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Multiple Myeloma Presenting as Bilateral Foot Drop: A Diagnostic Dilemma

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Abstract

Peripheral neuropathy is one of the commonest presentations seen in the spinal cord or vertebral column disorder. Injury to the spinal cord, degenerative pathologies, osteomyelitis, malignancies, genetic disorders, viral infections, and vitamin deficiencies can cause peripheral neuropathy secondary to spinal cord and vertebral column involvement.

Keywords: Multiple Myeloma; Bilateral Foot Drop; Diagnostic

Introduction

Peripheral neuropathy is associated with monoclonal gammopathies like multiple myeloma (MM), monoclonal gammopathy of undetermined significance (MGUS), and Waldenström macroglobulinemia (WM); which are difficult to diagnose [1]. Multiple myeloma is one of the most common monoclonal gammopathy and the prevalence of peripheral neuropathy in MM varies between 7% to 13% [1-3]. Different pathophysiology of peripheral neuropathy in MM has been described; deposition of the immunoglobulins in the in the nerve cells leading to axonal damage and demyelination [4] increased M protein level causes hyperviscosity of the blood which slows down blood supply, causing neurological symptoms [5]. Usually patients with peripheral neuropathy in MM presents with symmetric, distal sensory or sensorimotor neuropathy. We present a rare case of peripheral neuropathy in MM and its management.

Case Report

A 50 year old male patient was admitted with complaints of weakness of the lower limbs since past 2 months. The weakness in lower limb was gradual in onset and progressive till date and associated with tingling sensation in both upper limbs. Also, he had significant loss of weight in past few months with loss of appetite. He gave no history of pain and swelling in any part of the body. His vitals were normal, pulse rate was 78/min; blood pressure was 126/80mmHg; respiratory rate was 20/min; temperature was 98.8 F. No apparent swelling or list/deformity was visible in the back. Bony tenderness was absent in the vertebral column. His range of motion of the vertebral column was within normal

limits. Special test like SLR and figure of four were within normal limits. His higher motor functions and cranial nerves were normal. There was weakness in all the extremities, more in the lower limbs than the upper limbs. His distal group of muscles were weaker than the proximal muscle groups. He had loss of sensation to pinprick, heat/cold and touch on the feet and legs till the knees and on the hands bilaterally. Position and vibration sense were lost in the toes and diminished in the fingers. The ankle reflex was absent bilaterally while rest deep tendon reflexes were normal. The superficial plantar reflex was cremasteric response was normal. No cerebellar signs were detected. Radiograph of vertebral column showed normal bone density and alignment. Spine screening showed lytic changes in left half of the S1-S3 vertebrae suggestive of metastasis, no evidence of pathological fracture, canal or neuroforaminal stenosis.

Laboratory investigations: Haemoglobin was 17.9g/dl; leukocyte count was 9200/cumm; Platelets counts was elevated 564000/cumm; Erythrocyte sedimentation rate was 11 mm/hour. Serum calcium was 10.1 mg/dl, serum phosphorus was 5.4 mg/dl. Total serum proteins were 6.7g/dl; serum albumin 3.58g/dl; globulins-Alpha 1- 0.25, Alpha 2- 0.74, Beta 1- 0.36, Beta 2-0.25, Gamma-1.51; Albumin/globulin ratio 11.15 with a monoclonal band seen in Gamma region pattern (Figure 1).

PET-scan showed hypermetabolic lytic lesion in left sacral ala from L2 to L3 vertebral bodies with soft tissue component extending into sacral spinal canal with few small nodules in left lower lobe

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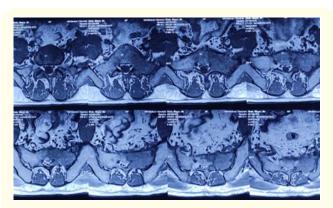


Figure 1: Lytic lesion in left half of of S1 to S3 vertebral bodies and left sacral ala

of lung (Figure 2). After the non-invasive investigations biopsy was taken from the sacral ala with J needle in all aseptic precautions. It showed increase in the atypical plasma cells, suggestive of Multiple Myeloma.

