Unusual Cause of Back Pain in a Multiple Myeloma Patient: Infectious Discitis


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ABSTRACT

A 58 year old patient, followed up for diabetes mellitus and multiple myeloma for ten years and six months, respectively, presented with persistent back pain, weakness at his lower extremity and intermittent fever. Activation markers of myeloma were in normal limits except high erythrocyte sedimentation rate and elevated C-reactive protein. Magnetic resonance imaging revealed increased T1 spinal intensity at the suspected disc level. Discitis was suspected on the basis of MRI and high fever. An intraoperative examination of specimens revealed *Staphylococcus aureus*. Detailed laboratory and radiological investigations should be performed to diagnose unusual causes of persistent back pain, such as discitis in patients with myeloma.

Key Words: Back pain, Multiple myeloma, Discitis.


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INTRODUCTION

Most multiple myeloma (MM) patients have low-back pain as their presenting symptom, and also skeletal lytic lesions, with or without osteoporosis, wedging or collapse of vertebral bodies or pathological fractures[1]. Initially the pain is often rheumatic, wandering and intermittent, and most commonly involves the back, and less often the chest or extremities[2]. In some cases, pain is the result of nerve or spinal cord damage from direct compression by the tumor, or it may be secondary to peripheral neuropathies caused by amyloid deposition or ill-defined nonmetastasis of the malignancy.

Increased susceptibility to infection is another common complication of MM. A number of factors may be responsible for the increased susceptibility of multiple myeloma patients to infections. Most patients with MM have decreased levels of normal immunoglobin, a defective response to primary and secondary immune response, and a diminished antigen and mitogen-induced blast
transformation. Quantitative and qualitative abnormalities of T and B cell subsets have been identified in MM patients, the most important of which appears to be a decrease in the number of CD4+ helper cells, a reduction in CD4/CD8 ratios and the loss of antigen-experienced T and B cells. These immunological defects predisposes infectious complications of myeloma[3].

We report a patient who was admitted with one of the major symptoms of myeloma, persistent back pain, but of an unusual etiology, spontaneous infective discitis (SID).

**CASE REPORT**

A 58-year-old male diabetic patient, who had been treated with oral antidiabetic agents for ten years, was diagnosed as having multiple myeloma in January 2000 when he was admitted with normochromic normocytic anemia [Hb: 10.4 g/dL (12-16) MCV: 88.4 fl (81.0-99.0), MCH: 23.6 pg (27.0-31.0), MCHC: 33.5 g/dL (33-37)] (ESR > 100 mm/h), and bone marrow infiltration with abnormal plasma cells (> 30% of nucleated bone marrow cells). The peripheral blood total white cell and platelet count were 8.1 x 10^9/L (4.0-10.0) and 490 x 10^9/L (140-440), respectively. Roule-oux formation was observed in peripheral slide examination. Serum immunoglobulin electrophoresis showed a monoclonal IgGk paraprotein band [lgG: 4795 mg/dL (696-1618), kappa (k) light chain: mg/dL (200-440)] with reduced levels of normal immunoglobulin A, M and lambda light chain [131 mg/dL (68-378), 80 mg/dL (60-263) and 102 mg/dL (110-240), respectively]. The skeletal survey showed no myelomatous bone lesions at presentation. The patient was accepted as stage IIA myeloma and was started on combination chemotherapy (MP regimen consisting melphalan and prednisolone). The patient was admitted to the Hematology Clinic for close follow up a few months later due to persistent back pain and intermittent fever particularly rising at night. The patient had fever during his five day stay at hospital. The fever was intermittant, varied between 37.6°C and 38.2°C, associated with diaphoresis and chill, and generally occurred at night. The patient had no fewerish or infectious period during the myeloma treatment. Initially back pain was attributed to myelomatous deposit in the spine but it remained refractory to morphine basic analgesic. Microbiologic investigations did not show any focus of infection. No microorganisms was detected in blood and urine cultures during this follow up. Although all systems were examined in detail, no primary focus was found. In this period, the patient’s erythrocyte sedimentation rate (ESR) was 140 mm/h and bone marrow infiltration with abnormal plasma cell was < 10% of the nucleated cells. Although his CRP level was very high [CRP 124 mg/L (N < 6 mg/L)]. Over the next few weeks he continued to complain of severe lower back pain. The patient pointed out that he had no fever in this period contrary to the past. There was hypoesthesia at the left lower extremity of L3,4 dermatome and bilaterally weakness at his lower extremity. Physical examination revealed severe pain in lumbar flexion, extension and rotation. Radiographic examination of lumbar spine showed loss of height of the intervening disc and scoliosis (Figure 1). Magnetic resonance imaging (MRI) revealed low signal intensity on T1 and high signal intensity on T2 weighted images at the suspected disc level and adjacent bone marrows. Additionally there was spinal stenosis at this spinal segment (Figure 2 a-b). This appearance was diagnosed as spondylodiscitis or plasmacytoma. These clinical findings was thought to be compatible with a discitis of malignant infiltration of myeloma. Because of this, we performed computed tomography (CT)-guided needle-biopsy. Histopathologic examination at the specimen showed chronic inflammation process and the aerobic culture of the material showed S. aureus infection. The patient was prescribed cefazolin sodium (1000 mg, three times a day). Two weeks after the first visit, the patient was suffering from low-back pain, paresthesia at his feet and had difficulty standing-up. Physical examination showed hypoesthesia at his left low extremity, compatible with the L3,4-5 segments and the straight-leg raising test was bilaterally positive at 60 degrees. Patella and aschill reflexes were weak, particularly in left lower extremity. A wide debridement of intervertebral disc and the adjacent vertebral corpus were performed by anterior exposure because of advanced neurologic symptoms. Two cages, filled by autogenous iliac bone graft, were placed in the described...
area. In the postoperative period, the patient was treated by intravenous antibiotic therapy for six weeks and brace application for four months. At the end of the first year, the patient was free of any symptoms (Figure 3). His high sedimentation rate and CRP level returned to normal. Subsequently, the patient continued to receive MP therapy and is currently in remission. He remains well with no skeletal symptoms.

