Infectious Spondylodiscitis, Epidural Phlegmon, and Psoas Abscess as a Complication of a Diabetic Foot Infection: Case Report

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Statement of Purpose

To increase awareness of a potential severe complication of diabetic foot ulceration; vertebral osteomyelitis and associated pathology. A review of the literature, presenting signs and symptoms, and treatment options will be discussed in concomitant with a case report.

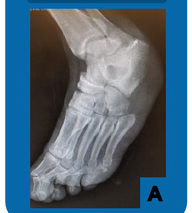
Literature Review

Vertebral osteomyelitis, or infectious spondylodiscitis, may present from via hematogenous spread from a distant source, contiguous from an adjacent source, or direct inoculation during surgical spinal procedures. Staphylococcus aureus is the most common cause of adult hematogenous vertebral osteomyelitis and MRSA has been shown in over 67% of cases.[1] A study by Priest in 2005 reports a 55% causal incidence of vertebral osteomyelitis after an invasive procedure. A 2008 study by Grammatico et al estimates overall incidence of vertebral osteomyelitis far less than one percent, approximately 2.4 per 100,000 people. However vertebral osteomyelitis incidence is increasing and more prominent in patients with co-morbidities, especially diabetics.[3] Currently, there are only a few studies which site the foot as the hematogenous source, and none specifically naming the calcaneus. Ceilley in 1977 discussed a case report of a 71 year-old diabetic female with pedal osteomyelitis who vertebral cultures which had the same organism as those found in the pedal wound and blood: corynebacterium haemolyticum. Cechurova et al in 2013 published a review of three cases of hematogenous spread of Methicillin-resistant Staphylococcus aureus (MRSA) from recurrent neuropathic diabetic foot ulcers.
The following case report illustrates a hematogenous osteomyelitic spread from calcaneal origin. The purpose of this case report is to inform podiatrists of this potential severe osteomyelitis complication and describe what symptoms to be diligent of.

Case Report

A 63 year old male was transferred from an outside hospital to the Cleveland Clinic in Cleveland, Ohio, for management. A magnetic resonance image (MRI) at an outside hospital revealed an epidural abscess T9-L1 level, a ventral fluid collection at L4-5, S1 with extension into the Paraspinous muscle, a right psoas muscle abscess, and vertebral osteomyelitis of L4-5. His medical history included type II Diabetes Mellitus, atrial fibrillation, hypertension, peripheral vascular disease, hyperlipidemia, chronic renal disease, history of renal calculi, sciatica, and recent cessation of tobacco smoking with a 30 pack-year history.
Five months prior, in October 2012, the patient developed a plantar right heel ulceration secondary to a blister.
Wound care treatment from a local podiatrist included wet-to-dry dressings with saline and ambulation in an athletic shoe. The frequency of visits and whether debridement was performed is not known. The ulceration progressed in size in 1.5 months to measure approximately 4.0 cm x 3.0 cm x 2.0 cm. The patient developed right lower extremity cellulitis and edema, and the calcaneus was visible within the ulceration. The patient was admitted to an outside hospital for calcaneal osteomyelitis, and wound cultures revealed MRSA. The patient was treated with intravenous (IV) vancomycin for 4 days, and was discharged on intravenous cefepime for 4 weeks followed by a 10 day course of oral ciprofloxacin.

Figure 1



Case Report Continued

The patient was without antibiotics for 1 week subsequent to this treatment when the patient was admitted to an outside hospital with a sudden onset of severe back pain, fever, and chills. There was a delay in obtaining a spinal MRI because the patient could not tolerate lying in a supine position due to pain. Both blood and calcaneal wound cultures at this admission revealed MRSA. Transesophageal echocardiography (TEE) and transthoracic echocardiogram (TTE) were negative for vegetations. The patient's hospital course was complicated by Clostridium difficile for which the patient was treated with oral Flagyl. The patient then underwent a partial calcanectomy and was placed on IV daptomycin. On the 9th Day of admission, the patient was placed under general anesthesia to obtain a spinal MRI and, after the image was read, the patient was transferred to Cleveland Clinic for further management. Upon arrival, the patient was placed on 1.5g of IV Vancomycin and 3.375g of IV Zosyn.
The physical examination revealed spinal tenderness around the mid-thoracic level to the lower lumbar spine and superior anterior right thigh. The patient was unable to perform hip flexion on the right secondary to pain. Laboratory values revealed a white blood cell count of 13.86 k/uL and an elevated blood glucose of 380 mg/dl.
The patient underwent a spinal and right psoas abscess incision and drainage followed by a direct lumbar inter-body fusion of lumbar vertebrae 4 and 5 with right iliac crest allograft. Bone and abscess cultures were positive for MRSA. Status post surgery, the patient's pain resolved in his back and hip and he regained full right lower extremity function. A repeat vertebral MRI revealed complete resolution of the psoas abscess and stable epidural phlegmon dorsal to the L4 vertebra which measures 5 x 7 x 25 mm, and continued osteomyelitis of L4 and L5 vertebra. Patient was placed on a three month course of 1.5 grams of intravenous vancomycin and has had no complications to date.

