International Journal of Research in Medical Sciences
Barysenka T et al. Int J Res Med Sci. 2025 Dec;13(12):5498-5501
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20253985

Case Report

A clinical case of multiple myeloma complicated by renal amyloidosis

Tatsiana Barysenka, R. B. S. W. M. Pasan M. Kokwewa*, M. M. Shalini P. Gawarammana

Department of Internal Diseases, Grodno State Medical University, Grodno, Belarus

Received: 19 October 2025
Accepted: 17 November 2025

***Correspondence:**
Dr. R. B. S. W. M. Pasan M. Kokwewa,
E-mail: pasanpasankokwewa@gmail.com

ABSTRACT

Multiple myeloma is a hematological malignancy effecting multiple organ systems. One of the commonly effected organs are the kidneys leading to renal amyloidosis and nephrotic syndrome. This article describes a clinical case of a patient presenting with end-stage renal failure. It mainly discusses the investigations used to come to a clinical diagnosis of multiple myeloma and renal amyloidosis and the treatment strategies used in managing the patient.

Keywords: Multiple myeloma, Renal amyloidosis, End-stage chronic kidney disease, Nephrobiopsy, ELISA analysis Beta-2 microglobulin

INTRODUCTION

Multiple myeloma (MM) can be defined as a hematological disease which primarily causes proliferation of malignant plasma cells which later manifest as anemia, renal disease, bone disease and hypercalcemia.[1] While the exact cause of MM is unknown, it's associated with translocations and frequent alterations in the promoter genes, mainly chromosome 14. Other oncogenes such as KRAS, BRAF and NRAS are also associated with plasma cell proliferation.[2] Other contributing factors such as alcohol consumption, obesity, insecticides, organic solvents and radiation exposure plays a role in disease development.[3] Paraprotein secretion, bone marrow involvement and osteoclast activation cause end organ damage, mainly renal dysfunction, pancytopenia and bone fractures.[2]

Around 12-15% of MM patients eventually develop amyloidosis over the years while about 30% have subclinical amyloid deposits.[5] Amyloid deposits in the extracellular matrix of blood vessels disrupts normal function leading to ischemic changes and apoptosis of cells. This is further aggerated by the physical obstruction by amyloid deposits. The blood vessels, particularly the endothelium, are often affected first leading to microcirculatory issues.[7]

Kidney diseases associated with MM include cast nephropathy, AL amyloidosis, monoclonal immunoglobulin deposition disease, cryoglobulinemic glomerulonephritis and proliferative glomerulonephritis. Amyloid nephropathy typically presents with asymptomatic proteinuria, nephrotic syndrome, and either acute or chronic renal failure.[4] AL amyloidosis frequently impacts the glomerulus, and many patients with AL renal amyloidosis present with significant proteinuria, with over 65% experiencing nephrotic syndrome.[6]

CASE REPORT

A 45-year-old female was presented to the Grodno University Clinic with swelling of the lower extremities, swelling of the face, periodic increases in blood pressure, palpitations, headache, sweating, shortness of breath during exercise, feeling of nausea. She considers herself sick for a year, when swelling of the legs and feet first appeared, she was not examined by doctor, and did not seek medical help. She also notes a periodic increase in blood pressure up to 140/90 mmHg; in this case, she took captopril 25 mg sublingually. The condition worsened in the morning, when blood pressure first increased to 190/110 mmHg., swelling of the lower extremities increased. She decided to visit a therapist. She worked at a chemical plant for 10 years. In 2020, suffered from

COVID-19. There were no significant family history, allergic history and past medical history.

Laboratory and instrumental investigations are listed in Table 1.

Daily proteinuria

Daily proteinuria amount 1.7; protein 4.59 g/l.

Daily monitoring of blood pressure

The average indicators of BP were 150/90 mmHg during the day and 115/70 mmHg at night. Maximum systolic and diastolic BP 175/100 mmHg according to the patient's diary (fast walking around the hospital).

Ultrasound of the heart

Echo-signs of the atherosclerotic damage to the aorta. Hypertrophy of the myocardium LV. The insufficiency of the mitral valve with the regurgitation of the 1st degree. The insufficiency of the tricuspid valve with regurgitation of the 1st degree.

Ultrasound of brachiocephalous arteries

Atherosclerotic plaques with stenosis of 20-30%.

Holter monitoring ECG

Single ventricular extrasystoles and single supraventricular extrasystoles 35/55, respectively. The average heart rate during the study 76 beats per minute in the daytime and 55 beats per min at night. Min heart rate 49 beats per minute at night. Max heart rate 125 beats per minute according to the patient's diary (fast walking around the hospital).

Ultrasound of the kidney

Kidneys with smooth contours, mobility during breathing is limited. The size of the right kidney is normal, the thickness of the parenchyma is 17-18 mm, the echogenicity of the cortical layer is increased. There is no urostasis. The size of the left kidney is normal, the thickness of the parenchyma is 17-18 mm, the echogenicity of the cortical layer is increased. There is no urostasis. There is a moderate amount of free fluid in the abdominal cavity (ascites).