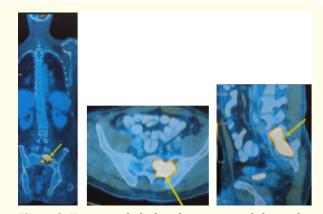


Figure 2: Hypermetabolic lytic lesion seen in left sacral ala from L1 to L3 vertebral bodies.

The patient was started on chemotherapy, 9 months post operatively had significant improvement in gait and motor recovery.

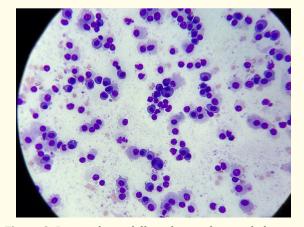


Figure 3: Biopsy shows diffuse sheets of atypical plasma cells s/o Multiple myeloma.

Discussion

The presented case report outlines a remarkable instance of multiple myeloma (MM) presenting with bilateral foot drop, a relatively rare neurological manifestation. This discussion delves into the clinical implications, possible underlying mechanisms, and the approach to conservative management in this unique scenario.

Clinical implications

The occurrence of bilateral foot drop as the initial presentation of MM is an uncommon manifestation, posing diagnostic challenges for clinicians. The classic symptoms of MM include bone pain, anemia, renal dysfunction, and hypercalcemia. However, neurological complications like peripheral neuropathies and spinal cord compression can also arise due to direct infiltration of the nervous system by malignant plasma cells or the deposition of amyloid proteins. This case underscores the importance of considering MM in the differential diagnosis of patients presenting with neurological deficits, even in the absence of overt bone pain or typical haematological abnormalities.

Possible underlying mechanisms

The aetiology of bilateral foot drop in MM can be multifactorial. The infiltration of malignant plasma cells into nerves or nerve roots might lead to nerve compression and subsequent motor deficits. Additionally, the presence of amyloid deposits within the nerves can cause disruption of normal nerve conduction. The production of inflammatory cytokines by malignant plasma cells could also contribute to nerve damage and dysfunction. Understanding these underlying mechanisms is vital for effective management and targeted interventions.

Conservative management

In this case, a conservative management approach was adopted, which included close monitoring, physiotherapy, and pain management. The decision for conservative management was likely influenced by the patient's overall clinical condition, the extent of neurological deficits, and the aggressiveness of the underlying MM. Conservative management aims to alleviate symptoms, improve functional status, and enhance the patient's quality of life.

Physiotherapy plays a pivotal role in restoring muscle strength, enhancing mobility, and promoting neurological recovery. It often involves exercises to improve muscle tone, gait training, and functional adaptations to assist with daily activities. Pain management strategies, including non-steroidal anti-inflammatory drugs (NSAIDs) or mild analgesics, can contribute to the patient's comfort and participation in rehabilitation.

Limitations and Future Considerations

While conservative management can be effective in alleviating symptoms and improving functionality, the long-term outcomes in cases of MM-associated neurological deficits are variable. The potential progression of the underlying disease and the risk of further neurological deterioration underscore the need for continuous monitoring and timely adjustments in the treatment plan.

Future research could focus on elucidating the underlying molecular and cellular mechanisms contributing to neurological complications in MM. Additionally, the exploration of novel therapeutic agents that specifically target nerve infiltration by malignant plasma cells or amyloid deposition could hold promise for improving outcomes in cases of MM-related neurological deficits.

Conclusion

The presented case of MM presenting with bilateral foot drop demonstrates the diverse clinical spectrum of this complex haematological malignancy. The utilization of a conservative management approach involving physiotherapy and pain management highlights the need for tailored interventions in such atypical cases. Further research and collaborative efforts among haematologists, neurologists, and other specialists are essential to enhance our understanding of MM-associated neurological complications and to refine treatment strategies for optimal patient outcomes.

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