CLINICAL VIGNETTE

Vertebral Osteomyelitis in Intravenous Drug Abuser

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A 28-year-old male IV drug abuser came to our clinic as a new patient with complain of back pain for 3 months that became worse with activity. Pain mid-lower back with no radiation. He was given Ibuprofen and Norco by his previous physician. He denied weight loss, fever, night sweat nausea and vomiting. No history of injury. He had a significant past medical history with alcohol and drug abuse.

His vitals showed: BP 135/90 mm/hg, Pulse 96/min, T 98 F, W 270 Ibs, Height 179 cm. Physical exam was normal except local tenderness over L1 and L2 and bilateral muscle spasm. His labs showed normal WBC, CMP, blood culture with elevated ESR at 86 mm/hr and normal CRP. LS Spine was normal. Because of the elevated sedimentation rate, he was admitted to the hospital and MRI showed osteomyelitis of L2. CT guided biopsy was also performed and was positive for Pseudomonas.

Patient was initially started on IV Ciproflaxin and vancomycin. After the culture results, we stopped Vancomycin and continued ciproflaxin pf parenteral antibiotics. After 3 weeks he was switched to oral ciproflaxin and discharged home to follow up as outpatient. His weekly ESR improved decreasing to 20 mm/hr at six weeks of treatment. Since patient was still complaining of pain we continued oral Ciproflaxin for another 2 weeks and referred him to physical therapy.

He returned for monthly follow up for 6 months with normal sed rates. His back pain persisted prompting we repeat MRI which was normal. He was started on gabapentin and referred to pain clinic and rehab.

Discussion

Vertebral osteomyelitis most often occurs as a result of hematogenous seeding of one or more vertebral bodies from a distant focus.[1] Vertebral osteomyelitis is primarily a disease of adults with most cases in male patients >50 years old.[2] Risk factors for vertebral osteomyelitis include injection drug use, infective endocarditis, degenerative spine disease, prior spinal surgery, diabetes mellitus, corticosteroid therapy, or other immunocompromised state.

Potential sources of hematogenous or contiguous spread of infection include the genitourinary tract, skin and soft tissue (eg, injection drug use), respiratory tract, infected intravascular devices, postoperative wound infection, infective endocarditis, and dental infection. In many cases, the primary site of infection cannot be identified.[3]

The most common cause of vertebral osteomyelitis is *Staphylococcus aureus*, with many other organisms also reported. These include Enteric gram-negative bacilli, Streptococci, *Pseudomonas aeruginosa*, coagulase-negative staphylococci, *Candida*, especially in association with intravascular access, sepsis, or injection drug use.[4] Tuberculous and Brucellosis are other rare causes.

Pain is the major clinical manifestation of vertebral osteomyelitis. Pain is typically localized to the infected disc space area and is exacerbated by physical activity or percussion to the affected area. Pain may radiate to the abdomen, leg, scrotum, groin, or perineum. Spinal pain usually begins insidiously and progressively worsens over several weeks to months. Fever is an inconsistent finding.[2] Local tenderness to gentle spinal percussion is the most useful clinical sign but is not specific.

The leukocyte count may be elevated or normal. Elevations in the erythrocyte sedimentation rate (ESR), which can exceed 100 mm/h, and C-reactive protein (CRP) are observed in more than 80 percent of patients.[5] If elevated, the ESR and CRP are useful for following the efficacy of therapy. Initial diagnostic tests include ESR, CRP, blood and urine cultures and spinal MRI.

Patients with radiographic evidence of vertebral osteomyelitis should undergo CT-guided needle biopsy of the affected bone and aspiration of abscess if present.[6] If possible, antimicrobial therapy should be withheld until a microbiologic diagnosis is confirmed. MRI is the most sensitive radiographic technique for diagnosis of vertebral osteomyelitis and epidural abscess.[7] MRI abnormalities consistent with osteomyelitis can be observed long before plain films become abnormal. CT is a reasonable alternative imaging modality when MRI is not available. Radionuclide scanning may be useful if MRI is contraindicated.[1]

Management of vertebral osteomyelitis consists of antimicrobial therapy and percutaneous drainage of paravertebral abscess if present. Timely surgical intervention is usually warranted for patients with neurologic deficits, radiographic evidence of epidural or paravertebral abscess, and/or cord compression.

Choice of antibiotic therapy should be guided by biopsy or blood culture results.

Patients with negative Gram stain and culture results should be treated with an antimicrobial regimen with activity against the common causes of vertebral osteomyelitis, including staphylococci, streptococci, and gram-negative bacilli.

Anaerobes are uncommon pathogens in patients with vertebral osteomyelitis.

If empiric therapy does not result in objective clinical improvement in three to four weeks, repeat percutaneous needle biopsy or open surgical biopsy is required.

We routinely treat for a minimum of six weeks, with careful review to determine if further treatment is required.[8] Longer duration of therapy is warranted for patients with undrained paravertebral abscess and/or infection due to drug-resistant organisms. In some cases, up to 12 weeks of therapy may be necessary, particularly in the setting of extensive bone destruction.

During antimicrobial therapy, patients should be followed carefully for clinical signs of soft tissue extension, paraspinal abscess, and cord compression.

Patients should also be followed with weekly monitoring of inflammatory markers (erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP]). One retrospective study included 44 patients with vertebral osteomyelitis found, those without a significant decline in the ESR during the first month of therapy were more likely to fail medical therapy (9 of 18 cases compared with 3 of 26 cases with a >50 percent fall in ESR).[9]

Routine follow-up imaging studies are not necessary. Regular imaging during treatment and in the presence of clinical improvement will not impact the outcome. Magnetic resonance imaging (MRI), CT, and plain films may appear to worsen for several weeks after the initiation of antibiotic therapy that will be ultimately successful.[10]

Follow-up imaging studies are warranted in patients whose clinical status has not improved at the planned time for discontinuation of antibiotics.[1]

Early bed rest may be particularly important, especially in lumbar osteomyelitis. When the patient is upright, the whole weight of the upper body is transmitted to the point of active infection. Many recommend at least 10 days bed rest for patients with severe pain, with intensive in-bed, non–weight bearing physical therapy and long-acting oral analgesics.

The most serious complication of vertebral osteomyelitis is neurologic impairment secondary to either abscess formation or bony collapse. Most patients have gradual improvement in back pain after therapy is begun. Mortality due to vertebral osteomyelitis in the antibiotic era is less than 5 percent, and the rate of residual neurologic deficits among survivors is less than 7 percent. Delays in diagnosis can lead to disabling complications.[1]

Conclusion

Since our patient had a history of drug abuse and was opiate dependent, his workup for back pain took longer .His initial labs and lumbar x ray were normal. The high sed rate (ESR) was key and prompted the MRI. Although his blood culture was negative, since the most common organizism in IV drug users is pseudomonas we covered pseudomonas and added vancomycin to cover the other common infections. Most likely he got the infection because of IV drug use. After negative blood culture, CT guided biopsy resulted in the positive pseudomonas culture.

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