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(CASE REPORT)

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Severe anemia and renal impairment: A case report on multiple myeloma

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Abstract

A 51-year-old female patient presented to the emergency department with severe body aches, generalized weakness, decreased appetite, recurrent fever, weight loss, cough with sputum, and shortness of breath for one week. The patient had a history of closed reduction and internal fixation (CRIF) of the right femur two months ago. Physical examination revealed pallor, bilateral air entry positive in the lungs, and normal heart sounds. Laboratory investigations showed severe anemia (hemoglobin 7.1 g/dL), leukocytosis (WBC 20.3 x10^3/µL), thrombocytosis, elevated serum creatinine (2.11 mg/dL), mild hypoalbuminemia (2.5 g/dL), hypokalemia (2.3 mEq/L), proteinuria, and elevated serum calcium (11.2 mg/dL). Bence-Jones protein was positive, and beta-2 microglobulin was elevated at 4.0 mg/L. X-ray and MRI findings revealed multiple lytic lesions and vertebral compression fractures, suggesting skeletal involvement. CT scans confirmed rib fractures and vertebral collapse due to osteolytic activity.

Bone marrow biopsy showed hypercellular marrow with 30% plasma cells, predominantly immature and plasmablastic forms, expressing monoclonal kappa light chains. Flow cytometry confirmed the clonal nature of plasma cells, diagnosing multiple myeloma. The patient's clinical presentation and diagnostic findings were consistent with a diagnosis of multiple myeloma, characterized by anemia, renal dysfunction, hypercalcemia, lytic bone lesions, and monoclonal plasma cells.

Keywords*:* Multiple Myeloma; Plasma Cells; Bone Marrow Biopsy; Hypercalcemia; Renal Dysfunction; Lytic Bone Lesions**.**

1. Introduction

Multiple myeloma (MM) is a type of blood cancer that arises from the abnormal proliferation of plasma cells in the bone marrow, leading to excessive production of monoclonal immunoglobulins. This uncontrolled growth can result in various clinical complications, such as anemia, high calcium levels, kidney dysfunction, and bone damage. MM constitutes approximately 10% of all hematologic malignancies and accounts for roughly 1.6% of all cancers. The average age at diagnosis is around 70 years, with a higher prevalence among men and African Americans [1, 2].

The development of MM is a complex, multistep process involving genetic mutations and interactions within the bone marrow microenvironment. This progression typically follows a sequence from monoclonal gammopathy of undetermined significance (MGUS) to smoldering multiple myeloma (SMM) before advancing to symptomatic MM [3, 4].

Advancements in the molecular understanding of MM have significantly improved diagnostic methods, risk assessment, and treatment approaches. The introduction of novel therapies, including proteasome inhibitors, immunomodulatory drugs, and monoclonal antibodies, has considerably enhanced patient outcomes. Additionally, high-dose chemotherapy followed by autologous stem cell transplantation remains a fundamental treatment option, providing extended

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remission for eligible individuals [5, 6]. Despite these progressions, MM remains an incurable disease characterized by cycles of remission and relapse. Current research is focused on developing innovative treatments, refining existing therapies, and identifying prognostic biomarkers to further improve survival rates and quality of life for patients [6, 7].

Recent studies suggest that monoclonal antibodies, such as daratumumab, may slow disease progression in high-risk SMM patients. Additionally, novel drug combinations are under investigation to enhance treatment effectiveness and combat resistance [8].

The incorporation of chimeric antigen receptor (CAR) T-cell therapies into earlier treatment phases has shown promise, particularly in relapsed or treatment-resistant MM cases. Moreover, clinical trials are assessing the potential of bispecific T-cell engagers and antibody-drug conjugates to expand available treatment options [9].

Furthermore, identifying specific genetic mutations and alterations in MM patients has paved the way for personalized medicine, enabling more precise risk classification and targeted therapies [10].

2. Case report

A 51-year-old female presented to the emergency department with severe body aches persisting for one week, associated with generalized weakness, reduced appetite, intermittent fever, weight loss, cough with sputum, and shortness of breath (Grade 2 dyspnea). Her past medical history was notable for a closed reduction and internal fixation (CRIF) surgery performed two months earlier for a right femur fracture. On admission, her blood pressure was 130/90 mmHg, pulse rate was stable, and no abnormal heart or lung sounds were detected during cardiovascular and respiratory examinations. The abdominal examination was unremarkable, with a soft, non-tender abdomen. However, she showed severe pallor, although jaundice and cyanosis were absent.

