RJCCN CASE REPORT
KIDNEY DISEASE

Plasmacytoma in Membranoproliferative Glomerulonephritis: A Case Report

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Multiple myeloma is a prevalent disease, whereas bone plasmacytoma is a localized neoplasm of monoclonal plasma cells, constituting a distinct plasma cell disorder that falls between monoclonal gammopathy of undetermined significance (MGUS) and multiple myeloma (MM). We present a 63-year-old woman who exhibited proteinuria and acute kidney injury (AKI), subsequently diagnosed with membranoproliferative glomerulonephritis (MPGN) of unknown etiology. One year later, the patient developed a lytic bone lesion and diagnosed as having a solitary bone plasmacytoma. MPGN may be caused by immunological complexes or associated with monoclonal gammopathy; therefore, it is essential to assess individuals with MPGN to identify any typical or atypical etiologies of the condition.

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INTRODUCTION

Multiple myeloma (MM) is a common malignancy, constituting almost 1% of all malignancies and 10% of all hematologic cancers, with more cases occurring in men. The major clinical manifestations are the osteolytic bone lesions, anemia, hypercalcemia, renal failure, and an increased risk of infection. Extramedullary disease (EMD) is reported in 1 to 2% of patients at the time of initial diagnosis, increasing to 8% upon disease progression.[1]

Plasmacytoma, a localized neoplasm, is diagnosed based on the fulfillment of four specific criteria: Biopsy-confirmed clonal plasma cells in an isolated lesion of bone or soft tissue, no sign of clonal plasma cells in the bone marrow standard skeletal examination of the spine and pelvis, lack of end-organ damage, renal failure, anemia, or bone lesions. Plasma cell disorders, including plasmacytomas, may lead to kidney failure (Ig); the mechanisms of renal damage in plasma cell malignancies may be either immunoglobulin-dependent or immunoglobulin-independent.[2,3]

Membranoproliferative glomerulonephritis (MPGN) can occur in the context of plasma cell neoplasms such as plasmacytoma or MM or as a paraneoplastic glomerulopathy due to deposition of monoclonal immunoglobulins or light chains. These monoclonal proteins can also act as autoantibodies or form immune complexes that deposit in the glomeruli, activating complement pathways and causing the characteristic MPGN pattern.[4]

MPGN associated with monoclonal gammopathies is considered a component of the spectrum of monoclonal gammopathy of renal significance (MGRS), wherein the kidney damage is solely attributable to the monoclonal protein, in the absence of overt MM.

CASE PRESENTATION

A 63 years old woman was admitted to

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Hasheminejad kidney center due to peripheral edema and elevated serum creatinine. She had a recent history of non-steroidal anti-inflammatory drugs (NSAID) usage due to low back pain for two weeks before admission. On admission the serum creatinine level was 3.2 mg/dL (increased to 5 mg/dL), and she had proteinuria (5900 mg/24h) and microscopic hematuria. The result of Anti-nuclear antibody (ANA), Anti-double stranded DNA (Anti-ds DNA), C3, C4, and CH50 tests were all normal. A kidney ultrasound showed 137 mm for the right kidney and 147 mm for the left kidney, with normal parenchymal thickness. Serum and urine protein electrophoresis immunofixation was ordered.

The patient underwent five sessions of hemodialysis and a kidney biopsy was done, which revealed mostly enlarged and hypercellular glomeruli with increased mesangial cellularity and endocapillary proliferation making lobular accentuation; two glomeruli showed fibro-cellular crescents. Tubular atrophy and interstitial fibrosis were about 5%. Immunofluorescence study showed 3+ IgG, 3+ C3, 2+ C1q, and 1+ Kappa and Lambda deposition.

The diagnosis of type I MPGN was made according to the pathologic findings. Treatment was initiated with five sessions of plasmapheresis, three pulses of 500 mg methylprednisolone, and 500 mg cyclophosphamide; after which the serum creatinine level reduced to 1.7mg/dL. The patient was administered two grams of mycophenolate mofetil (MMF) daily as maintenance therapy. After two months serum creatinine was 1.18 mg/dL and the urine protein was 750 mg/24h. Re-check of serum and urine immunofixation and electrophoresis were normal.

One year following the first admission the patient was readmitted due to recurrence of low back pain. A lumbar MRI revealed a 35 to 37 mm solid intraosseous mass lesion in the posterior aspect of the ninth thoracic vertebra necessitating surgical excision (Figure 1); the pathology indicated plasmacytoma characterized by CD 138 positive cells (Figure 2). Bone marrow aspiration and biopsy showed mildly hypercellular marrow (45 to 50% cellularity) with less than 1% plasma cells (Figure 3).

According to the pathology report of bone plasmacytoma a local radiotherapy was considered in conjunction with lenalidomide, while prednisolone and MMF were tapered and ultimately discontinued gradually over two months; serum creatinine was 0.7 mg/dL and 24h urine protein was 123 mg.