DISCUSSION

Difficulties arise in the differential diagnosis of severe back pain because clinical signs and laboratory findings may be unremarkable, or misleading, and major etiologic categories of severe back pain include degenerative infiltrative and infective disc diseases, metastatic or neoplastic and infective vertebral involvement.[4]

Spontaneous infective discitis—primary infection—within the disc space with or without involvement of contiguous vertebral end-plates is uncommon but a recognized cause of back pain in adults. This condition appears to be rare complaint in patients with immunosuppression; for example leukemia, cytotoxic chemotherapy, long term steroid treatment, organ transplantation and

Figure 1. X-ray examination of the lumbosacral region: Third and fourth intervertebral disc space was narrowed and end plates of the vertebral bodies were sclerotic and irregular.

Figure 2a. Sagittal T1 weighted MR images show low signal intensity of the narrowing of the third and fourth lumbar vertebral bodies. Intervertebral disc and end plates were destructed.

Figure 2b. Axial T1 weighted MR images at the level of the third and fourth intervertebral disc space revealed vacuum sign and contrast enhancement of the disc suggesting spondylodiscitis.
diabetes mellitus\[5\]. The common presenting features of SID are persistent backache and raised ESR. Gram-positive (especially S. aureus, S. epidermidis) bacterial, gram-negative bacterial, mycobacterium and fungal microorganisms may be responsible for SID\[6,7\]. MRI is more sensitive and specific in the diagnosis of discitis than other imaging methods such as X-ray of spine, technetium and gallium scan and CT\[8\]. Confirmation of the diagnosis depends on isolation of the causative organism\[9-12\]. Imaging techniques may prove helpful in the diagnostic process and facilitating needle aspiration or regional surgery SID similar to the myeloma in respect of persistent back pain, high ESR with or without fever and high CRP level. In some situations, other activation markers of myeloma, such as bone marrow infiltration with abnormal plasma cells, increased monoclonal immunoglobulin level, hypercalcemia, hyperuricemia, anemia etc. should be kept in mind because of aggravation of SID with myeloma treatment. The immune defence is compromised due to MM or administration of chemotherapeutic agents, and consequently, this results in formation of infectious foci and causes SID. If the patient’s pain and neurological symptoms persist, an extensive debridement and stabilization should be carried out\[13\].

In our patient MRI revealed a suspicious discitis. Although the patient was in remission for myeloma, his ESR and CRP levels were higher than pre-treatment levels. Moreover, fever and persistent back pain arise following myeloma therapy. Because of pain and some neurological deficit, the patient was treated by wide debridement and autogenous bone graft application without any recurrence. Both myeloma and diabetes were responsible for severe immunodeficiency in this patient.

In conclusion, detailed radiological and microbiological investigations should be performed in order to diagnose unusual cause of persistent back pain in immunocompromised patients such as diabetes, malignancies and especially multiple myeloma.

REFERENCES


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