Figure 1

A – Non-weight bearing medial oblique right foot/ankle radiograph S/P Right partial calcanectomy

B – Sagittal Lumbar MRI upon admission to Cleveland Clinic. Green arrow points to the epidural abscess superior to L4.

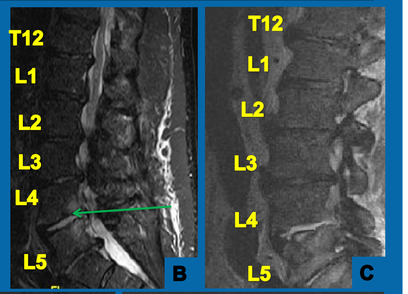
C – Sagittal Lumbar MRI s/p intervention. Note stable epidural abscess superior to L4. Patient now with evidence of L5 vertebral fracture.

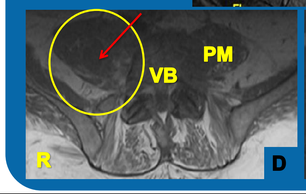
D – Transverse Lumbar MRI upon admission to Cleveland Clinic. PM illustrates left Psoas Major and the yellow circle highlights the enlarged right Psoas Major muscle. The red arrow points to the abscess located within the right Psoas Major muscle. VB illustrates the vertebral body.

E – Transverse Lumbar MRI s/p intervention. PM illustrates left Psoas Major . Note the right psoas Psoas Major muscle has decreased in size and is comparable in size to the left psoas major muscle.

F – Non-weight bearing lateral right foot radiograph S/P Right partial calcanectomy

G – Sagittal Lumbar MRI upon admission to Cleveland Clinic. Yellow arrow points to L4 and L5 vertebrae infected with MRSA.





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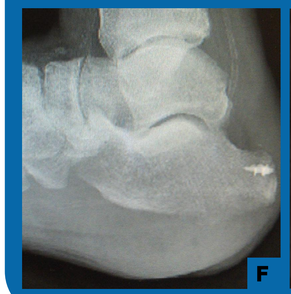