Laboratory investigations revealed severe anemia with hemoglobin of 7.1 g/dL, a low red blood cell count of 2.53 million/μL, and an elevated white blood cell count of 20.3 $x10^3$ μL, indicating leukocytosis. Platelet count was mildly elevated at 285 $x10^3$/μL. Renal function tests showed serum creatinine of 2.11 mg/dL and blood urea nitrogen (BUN) of 27 mg/dL, indicating renal impairment. Biochemical studies revealed low albumin (2.5 g/dL), and hypokalemia (2.3 mEq/L). Urinalysis revealed significant proteinuria, and the Bence Jones protein test was positive, a hallmark indicator of multiple myeloma.

Further investigation of calcium metabolism showed elevated serum calcium (11.2 mg/dL), another classic finding in multiple myeloma. The beta-2 microglobulin level was significantly elevated at 4.0 mg/L (normal range: 0.7-1.8 mg/L), suggesting both high tumor burden and poor prognosis. In light of these findings, imaging studies were performed. A spinal X-ray showed multiple lytic lesions and compression fractures at L2-L3, consistent with myeloma bone disease. An MRI scan revealed extensive bone marrow involvement, with multiple focal lytic lesions across the thoracic and lumbar spine, pelvis, and ribs, confirming widespread skeletal disease. A CT scan of the abdomen and chest corroborated these findings, showing rib fractures and vertebral collapse due to osteolytic destruction.

The diagnostic gold standard, bone marrow biopsy, was performed to confirm the suspicion of multiple myeloma. The biopsy showed hypercellular marrow with reduced megakaryocytes and features of megaloblastic and normoblastic erythropoiesis. Importantly, plasma cells accounted for 30% of the cellularity, with a predominance of immature and plasmablastic forms. These cells exhibited eccentric nuclei, prominent nucleoli, and moderate cytoplasm, with bi-nucleated and multinucleated plasma cells also observed. Immunophenotyping by flow cytometry confirmed monoclonal plasma cells expressing monoclonal kappa light chains, thereby confirming multiple myeloma as the final diagnosis.

The patient's haematology report further reinforced these findings, with hemoglobin at 7.1 g/dL, red blood cell counts of 2.53 mil/μL, and total leukocyte count of 8.02 $x10^3$/μL. The packed cell volume was reduced at 22.5%, and other red cell indices (MCV 88.9 CU, MCH 26.5 pg, and MCHC 29.8%) were consistent with anemia of chronic disease. The differential count showed a predominance of polymorphs (48.4%), with lymphocytes (33.9%), monocytes (13.0%), eosinophils (4.6%), and basophils (0.1%).

The patient's treatment was tailored to manage her infection, anemia, pain, electrolyte imbalance, and suspected plasma cell disorder. On Day 1, she was initiated on intravenous ciprofloxacin (500mg BD) and metronidazole (500mg TID) for broad-spectrum infection coverage, alongside ondansetron (4mg IV BD) for nausea, and pantoprazole (40mg IV OD) for gastroprotection. Oral Telmisartan (40mg OD) was also given for blood pressure control.

On Day 2 and Day 3, diuretics were introduced with furosemide (40mg IV BD) if systolic blood pressure exceeded 110 mmHg. Antibiotic therapy continued with amoxicillin-clavulanate (1.2g IV BD). Supportive care included paracetamol (1g IV BD) for fever and pain, multivitamins (OD), and iron-folic acid with vitamin C (335 µg + 500mg OD) to address anemia. Respiratory symptoms were managed with ambroxol syrup (10ml TID). Arterial blood gas (ABG) monitoring was done every six hours.

On Day 4, prednisolone (40mg OD) was introduced, aligning with treatment protocols for multiple myeloma, particularly to reduce plasma cell proliferation. The supportive medications (pantoprazole, paracetamol, antibiotics, multivitamins, ambroxol, and iron-folic acid with vitamin C) were continued. This regimen remained unchanged on Day 5, with ondansetron (4mg IV BD) added back to address nausea. This multidisciplinary approach focused on infection control, pain relief, supportive care for anemia, and early initiation of corticosteroids, which play a critical role in anti-myeloma therapy.

3. Discussion

Multiple myeloma (MM) is a plasma cell malignancy characterized by clonal proliferation of abnormal plasma cells within the bone marrow, leading to excessive production of monoclonal immunoglobulins, which can cause extensive organ damage [1]. The clinical presentation in this case—severe anemia, renal dysfunction, hypercalcemia, and lytic bone lesions—is consistent with the classic diagnostic criteria for symptomatic multiple myeloma [2]. Anemia in multiple myeloma is multifactorial, arising from marrow infiltration by plasma cells, reduced erythropoietin production due to renal impairment, and chronic inflammation suppressing erythropoiesis [1, 5]. The severe pallor seen on examination correlates with this profound anemia, which is a hallmark finding in up to 73% of newly diagnosed cases [4].

