

AN INSIGHT INTO THE COMPLEXITY OF MULTIPLE MYELOMA: CASE STUDY

General Medicine

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ABSTRACT

Multiple myeloma is a malignant disorder of plasma cells—specialized B lymphocytes responsible for producing antibodies essential to immune function. Also referred to as plasma cell myeloma, myelomatosis, or Kahler's disease, it is typically manageable but rarely curable. The condition is characterized by the clonal proliferation of abnormal plasma cells, primarily within the bone marrow. Although bone involvement is common, multiple myeloma must be distinguished from metastatic cancers originating in organs such as the breast or lung that secondarily affect the skeleton. Accounting for roughly 1% of all cancers, multiple myeloma is relatively uncommon, with an estimated incidence of 40 cases per year per million individuals. While earlier reports suggested a two- to threefold rise in incidence over the past 40 years, robust epidemiological studies from the United States and Europe indicate that this trend is largely due to improved diagnostic capabilities and greater access to healthcare, rather than a true increase in disease prevalence.

KEYWORDS

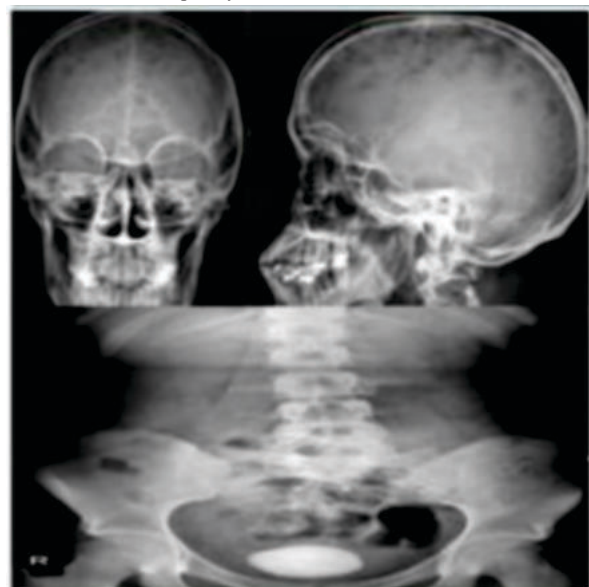
INTRODUCTION

Multiple myeloma originates from an asymptomatic, premalignant expansion of monoclonal plasma cells, which arise from post-germinal center B lymphocytes. Through a series of genetic alterations and interactions with the bone marrow microenvironment, these cells undergo malignant transformation. The disease typically develops in a stepwise fashion, beginning with monoclonal gammopathy of undetermined significance (MGUS), progressing to smoldering (asymptomatic) myeloma, and ultimately advancing to symptomatic multiple myeloma. Several genetic abnormalities within the tumor plasma cells play pivotal roles in driving this transformation and are central to the pathogenesis of the disease.

Primary early chromosomal translocations occur at the immunoglobulin switch region on chromosome 14 (q32.33), which is most commonly juxtaposed to MAF (t[14;16](q32.33;23)) and MMSET on chromosome 4p16.3.

Case Report

A 60-year-old woman presented with complaints of fatigue, unintentional weight loss, abdominal discomfort, and episodes of vomiting. Physical examination revealed marked pallor, with no other remarkable findings. Laboratory investigations demonstrated severe anemia, renal dysfunction, hypercalcemia, elevated uric acid levels, and raised inflammatory markers. Urinalysis confirmed the presence of proteinuria along with Bence Jones proteins. Imaging studies identified nephrolithiasis, osteolytic bone lesions, and findings consistent with multiple myeloma.



Observations

The patient initially presented with vesicular calculi and elevated renal function parameters. Hypercalcemia raised clinical suspicion for multiple myeloma, which was further supported by abnormal findings in both serum and urine studies. The presence of Bence Jones proteins in the urine, along with corroborative imaging and hematological results, confirmed the diagnosis. The patient was subsequently initiated on a treatment regimen consisting of thalidomide and dexamethasone.

DISCUSSION

Multiple myeloma is a malignant disorder of plasma cells that predominantly affects the bone marrow, often leading to skeletal destruction, anemia, renal impairment, and immunodeficiency. Patients commonly present with symptoms such as persistent bone pain, fatigue, recurrent infections, and signs of kidney dysfunction. The diagnosis is established through a combination of bone marrow biopsy, detection of monoclonal proteins in serum or urine (including Bence Jones proteins), and radiological evidence of osteolytic bone lesions. Advanced imaging techniques and laboratory markers further aid in staging and risk stratification.

Management of multiple myeloma has evolved significantly, with treatment strategies tailored to disease stage, patient age, and comorbidities. Standard therapeutic approaches include chemotherapy, immunomodulatory agents, proteasome inhibitors, corticosteroids, and, in eligible patients, autologous stem cell transplantation. Supportive care—such as bisphosphonates for bone disease, hydration for hypercalcemia, and erythropoiesis-stimulating agents—also plays a crucial role in improving quality of life. While multiple myeloma remains incurable, recent advances in targeted therapies and combination regimens have significantly improved survival and disease control.

CONCLUSION

This case highlights the importance of considering multiple myeloma in patients presenting with non-specific symptoms such as fatigue, weight loss, and renal impairment, especially when accompanied by hypercalcemia and proteinuria. Early recognition and prompt diagnosis using serum/urine electrophoresis, bone marrow biopsy, and imaging are critical for initiating effective treatment and preventing organ damage. The presence of Bence Jones proteins and lytic bone lesions further confirmed the diagnosis in our patient. Initiation of appropriate therapy, including immunomodulatory agents and corticosteroids, led to symptomatic improvement. This case underscores the need for heightened clinical suspicion and a multidisciplinary approach in the evaluation of unexplained systemic symptoms in older adults.

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