Ultrasound of the abdominal cavity, pleural cavities

In the abdominal cavity there is a significant amount of free fluid (ascites) 600-700 ml. In the pleural cavity on the right there is free fluid with a layer thickness of 70-80 mm (V-1000 ml), on the left there is free fluid with a layer thickness of 75-80 mm (V-1000 ml).

Table 1: Laboratory investigations.

Investigations	Results
Complete blood count	
WBC	4.47×10^{9}/l
RBC	3.5×10^{12}/l
MCV	88.9
MCH	28.6
Hemoglobin	98 g/l
Color Index	0.87
Hematocrit	31[1]%
PLT	422×10^{9}/l
Basophils	1%
Eosinophils	4%
Neutrophils band	5%
Neutrophils segmented	61%
Lymphocytes	18%
Monocytes	3%
ESR	62 mm/hour
Urine analysis	
Sspecific gravity	1.01
Leukocytes	17
Protein	3.6 g/l
Hyaline casts	2-3
Color	Yellow
Transparency	Transparent
Coagulogram	
Activated partial thromboplastin clotting time, sec	24.9
Prothrombin time, sec	10.4
International normalised ratio	0.96
Fibrinogen, g/l	3.43
Biochemical blood test	
Total protein, g/l	41
Albumin, g/l	16
Urea, mmol/l	11.5
Creatinine, µmol/l	532
Uric acid, µmol/l	270
Total cholesterol, mmol/l	6.8
Low-density lipoproteins, mmol/l	3.12
Very low-density lipoproteins, mmol/l	0.83
High-density lipoproteins, mmol/l	1.26
Triglycerides, mmol/l	1.4
Atherogenic coefficient	2.8
Total bilirubin, µmol/l	11.9
Glucose, mmol/l	4.7
Aspartate aminotransferase, units/l	35
Alanine aminotransferase, units/l	37
Calcium, mmol/l	2.7
Potassium, mmol/l	4.3
Sodium, mmol/l	138
Chloride, mmol/l	106
Iron, µmol/l	8,2
CRP, mg/l	0,7

GFR 10 ml/min (according to the SKD-EPI formula)

X-ray of the chest

No inflammatory changes were detected. Small bilateral hydrothorax.

Nephrobiopsy

Morphological examination of the biopsy specimen after staining it with Congo red in polarized light revealed a green glow, characteristic of renal amyloidosis.

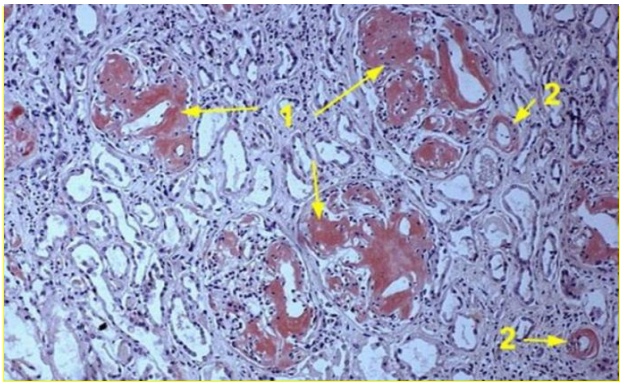

Figure 1: Biopsy specimen after staining it with Congo red in polarized light.

ELISA analysis Beta-2 microglobulin

ELISA analysis beta-2 microglobulin was found to be 6,87 mg/l (N less 2,4).

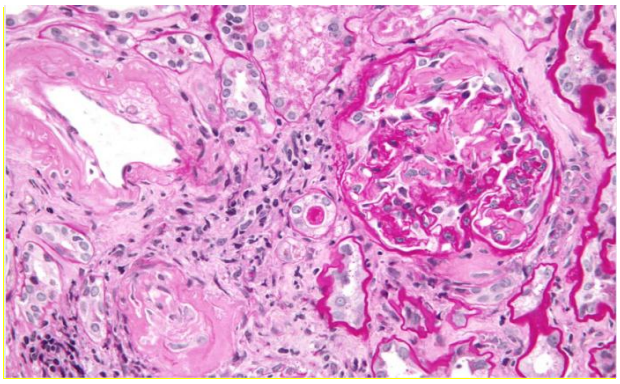

Figure 2: Positive staining of kidney tissue with Congo red stain.

Sternal punctate

Bone marrow is rich in cellular elements. The number of plasma cells increased to 29.3%. Immunophenotyping of this bone marrow sample revealed a population of 20.4% monoclonal plasma cells.

Detection of monoclonal light chains in urine using urine protein electrophoresis with immunofixation

A paraprotein consisting of kappa/lambda light chains was detected.

M-gradient, typing. Serum electrophoresis and immunofixation with a panel of antisera (IgG/A/M/kappa/lambda) with quantitative assessment of the M-gradient

Ig G higher than 30; Ig A higher than 10.

