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Beyond Bone Pain: Paraparesis as an Unusual Initial Manifestation of Multiple Myeloma

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Abstract: ***Multiple Myeloma (MM) is a clonal plasma cell malignancy characterized by the overproduction of monoclonal immunoglobulins and complex interactions within the bone marrow microenvironment. It can rarely manifest as neurological disease. We report case of 49 year old female presented with lower limb para paresis and Acute Kidney injury. Investigations revealed renal dysfunction, anaemia, and leucocytosis. Radiology scans revealed Right adnexal mass with lytic lesions of bone. Bone marrow biopsy and aspiration revealed plasma cell disorder. Hence the diagnosis of Multiple Myeloma with Metastatic carcinoma was made and patient was started on chemotherapy. This report highlights the atypical presentation of a common hematological malignancy and emphasizes the need for a comprehensive diagnostic approach in patients with unexplained neurological compromise.***

Keywords: Multiple Myeloma, Para paresis, Spinal cord compression, neurological manifestations, AKI

1. Introduction

Multiple myeloma, also known as plasma cell myeloma, myelomatosis, and Kahler's disease, arises from a clonal population of plasma cells. Multiple myeloma accounts for approximately 10% of all hematologic malignancies. It is a systemic disease caused by a clonal bone marrow plasma cell population. It is more common in men than women, and occurs more frequently between the ages of 65–74 years, with a median age at diagnosis of 69 years.

It is diagnosed when a clonal plasma cell percentage of ≥10% in the bone marrow is seen in conjunction with CRAB symptoms, which include the following:

- C: Hyper calcemia
- R: Renal insufficiency
- A: Anemia
- B: Osteolytic bone lesions

2. Case Report

A 49 year old lady presented with c/o lower back pain associated with difficulty moving of lower limbs, abdomen distension since 2 weeks and reduced urine output since 2 days. She had no history of fever/ rash/ oedema/ oral ulcer/ Coronary Artery Disease (CAD)/ liver disease/ significant obstetric history/ T2DM/HTN.

On physical examination her pulse rate -90 bpm, Blood pressure was 150/100 mmHg, Saturation was 90% RA. She had mild pallor. Per Abdomen was soft and distended.

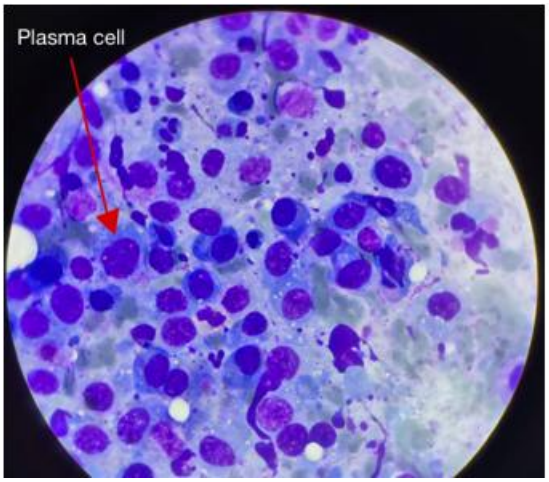
CNS: Motor examination- Lower limb

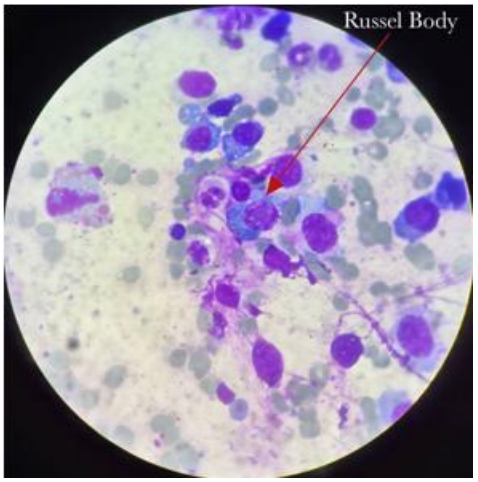
- Tone- Reduced in both lower limbs
- Power – 2/5 in both lower limbs (hips, knees, ankles and toes)
- Reflexes- 1+ (Ankle, Knee reflexes)

