Case Report

A case of multiple myeloma presenting with rapidly progressive glomerulonephritis

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Introduction
Multiple myeloma (MM) is a haematological malignancy characterized by uncontrolled proliferation of plasma cells in the bone marrow (BM) producing a monoclonal immunoglobulin. The hallmark clinical manifestations include hypercalcaemia, anaemia, bone lesions and renal involvement [1].

Renal insufficiency in MM is commonly due to cast nephropathy, monoclonal immunoglobulin deposition disease and amyloidosis [2]. Rapidly progressive glomerulonephritis (RPGN) is a rare renal manifestation of MM with only a few cases being reported [3,4]

Case presentation
A 70-year-old male with hypertension was admitted with progressively worsening shortness of breath for five days with gross haematuria and oliguria for two days. There was no orthopnoea. He had accompanying bilateral ankle oedema for one week with nausea, vomiting and intermittent hiccups. He had concomitant backache with nocturnal pain for five months with significant low grade evening pyrexia, loss of appetite and loss of weight which had not been evaluated.

On examination, he was moderately pale and dyspnoeic with saturation being 92% on air. Bilateral pitting ankle oedema was apparent up to knee joint level. Blood pressure was 180/100mmHg. Fine end inspiratory crepitations were heard over bilateral basal zones of lungs. There was no organomegaly. Fundi revealed grade 2 hypertensive changes.
His haemoglobin was 7.4g/dL while mean corpuscular volume was 112fl (79 - 97 fl). The white blood cell count was 8× 10⁹/L while the platelet count was 112× 10⁹/L. Blood picture revealed normochromic, normocytic cells and marked rouleaux formation. Serum creatinine rose from 284µmol/L to 1043µmol/L over 3 days. Erythrocyte sedimentation rate was 114mm/1ˢᵗ hr. Urine full report revealed 1+ proteinuria with moderately field full RBCs. Urinary dysmorphic red cells were 80%. Urine protein creatinine ratio was 340mg/g.

Reversal of the albumin:globulin ratio was apparent with albumin 2.2g/dL and globulin 5.8g/dL. Ultrasound abdomen revealed bilateral acute renal parenchymal injury. C3 level was low with a value of 70.2 mg/dL (80-178mg/dL). C4 level was normal with a value of 15.6mg/dL (12-24mg/dL). Antinuclear antibody, antineutrophil cytoplasmic antibody screening, hepatitis serology and cryoglobulin assay were negative. Since he had clinical evidence of RPGN, a renal biopsy was done which revealed interstitial damage with 65% plasma cells. Crescents were observed in four glomeruli. No necrotizing lesions were seen.

Skeletal survey revealed multiple lytic lesions in the skull. Serum protein electrophoresis showed a monoclonal gammopathy with a paraprotein concentration of 37.06g/L (<10g/L). BM biopsy revealed 40% plasma cells in favour of MM.

He was haemodialysed daily while being investigated. Unfortunately, he passed away while awaiting further treatment from the oncology team.

**Discussion**

MM is a monoclonal paraproteinaemic disorder with plasma cell dyscrasia leading to significant specific end organ damage, with renal insufficiency being a common manifestation [5]. Other typical characteristics are anaemia, hypercalcaemia, pathological bone fractures and immunoparesis leading to infections.

Cast nephropathy leading to tubular damage and obstruction is the most common cause of renal failure in patients with MM. Amyloidosis, light chain or heavy chain deposition disease, plasma cell infiltration and interstitial nephritis are also recognized causes of renal insufficiency in MM. Concurrent hypercalcaemia, hyperviscosity and prolonged use of non-steroidal anti-inflammatory drugs for backache are other well-known causes [2,3,5]. RPGN is characterized by features of nephritic syndrome and rapid loss of kidney function over a short period of time, usually within a few weeks to months. Kidney biopsy typically reveals necrotizing and crescentic glomerulonephritis [6]. RPGN is a rare renal manifestation of MM with only a few cases being reported in the literature. Renal involvement with RPGN is a medical emergency which requires prompt intervention. Hence knowledge of the possibility of RPGN as the first manifestation of MM is important [7,8].
The aetiopathogenesis of RPGN in MM is largely obscure with few hypotheses being postulated. One such hypothesis is that in certain circumstances glomerular deposits of immunoglobulins are able to elicit some local leukocyte or macrophage mediated inflammatory reaction leading to proliferation which would result in crescent formation [7,8]. The rare occurrence of RPGN in MM is attributed to the suppression of the synthesis of immunoglobulins other than the M protein that commonly occurs in this disorder [9]. A good response to chemotherapy has also been observed in the literature [9]. Hence, awareness of the possibility of concurrence of these two clinical entities is important.

References

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