

LITERATURE REVIEW AND STATEMENT OF PURPOSE

Pediatric calcaneal acute hematogenous osteomyelitis (AHO) is a rare condition with a reported annual incidence of 2-13 children per 100,000 in well developed countries. This condition most often affects young males between the ages of 2-9 (1,2,3,4). Prior to introduction of systemic antibiotic therapy, mortality rates were reported as high as 30% and 50% for pediatric femoral and tibial osteomyelitis, respectively. Today, the mortality rate has decreased to nearly zero (1,2,3). Hematogenous spread during an episode of bacteremia is the most common etiology for pediatric patients (1,3). In a systematic review published in 2012, Dartnell et. al. reported that the calcaneus is the 7th most frequently affected bone at 4.6% (2). Additional studies have shown similar incidence in the calcaneus, ranging from 3-12% (3,4,5). *Staphylococcus aureus* (*S. aureus*) is the most commonly isolated organism in cases of AHO worldwide, cultured in upwards of 80% of culture-positive cases (1,6).

Time from symptom onset to presentation has been reported between 1 to 34 days, with an average of 6.8 days (4,7). Diagnosis is often delayed due to the insidious onset of symptoms, with a diagnosis >5 days from symptom onset defined as late (8). Delayed diagnosis can be detrimental to the patient and result in devastating complications including angular deformity, joint disruption, growth arrest, chronic osteomyelitis, pathologic fractures, toxic shock and rarely death (3,9).

The purpose of this report is to present a complex case of extensive acute hematogenous osteomyelitis (AHO) of the calcaneus with 63 day delay in diagnosis in a pediatric patient. To our knowledge, this is the longest reported delay in diagnosis and treatment of pediatric calcaneal AHO to date.

CASE STUDY

A healthy 12 year old African American male presented to the Emergency Department (ED) with increasing left heel pain of 2 months of duration after he hit his left heel on a curb, with a sinus tract lesion present for 1 week on the lateral aspect of his heel (Figure 1).



Figure 1. Initial clinical presentation at our Emergency Department, 2 months after symptom onset.

CASE STUDY CONTINUED

Five days after initial symptom onset, the patient was evaluated at an unaffiliated emergency department for this left heel pain. During this visit, he was febrile to 38.5° C, tachycardic, but the physical examination was documented as grossly unremarkable with the exception of pinpoint tenderness to the left plantar calcaneus. Left foot radiographs were obtained without evidence of osseous abnormality. The patient was diagnosed with a left foot contusion and unknown viral illness. He was subsequently discharged with instructions to rest, ice and take NSAIDs as needed for pain. He was again re-evaluated for increasing heel pain at the 3 week mark in an unaffiliated walk-in clinic where repeat radiographs were obtained and found to be negative.

During his evaluation in our Emergency Department, physical examination demonstrated mild edema and warmth to the left heel with a sinus tract from the lateral heel probing directly to bone. There was significant point tenderness in the area. He was vitally stable with white blood cell count (WBC) of 8.19, an erythrocyte sedimentation rate (ESR) of 46, and C-reactive protein (CRP) of 13.6. Radiographs were significant for erosions and cortical destruction of the posterior calcaneus (Figure 2). MRI was revealing for extensive calcaneal osteomyelitis with serpiginous intraosseous abscess, two cutaneous sinus tracts at the lateral hindfoot and inter-muscular edema (Figure 3). The patient was admitted to Pediatric Medicine with Infectious Disease, Podiatric Surgery, and Hyperbaric Medicine as consulting teams.