Renal dysfunction, as seen in this patient (serum creatinine 2.11 mg/dL), is another frequent complication of multiple myeloma, occurring in approximately 50% of patients at diagnosis [2]. This results from a combination of factors, including direct tubular damage from monoclonal light chains (Bence Jones protein), hypercalcemia-induced nephrocalcinosis, and recurrent infections [7]. The presence of Bence Jones proteinuria in this case was particularly important, as this is a diagnostic hallmark of light chain myeloma and a contributor to renal tubular injury [5].

Bone involvement is another defining feature of multiple myeloma, present in up to 80% of cases at diagnosis [1]. The multiple lytic lesions, vertebral compression fractures (L2-L3), and rib fractures observed on imaging in this patient are classical for myeloma bone disease, resulting from osteoclast activation and osteoblast suppression driven by pro-inflammatory cytokines produced by myeloma cells [10]. These skeletal findings, combined with hypercalcemia (serum calcium 11.2 mg/dL), further reinforced the diagnosis and underscored the advanced nature of her disease [6].

The bone marrow biopsy, which demonstrated hypercellular marrow with 30% plasma cells (this meets the diagnostic threshold for MM ≥10% plasma cells), predominantly of immature and plasmablastic forms, confirmed the diagnosis. This high plasma cell burden, along with the monoclonal expression of kappa light chains detected by flow cytometry, fulfills the International Myeloma Working Group criteria for multiple myeloma. The elevated beta-2 microglobulin (4.0 mg/L) further indicated a high tumor burden and poor prognosis [2]. This marker is widely used in staging systems like the Revised International Staging System (R-ISS), where elevated beta-2 microglobulin, low albumin, and high lactate dehydrogenase (LDH) correlate with more aggressive disease and poorer survival [2, 4].

This case also highlights the diagnostic challenges posed by multiple myeloma, especially in patients with recent orthopedic trauma, such as the right femur fracture in this patient. Bone pain and fractures in myeloma often mimic osteoporosis or metastatic disease, delaying the diagnosis unless clinicians maintain a high index of suspicion [1]. Furthermore, the non-specific systemic symptoms—weakness, weight loss, fever, and dyspnea—could easily be misattributed to infection or post-surgical deconditioning, further complicating the clinical picture [4].

The treatment strategy for this patient focused on infection control, symptomatic relief, supportive care, and disease-modifying therapy. Empirical broad-spectrum antibiotics were started immediately due to febrile neutropenia risk common in myeloma [1]. Pain from lytic bone lesions was managed with diclofenac and paracetamol. Electrolyte imbalance (hypokalemia) was corrected, and diuretics were added to manage fluid overload caused by renal dysfunction.

Critically, corticosteroid therapy with prednisolone was started. Steroids are essential in initial myeloma management, reducing plasma cell proliferation and controlling hypercalcemia [2]. The planned next step included initiation of triplet chemotherapy (proteasome inhibitor, immunomodulator, and steroid), as current guidelines recommend this for newly

diagnosed multiple myeloma [6]. In eligible patients, such induction is followed by autologous stem cell transplantation (ASCT), known to significantly prolong progression-free survival [2].

Infection prophylaxis (antibiotics), bone health management (bisphosphonates or denosumab), renal support, and thromboprophylaxis (due to increased clot risk with immunomodulators) form critical aspects of comprehensive care [6]. This holistic management is vital because multiple myeloma remains incurable, and patients follow a relapsing-remitting disease trajectory requiring lifelong follow-up [7].

Recent advances in multiple myeloma treatment have significantly improved patient outcomes. Emerging therapies, including chimeric antigen receptor (CAR) T-cell therapy, bispecific T-cell engagers, and antibody-drug conjugates, are being integrated into earlier lines of treatment to improve response rates and prolong remission [9]. Personalized approaches based on genetic profiling of individual tumors are also gaining importance, with targeted therapies directed at specific mutations showing promising results [10].

This case highlights the importance of recognizing early warning signs of multiple myeloma, especially when constitutional symptoms coexist with bone pain, anemia, and renal dysfunction in middle-aged patients. A comprehensive diagnostic evaluation, including clinical assessment, laboratory investigations, advanced imaging, and bone marrow biopsy, is essential for early diagnosis and timely initiation of disease-directed therapy and supportive care. Although multiple myeloma remains a relapsing-remitting disease, recent therapeutic advancements, including novel immunotherapies and precision medicine, continue to improve both the quality of life and long-term outcomes for affected patients [2, 7].

4. Conclusion

This case highlights the importance of considering multiple myeloma in patients presenting with unexplained anemia, renal dysfunction, hypercalcemia, and lytic bone lesions. A thorough evaluation with laboratory tests, imaging studies, and bone marrow biopsy is crucial for an accurate diagnosis. Early recognition and appropriate treatment are essential to improve outcomes in patients with this condition.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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