative agents for osteomyelitis might have been incorrectly identified. Despite its proven virulence, *S lugdunensis* remains sensitive to most antistaphylococcal antibiotics. Clearly, *S lugdunensis* should be regarded and treated as a form of coagulase-negative staphylococcus that is pathogenic to human beings.

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