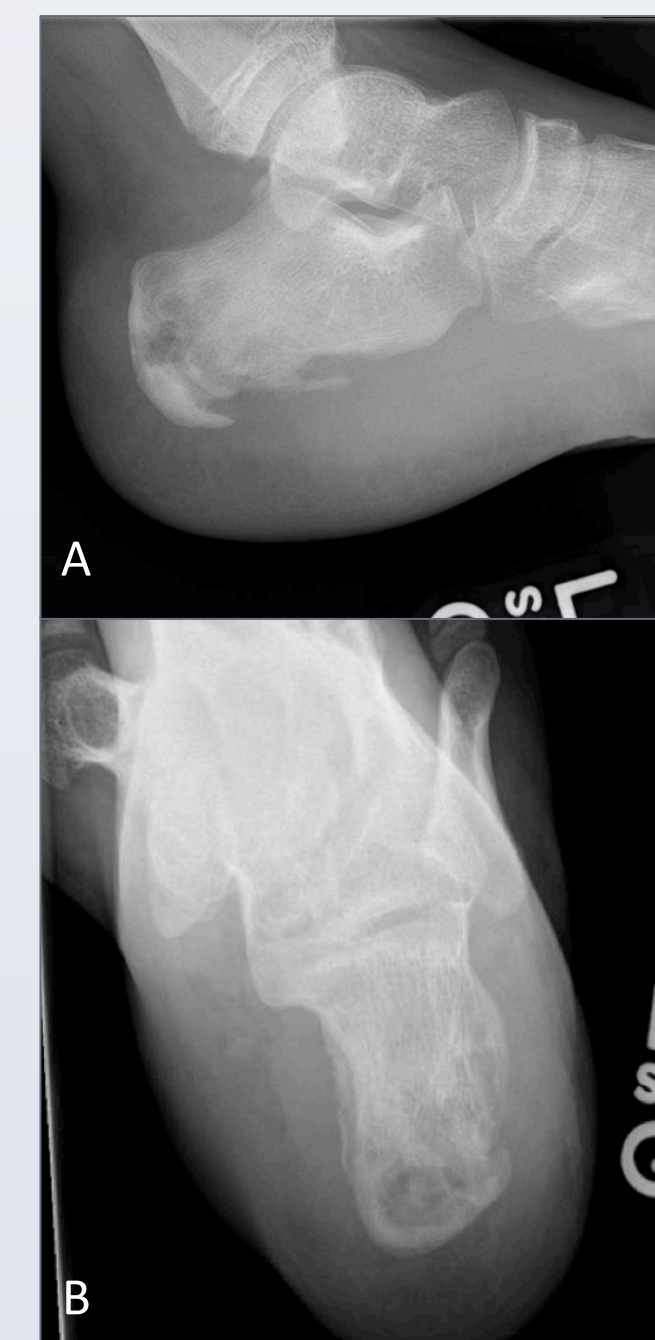


Figure 2. Lateral (A) and calcaneal axial (B) radiographs of the left foot demonstrate well-circumscribed lucency within the posterior calcaneus.