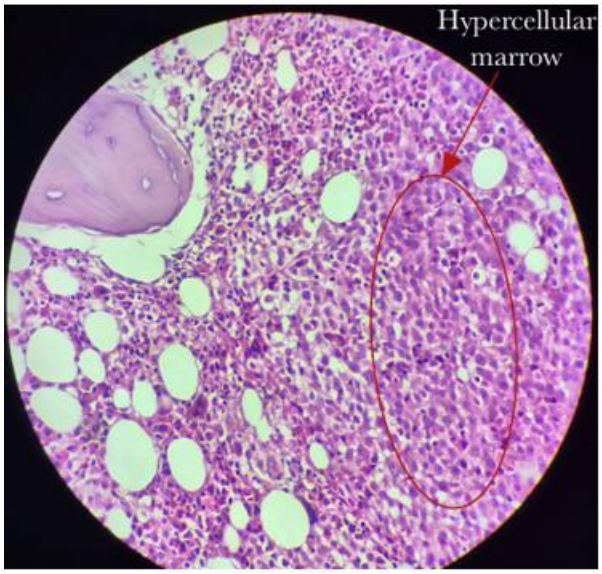
3. Lab Investigation

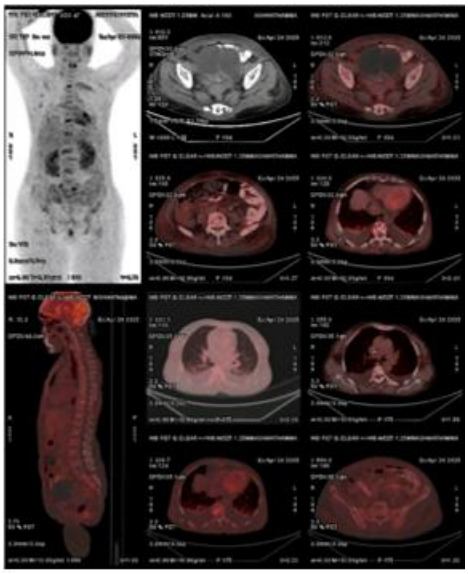
1) HB- 9.6 gm/dl
2) WBC- 24320 cells/cmm
3) Blood Urea- 105.1 mg/dL
4) Serum Creatinine – 10.07 mg/dL
5) Serum Uric acid- 10.91 mg/dL
6) Serum Potassium- 5.13 →5.4→5.8
7) Serum Albumin- 2.83 g/dl
8) Serum Calcium- 12.4 mg/dL
9) Serum Phosphorus- 7.03 mg/dL
10) CRP- 57.77mg/dl
11) Urine culture- E coli growth.
12) USG Abdomen & Pelvis- Grade II Nephropathy, Multi-locular cystic lesion of 8.1*10.3*7.1 cm in pelvis -? Ovarian, free fluid in POD.
13) MRI Pelvis Plain- Large complex cystic lesion likely arising from Right Ovary in the pelvis and extending into the mid abdomen-? Serous/Mucinous Cystadenoma/Cysteadenocarcinoma with numerous lytic lesions in bone- likely secondaries.
14) 18-FDG PET CT – Mildly hypermetabolic thick walled solid cystic mass lesion of size 10.5* 8.9 * 9.2 cm arising from right adnexa with multiple loculations and calcification of concern for neoplastic etiology.
    - Hypermetabolic multiple lytic lesions in bones of axial and appendicular skeleton with few of them showing soft tissue component.
    - Mild ascites in abdomen and pelvis and Mild bilateral pleural effusion.
15) Serum Electrophoresis: No M band observed. Hypoalbuminemia. Elevated peak at alpha-1 region.
16) Ca-125 - 71.74 U/ml
17) Skull X Ray: Punched out lesions.
18) Bone marrow aspiration: 72% Mature and immature plasma cells with prominent nucleoli and bi nucleation.
19) Bone marrow Biopsy: Hyper cellular marrow for age showing foci of trilineage hematopoiesis. Diffuse interstitial infiltrate of mature and immature plasma cells occupying about 80% of the marrow.

