When Anemia, Atypical Plasma Cells, and a Lytic Bone Lesion are not Myeloma: An unusual presentation of osteomyelitis

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Introduction

Osteomyelitis is an infection of bone that leads to tissue destruction and can be caused by a wide variety of organisms including bacterial, fungal, and viral organisms.[1] Osteomyelitis can present with a history of local inflammation, erythema, or swelling. It also can present as low grade fever, malaise, and fatigue, along with non-specific chronic pain at the site of infection. With its myriad of different presentations, osteomyelitis can be hard to diagnose. We describe an unusual case of osteomyelitis thought to be a lytic bone lesion of multiple myeloma.

Case Report

A 64-year-old male was referred for anemia. He had been anemic for six months and received four IV iron infusions. Past workup showed a high level of ferritin (1450 ng/mL). Esophago-gastroduodenoscopy and colonoscopy were negative. Erythrocyte sedimentation rate and C-reactive protein were elevated. Bone marrow biopsy revealed atypical plasma cells accounted for less than 5% of cellularity.

Myeloma workup included a skeletal survey, showing three lucencies in the calvarium, superiorly near the midline, with the largest measuring 6 mm in diameter. Another lucency was present in the proximal right humeral diaphysis. Analysis revealed a beta 2 microglobulin of 3.3 mg/L (normal range 1.21-2.70 mg/L), elevated free kappa (146.16 mg/L), and lambda (65.26 mg/L) light chains with free kappa/lambda ratio of 2.24. A CT of the skull without contrast confirmed a 3 mm lytic lesion within the left frontal skull region thought to be consistent with a myeloma lesion.

During the patient's follow-up visit, he complained of pain in the left forehead. He complained that this area was increasingly painful and tender. With anemia, atypical plasma cells on bone marrow biopsy, a lytic lesion in left frontal skull region, beta 2 microglobulin of 3.3 mg/L, and elevated kappa and lambda free light chains, multiple myeloma was diagnosed.

Biopsy of the growing skull lesion was not done due to the concern that with treatment of multiple myeloma, wound healing of a scalp incision might be impeded. Thus, treatment with bortezomib and dexamethasone was started. The patient tolerated bortezomib well, but the presumptive plasmacytoma on the forehead increased in size to that of an orange and was tender and fluctuant on exam. A CT scan showed an increased size of the bony lytic lesion involving the frontal skull region with adjacent extracranial fluid collection. A craniectomy revealed the presence of a mass and subgaleal abscess.

The resected left frontal mass showed acute and chronically inflamed granulation tissue reaction and marked chronic inflammation consistent with osteomyelitis and non-diagnostic for plasmacytoma. Gram stain showed numerous white blood cells with no micro-organisms and an anaerobic

culture positive for a small amount of Fusobacterium species.

Follow-up post-operative CT scanning showed no new lytic lesions. Treatment for multiple myeloma was discontinued. Repeat films of the humerus showed no evidence of abnormalities.

Discussion

Musculoskeletal infections like osteomyelitis are very common and can be life threatening. It is a diverse disease in its pathophysiology, clinical presentation, and management.[2] Osteomyelitis is an ancient disease and is one of the most difficult infectious diseases to treat. Progressive destruction of the bone and the formation of sequestra are characteristics of this disease.

Osteomyelitis can be due to contiguous spread from adjacent soft tissues and joints, hematogenous seeding, or direct inoculation of microorganisms into the bone as a result of trauma or surgery.[2] A multidisciplinary team is required to treat these patients optimally.[3] Osteomyelitis of the skull usually results from the contiguous spread from an infected sinus or from penetrating trauma (i.e., post-operative).[4]

Our case illustrated an atypical presentation of osteomyelitis with an unusual organism which led to the misdiagnosis of multiple myeloma and resultant cytotoxic treatment. The linchpin was the lytic bone lesions, which were the cornerstone of the diagnosis. There was some uncertainty with both kappa and lambda elevation and the less than 5% plasmacytosis on bone marrow exam, so a biopsy of the calvarial lytic lesion was planned, but not done due to clinical worsening. In this case, a biopsy for tissue confirmation to solidify the diagnosis of multiple myeloma would have resulted in the correct diagnosis and earlier institution of antimicrobial therapy.

Thus, this case appeared to be something that it was not. There were lytic bone lesions, anemia (which in retrospect was due to an inflammatory anemia), elevated beta-2-microglobulin, and elevated serum free light chains (knowing what we now know, this was from the infection), all of which pointed to multiple myeloma.[5] The patient met formal diagnostic criteria for multiple myeloma based on the presence of lytic bone lesions and anemia. In this case, these findings trumped the bone marrow plasma cell percentage, which can be less than 10%, especially in a case where the disease burden appeared to be low based on a paucity of lytic lesions and a minimally elevated beta-2-microglobulin.

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