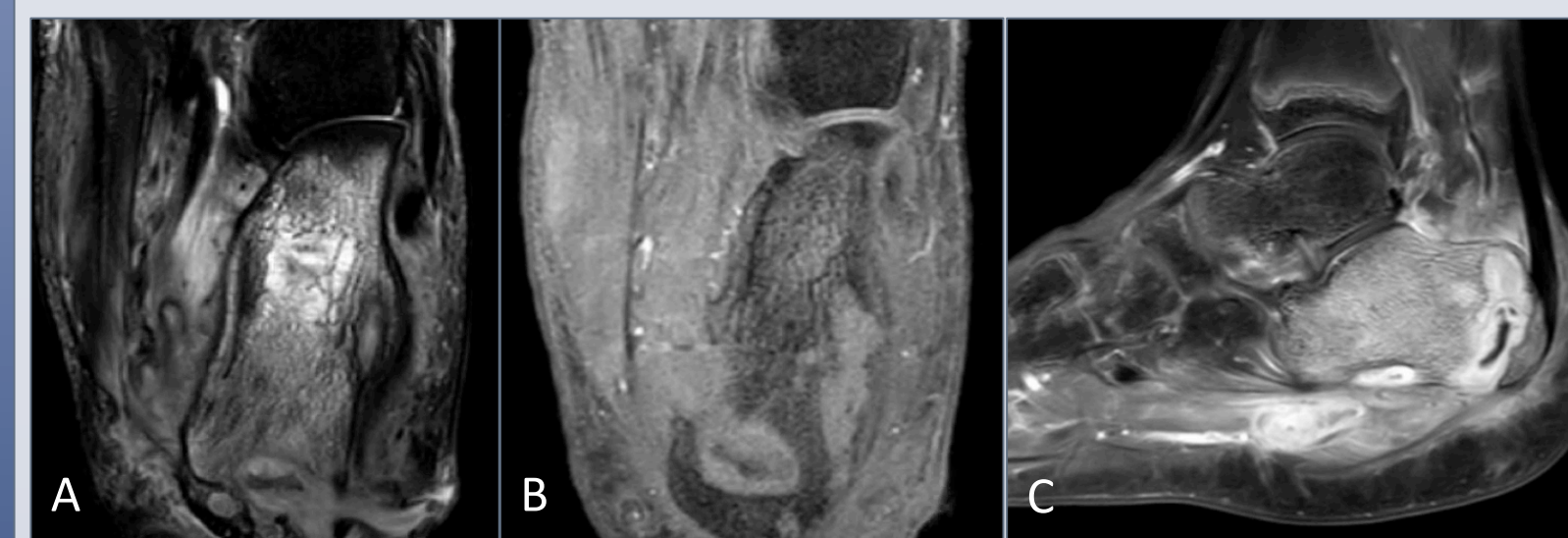


Figure 3. Fat-suppressed T2 axial (A) fat-suppressed T1 axial (B) and fat-suppressed T1 sagittal (C) MRI images of the left foot demonstrate evidence of extensive calcaneal osteomyelitis with serpiginous intraosseous abscess formation.

CASE STUDY CONTINUED

After admission, the patient was immediately scheduled for incision and drainage (I&D) of the left foot with intra-operative bone biopsy for the following morning. Antibiotic therapy was delayed until bone biopsy was obtained, per Infectious Disease recommendations. After consultation with Hyperbaric Medicine, the patient was deemed appropriate for Hyperbaric Oxygen Therapy (HBOT) with a planned course of 20 daily treatments.

The following morning, the patient was taken to the operating room. He was placed on the operating table in the supine position, general anesthesia was administered and an ankle block was performed with local anesthetic. A thigh tourniquet was placed; however, it was note inflated for the case. A single incision was made along the lateral heel, completely excising the two draining sinuses. A subcutaneous abscess was encountered along with a large cavernous defect in the lateral calcaneal wall measuring approximately 0.75cm x 1.0cm, tracking 5.0cm across the body of the calcaneus. A curette was utilized to remove all soft, non-viable bone of which a sample was sent to pathology and microbiology for culture and antibiotic sensitivities. A second incision was made along the plantar medial calcaneus, revealing a deep soft tissue abscess which communicated with the laterally based sinuses. The area was then irrigated with sterile saline, incisions partially closed with retention sutures and packed with betadine soaked packing strips and a sterile dressing was applied. The patient was instructed to be strictly non-weight bearing on the left lower extremity.

Bone cultures grew methicillin sensitive *Staphylococcus aureus* (MSSA) and the patient was started on IV Vancomycin. He was transitioned to IV Nafcillin for a 42 day duration based on culture sensitivities. The patient underwent 7 additional operative I&Ds which included placement of vancomycin antibiotic impregnated beads, tobramycin antibiotic spacer and final

placement of synthetic bone graft substitute bone filler to occupy the large cavernous defect in the lateral calcaneal wall (Figure 4). Calcaneal biopsies were obtained at each procedure, and grew MSSA until the 6th procedure (Figure 5). Inflammatory markers were trended throughout treatment, with both ESR and CRP normalizing to 15 and 0.7.



Figure 4. Lateral calcaneal wall defect during 6th I&D procedure.

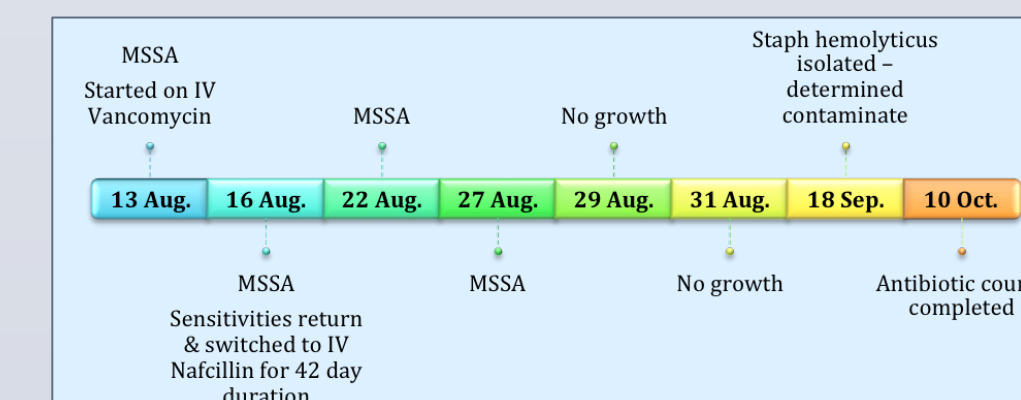


Figure 5. Timeline of calcaneal biopsy culture results and antibiotic course.