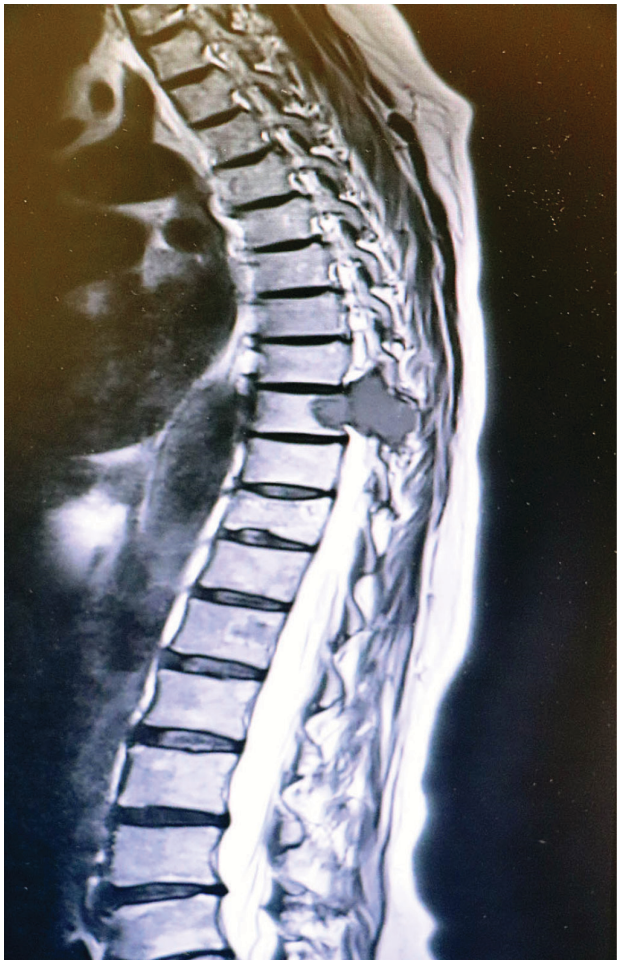

Figure 1. Thoracic MRI shows a mass lesion in posterior aspect of ninth thoracic vertebra.

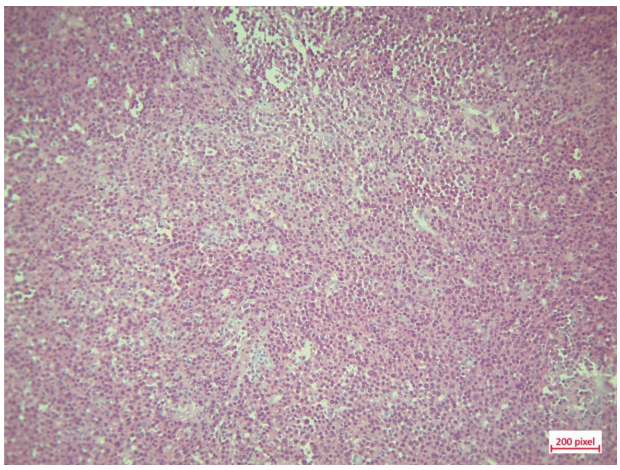

Figure 2. Plasmacytoma of Thoracic Lesion.

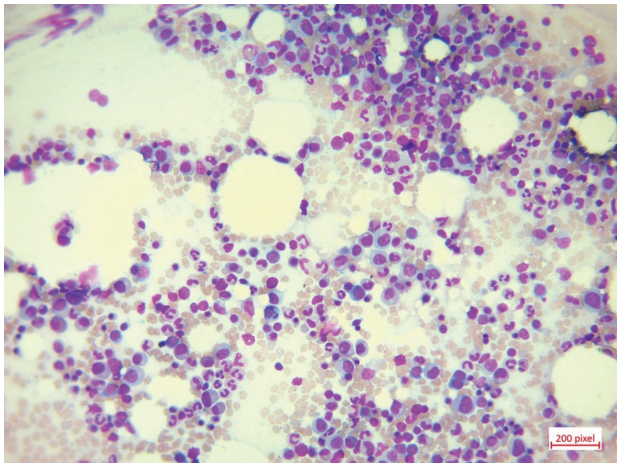

Figure 3. Bone marrow biopsy shows less than 1% plasma cells.

DISCUSSION

Bone plasmacytoma is a localized neoplasm of monoclonal plasma cells that typically manifesting as a solitary mass in bone, predominantly in the vertebrae or other skeletal sites. Bone marrow infiltration comprises fewer than 10% clonal plasma cells. This tumor can induce local bone destruction, lytic lesions, and pathological fractures. The risk of progression of bone plasmacytoma to multiple myeloma is high, with studies suggesting progression rates up to 60 to 85% over 10 years, particularly when clonal plasma cells are detected in the bone marrow or when abnormal serum kappa to lambda free light chain ratios are present. Localized radiotherapy is the mainstay of treatment, sometimes supplemented by surgery, with careful long-term monitoring due to risk of systemic progression.[5-7]

MPGN is a pattern of glomerular injury classified into immune complex-mediated and complement-mediated types and can be associated with infections, autoimmune disorders, or monoclonal gammopathies. In the context of plasma cell disorders, monoclonal gammopathy can drive complement activation or immune complex deposition that leads to MPGN-type injury in the kidneys.[8,9] Early detection and management of plasmacytoma are crucial to prevent progression to multiple myeloma and related complications.

CONCLUSION

Solitary bone plasmacytoma and solitary extramedullary plasmacytoma represent rare subgroup of plasma cell dyscrasias. In our case bone plasmacytoma was diagnosed a year after clinical presentation of MPGN however, a potential causal relationship between plasmacytoma and MPGN may exist. Therefore, when diagnosing MPGN in a patient, it is imperative to investigate not only the common etiologies but also the rarer ones.

CONFLICT OF INTEREST

Atefeh Amouzegar is a member of the editorial team of RJCCN. The author had no involvement in the peer-review or editorial decision-making process for this manuscript.

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