


## Case Report

# A case of multiple myeloma presenting with rapidly progressive glomerulonephritis

**Kithmini Dinushi Ellepola, Inoka Kumudini Jayasinghe**

National Hospital, Kandy, Sri Lanka

**Key words: multiple myeloma, RPGN, renal involvement**

Corresponding Author: Kithmini Dinushi Ellepola, E-mail:< kithminiel@gmail.com>  <https://orcid.org/0000-0002-7800-8710>  
Received: 17 May 2021, Accepted: revised version 06 Sep 2021, Published: 15 Sep 2021  
Competing Interests: Authors have declared that no competing interests exist

© *Author*. This is an open-access article distributed under a Creative Commons Attribution-Share Alike 4.0 International License (CC BY-SA 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are attributed and materials are shared under the same license.



## 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  $8 \times 10^9/L$  while the platelet count was  $112 \times 10^9/L$ . Blood picture revealed normochromic, normocytic cells and marked rouleaux formation. Serum creatinine rose from 284 $\mu$ mol/L to 1043 $\mu$ mol/L over 3 days. Erythrocyte sedimentation rate was 114mm/1<sup>st</sup> 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

1. Fairfield H, Falank C, Avery L, Reagan MR. Multiple myeloma in the marrow: pathogenesis and treatments. *Ann N Y Acad Sci.* 2016 Jan;1364(1):32-51. <https://doi.org/10.1111/nyas.13038>
2. Dimopoulos MA, Kastiris E, Rosinol L, Bladé J, Ludwig H. Pathogenesis and treatment of renal failure in multiple myeloma. *Leukemia.* 2008 Aug;22(8):1485-93. <https://doi.org/10.1038/leu.2008.131>
3. Heher EC, Rennke HG, Laubach JP, Richardson PG. Kidney disease and multiple myeloma. *Clin J Am Soc Nephrol.* 2013 Nov;8(11):2007-17. <https://doi.org/10.2215/CJN.12231212>
4. Stephen M. Korbet and Melvin M. Schwartz. Multiple myeloma. *Journal of the American Society of nephrology.* 2006 Sept 17 (9) 2533-2545 <https://doi.org/10.1681/ASN.2006020139>
5. Al-Farsi K. Multiple myeloma: an update. *Oman Med J.* 2013 Jan;28(1):3-11 <https://doi.org/10.5001/omj.2013.02>
6. Arimura Y, Muso E, Fujimoto S, Hasegawa M, Kaname S, Usui J, Ihara T, Kobayashi M, Itabashi M, Kitagawa K, Hirahashi J, Kimura K, Matsuo S. Evidence-based clinical practice guidelines for rapidly progressive glomerulonephritis 2014. *Clin Exp Nephrol.* 2016 Jun;20(3):322-41. <https://doi.org/10.1007/s10157-015-1218-8>
7. Meyrier A, Simon P, Mignon F, Striker L, Ramée MP. Rapidly progressive ('crescentic') glomerulonephritis and monoclonal gammopathies. *Nephron.* 1984;38(3):156-62. <https://doi.org/10.1159/000183299>
8. Sirsat RA, Deshpande RB. Multiple myeloma presenting as proliferative (crescentic) glomerulonephritis. *J Postgrad Med.* 1994 Apr-Jun;40(2):92-3. PMID: 8737563.
9. Moscoso-Solorzano GT, Madureira-Silva MV, Balda C, Franco MF, Mastroianni-Kirsztajn G. Crescentic glomerulonephritis in IgA multiple myeloma: a case report. *Nephron Extra.* 2011 Jan;1(1):69-72. <https://doi.org/10.1159/000331217>