RESULTS

After completion of antibiotics, 20 HBOT sessions and 3 consecutively negative calcaneal biopsies, the patient was discharged. Radiographs prior to discharge revealed early incorporation of bone filler (Figure 6). He was kept non weight bearing in a posterior splint for an additional 6 weeks past the date of last surgery. His postoperative course was uneventful. Follow up radiographs obtained 8 months after initial presentation revealed full incorporation of bone filler with no acute osseous pathology (Figure 7). At 12 month follow up the patient had returned to all previous activities without pain or functional disability. He denied any sensory deficits, was ambulating pain free and all incisions remained healed (Figure 8).

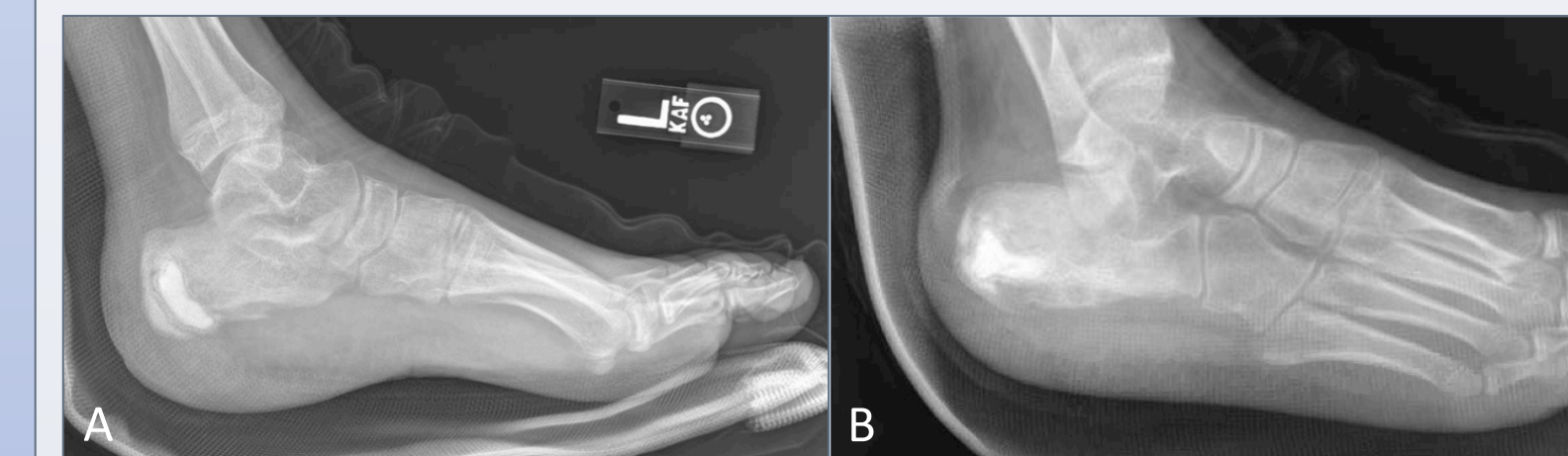


Figure 6. Lateral (A) and oblique (B) radiographs on date of discharge reveal early incorporation of bone filler.