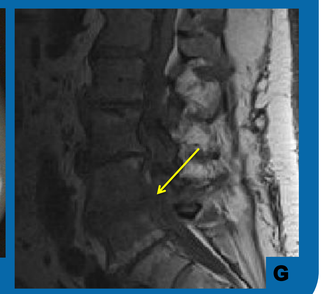

Analysis & Discussion

With reasonable assumption, the pathogenesis of this case was derived from the calcaneal osteomyelitis. The calcaneal ulceration and osteomyelitis antedated the patient's back pain and the patient had no history of a contiguous vertebral infectious source. The initial heel ulceration isolate was invariable to that of the second admission isolate cultured from the patient's blood, calcaneal wound, vertebral bone, and abscess specimens; Methicillin-resistant Staphylococcus aureus. Staphylococcus aureus is the most common isolated pathogen from infected bone and has varying virulence capabilities: glycocalyx secretion, biofilm production, and antibiotic resistance.[6] Biofilm results in bacterial evasion of host defenses and reduces its growth and metabolic activity. As a result, relapses of osteomyelitis have been described up to 80 years after initial presentation.[7] Biofilms are commonly found on retained hardware, prosthetic joints, chronic wounds, and in osteomyelitis.
There are reports of diabetic foot osteomyelitis remission in patients treated with only antibiotic treatment.[8] MRSA is commonly treated with parenteral Vancomycin, a glycopeptide.[6] Vancomycin resistance is still quite rare and effectively treated with linezolid and daptomycin.[6] Beta-Lactam antibiotics (penicillins, cephalosporins, and carbapenems) penetrate infected bone greater than uninfected bone.[6] However, this penetration is markedly decreased in patients with peripheral vascular disease and is likely low in sequestra.[6] Clindamycin has excellent bone penetrance.[6] Rifampin has been shown to penetrate biofilms and kill inactive bacteria. However since Rifampin has a predisposition for resistance development, it should only be used in combination with another MRSA active antibiotic, such as vancomycin, Bactrim, or floroquinolones. Three to four week duration of parenteral antibiotics stems from the belief that it takes 3-4 weeks for infected bone to vascularize.[7] Several infectious disease articles state that there are no clinical studies supporting the superiority of 4-6 week antibiotic duration therapy.[7,9] In the journal of Clinical Infectious diseases, Spellberg and Lipsky in 2012 propose that oral antibiotic therapy with highly bioavailable agents is an acceptable alternative to parenteral therapy, and the preference for parenteral therapy for chronic osteomyelitis is based on custom rather than evidence. In their literature review, there were few parenteral antibiotic studies and success rates were similar for both oral and intravenous routes.[7] They recommended fluoroquinolones or bactrim for 8-16 weeks in combination with surgical debridement.[7] However for gram-positive cocci they recommend the combination of bactrim and clindamycin since fluoroquinolones have a predilection for resistance development.
Many patients eventually require debridement or amputation to completely eradicate infection. Surgical debridement should resect all non-viable material, reduce the bacterial load, and aim to achieve "clear" margins to prevent any nidus for recurrence. Insufficient debridement is correlated with high recurrence rate.[9,11,12] A study by Atway et al. 2012 found residual osteomyelitis in 40.7% (11/27) after clear margins. Kowalski et al. in 2011 found similar findings where 35.14% (39/111) of patients had residual osteomyelitis after clear margins. Simpson et al. 2001 showed that a wide margin greater than 5 mm resulted in 0% recurrence of OM, compared with marginal resection of less than 5 mm, which resulted in 28% recurrence of OM. Functional optimization as well as predicted flap survival integrity should be evaluated prior to amputation level choice. Transmetatarsal amputations result in good functionality when a custom full length shoe is prescribed.[14]
IDSA Guidelines for diabetic bone infections state that if no infected tissue remains following surgery, 2-5 days of antibiotics are required. However, if infected tissue is still present, more than 4 weeks of antibiotics is recommended.[10] In order for antibiotic delivery to effectively penetrate bone, the serum concentration must exceed minimum inhibitory concentration of the organism.[6] Local serum concentration is also limited by adequate blood flow to the infected area, or lack thereof. The blood supply to the site should be assessed [e.g. peripheral vascular resistance (PVR) testing, transcutaneous oxygen (TCOM) testing, arteriograms, etc.] and any blockages should be optimized by vascular surgery.
Hematogenous osteomyelitis is the infectious spread from one osseous site to another through the bloodstream, usually caused by a single organism.[6] The incidence of calcaneal osteomyelitis seeding to the spine is unknown. The most common clinical presentation of vertebral osteomyelitis is back pain, cited to occur in approximately 86% of patients.[15] It occurs most frequently in the lumbar vertebrae (58%), followed by the thoracic (30%), and the cervical spine (11%).[15] Approximately one-third of patients will present with neurological symptoms, such as sensory deficits and weakness on the ipsilateral limb.[15] Severely sharp back pain suggests the presence of an epidural abscess.[15] The diagnosis of vertebral osteomyelitis is made with positive bone cultures. However, Zimmerli, in his review article on Vertebral Osteomyelitis in the New England Journal of Medicine, proposes that the clinical suspicion of vertebral osteomyelitis and two positive blood cultures corresponds to a "probable" diagnosis. Magnetic resonance imaging (MRI) with gadolinium is more sensitive than CT in diagnosing spinal osteomyelitis.[15] Treatment includes 6 weeks to 3 months of parenteral antibiotics followed by 3-6 months of oral therapy. A repeat MRI should only be performed following treatment if there is no clinical improvement at 4 weeks.

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