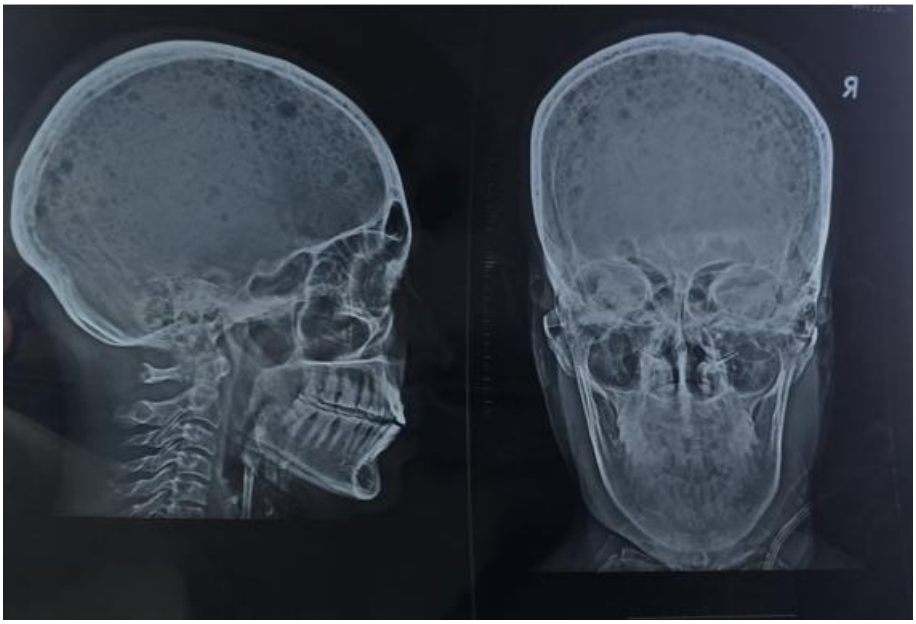
20) Urinary Bence Jones Proteins: Positive.











Hence a provisional diagnosis of Multiple myeloma was done. Bone marrow aspiration showed Plasma cell myeloma. Patient was started on Inj Bortezomib, Denosumab, Cyclophosphamide and IV corticosteroids. Patient was stable at discharge.

4. Discussion

Plasma cell disorders are monoclonal neoplasms related to each other by virtue of their development from common progenitors in the late B-lymphocyte lineage. Multiple Myeloma represents a malignant proliferation of plasma cells derived from a single clone. The tumor, its products, and the host response to it result in a number of organ dysfunctions and symptoms, including bone pain or fracture, renal failure, susceptibility to infection, anemia, hypercalcemia, and occasionally clotting abnormalities, neurologic symptoms, and manifestations of hyperviscosity.

Bone pain is the most common symptom in myeloma, affecting nearly 70% of patients. The bony lysis results in substantial mobilization of calcium from bone, causing hypercalcemia. Localized bone lesions may cause the collapse of vertebrae, leading to spinal cord compression causing paraparesis.
The next most common clinical problem in patients with myeloma is susceptibility to bacterial infections. These patients have very poor antibody responses, especially to polysaccharide antigens such as those on bacterial cell walls. UTI, especially Escherichia coli will be isolated in urinary tract.

Renal failure occurs in nearly 25% of myeloma patients, and some renal pathology is noted in >50%. Myeloma patients are susceptible to developing acute renal failure if they become dehydrated.

Many of the clinical features of myeloma, e.g., cord compression, pathologic fractures, hyperviscosity, sepsis, and hypercalcemia, can present as medical emergencies.

5. Conclusion

Multiple myeloma, though diverse in its clinical manifestations, rarely presents acutely with neurological emergencies such as para paresis as the initial symptom. This case report highlights a crucial example of such an atypical presentation, where a patient's progressive lower limb weakness and sensory deficits were ultimately traced back to an underlying multiple myeloma causing spinal cord compression. The delay in diagnosis, primarily due to the initial focus on more common neurological etiologies, underscores the importance of considering hematological malignancies in the differential diagnosis.

This case serves as a valuable reminder for clinicians to maintain a high index of suspicion for multiple myeloma in atypical presentations, advocating for a broader diagnostic workup when faced with neurological symptoms of unclear origin. Early recognition and prompt initiation of appropriate systemic therapy, coupled with supportive measures, are paramount in improving patient outcomes and preventing irreversible neurological damage in such emergent scenarios.

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