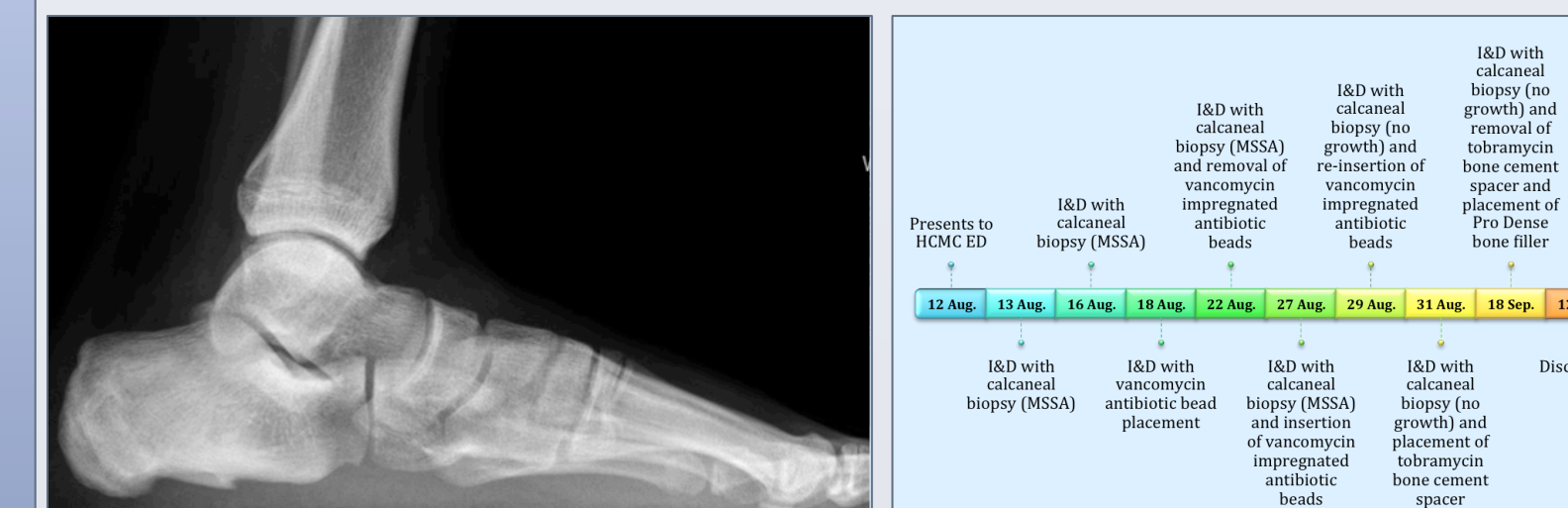


Figure 7. Radiographic evidence of full incorporation of bone filler at 8 month follow up.

Figure 8. Timeline of hospital course.

ANALYSIS AND DISCUSSION

Pediatric calcaneal AHO has a high incidence of delayed and misdiagnosed attributable to nonspecific symptoms that mimic more common, benign calcaneal pathologies. This can result in devastating complications. Diagnosis is based on history, clinical examination, laboratory values and imaging studies. Once confirmed, treatment should commence immediately.

Our case report substantiates much of the previously publicized literature on pediatric calcaneal AHO. Our patient was male, his bone cultures grew *S. aureus*, he initially sought medical care within 5 days of symptom onset, had a fever at initial presentation and reported a history of blunt trauma to the left heel. His laboratory values were consistent with AHO with an elevated ESR and CRP. Initial radiographs obtained at 5 and 22 days post symptom onset were negative for acute pathology and he was misdiagnosed with a deep heel contusion at two separate facilities prior to accurate diagnosis.

ANALYSIS AND DISCUSSION CONTINUED

Due to the advanced stage of AHO upon presentation to our facility, aggressive serial operative debridements were required along with 6 weeks of IV antibiotics and HBOT. Minimal research has been conducted on the efficacy of HBOT and pediatric AHO. Waisman et al. published a report in 1998 on utilization of HBOT in pediatric patients. The authors retrospectively reviewed 139 pediatric patients, 5 with refractory osteomyelitis. All patients participated in an average of 32 HBOT sessions at 2.5 ATA twice daily in conjunction with standard treatment. They observed a 93% favorable outcome in all pediatric patients who received HBOT and advocated for the use of HBOT in the treatment of certain conditions in the pediatric patient (10). Consultation to Hyperbaric Medicine has become an important cornerstone in treatment for pediatric AHO at our facility. In this case, we believe adjunctive HBOT did play an important role in our patient's ultimately successful outcome.

In conclusion, pediatric calcaneal AHO is a rare condition, often initially misdiagnosed. Delayed diagnosis can result in devastating complications with lifelong impacts. High clinical suspicion should exist when any child presents with localized bone pain with or without history of recent illness or inciting event. Advanced imaging should be considered when suspicion exists and if symptoms worsen. Approach to treatment should be multidisciplinary and standard care including bone/joint and blood cultures followed by initial IV antibiotic therapy targeted against *S. aureus* is well established. In complicated cases, addition of aggressive surgical intervention and HBOT may be indicated.

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CONTACT INFORMATION

Michelle L. Winder, DPM
Michelle.winder@hcmcd.org