Diagnosis

Multiple myeloma stage 3B, diffuse focal form. Renal amyloidosis (histologically verified). Myeloma nephropathy. Nephrotic syndrome. CKD C5. Chronic programmed hemodialysis. Formation of A-V fistula. Symptomatic hypertension.

The diagnosis was confirmed by calcium higher 2,6, renal dysfunction (creatinine 324 µmol/l), anemia (hemoglobin 100 g/l), nephrobiopsy (AL-amyloidosis), sternal punctate (Bone marrow is rich in cellular elements. The number of plasma cells is increased to 29.3%. Single young plasma cells are found), immunophenotyping: the bone marrow sample under study revealed 84.4% T-lymphocytes, 4.8% B-lymphocytes and 9.9% NK cells. B-lymphocytes are polyclonal. Immunophenotyping of this bone marrow sample revealed a population of 20.4% monoclonal plasma cells from the leukocyte region with an aberrant immunophenotype: CD45- CD38+CD138+cytLambda +CD56+CD19-CD20-CD200+CD117-CD27-CD28+, ELISA analysis Beta-2 microglobulin – 6,87 mg/l (N less 2,4). A paraprotein consisting of kappa/lambda light chains was detected. M-gradient, typing. Serum electrophoresis and immunofixation with a panel of antisera (IgG/A/M/kappa/lambda) with quantitative assessment of the M-gradient. Result: Ig G higher than 30; Ig A higher than 10.

Treatment

Bortezomib, Dexamethasone, cyclophosphamide, losartan, aspirin, Rosuvastatin, iron-reparations with folic acid, chronic programmed hemodialysis.

Thus, the difficulties in diagnosis are due to the atypical clinical picture, in which internal organ damage was dominant – renal failure, no damage to flat bones.

DISCUSSION

MM is a hematological myeloproliferative disease effecting multiple organ systems. The primary causes of renal injury were related to monoclonal immunoglobulins include cast nephropathy, monoclonal immunoglobulin deposition disease (MIDD), and light chain (AL) amyloidosis.[8] Patients often present with features of proteinuria and kidney failure.[9]

Investigations such as nephrobiopsy, ELISA analysis Beta-2 microglobulin, detection of monoclonal light chains in urine using urine protein electrophoresis with immunofixation, M-gradient, typing. Serum

electrophoresis and immunofixation with a panel of antisera (IgG/A/M/kappa/lambda) with quantitative assessment of the M-gradient can be used to make a definitive diagnosis on the condition. Pateints diagnosed with renal amyloidosis related to multiple myeloma can be treated with dialysis when progressed to end-stage renal disease.[10] The presence of AL amyloidosis is noticed as a poor prognostic factor in patients with multiple myeloma.[11]

CONCLUSION

This case discusses about a 45-year-old female patient presented with renal failure and later diagnosed with multiple myeloma stage 3 complicated by AL renal amyloidosis leading to stage 5 chronic renal failure. The diagnosis was made based on the CRAB criteria after an extensive and standard set of investigations. The patient was managed conservatively with recommendations for hemodialysis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Blommestein HM, van Beurden-Tan CHY, Franken MG, Uyl-de Groot CA, Sonneveld P, Zweegman S. Efficacy of first-line treatments for multiple myeloma patients not eligible for stem cell transplantation: a network meta-analysis. Haematologica. 2019;104(5):1026-35.
2. Pasca S, Tomuleasa C, Teodorescu P, Ghiaur G, Dima D, Moisoiu V, et al. KRAS/NRAS/BRAF Mutations as Potential Targets in Multiple Myeloma. Front Oncol. 2019;9:1137.
3. Dhodapkar MV. MGUS to myeloma: a mysterious gammopathy of underexplored significance. Blood. 2016;128(23):2599-606.
4. Lee IH, Kim CY, Kang S, Ahn DJ, Kim MK. Multiple myeloma-associated light chain amyloidosis involving heart, kidneys, and peripheral nerves. A case report. Medicine. 2021;2(8):e0128.
5. Leal G, Ballardin B, Rissete M, Amenábar J, Schussel J. Lingual amyloidosis associated to multiple myeloma: case report. RGO - Revista Gaúcha de Odontologia. 2022;70:e20220029.
6. Gurung R, Li T. Renal Amyloidosis: Presentation, Diagnosis, and Management. Am J Med. 2022;135:S38-43.
7. Bhuyan N, Chaubey M, Chakravarty J, Pal A. Renal amyloidosis causing nephrotic syndrome: An unusual presentation of light chain myeloma. Nat J Physiol Pharm Pharmacol. 2022;10(4):352-4.
8. Innocenti S, Bacchi B, Allinovi M, Perfetto F, Antonioli E, Marchionni N, et al. A multidisciplinary case report of multiple myeloma with renal and cardiac involvement: a look beyond amyloidosis. BMC Nephrol. 2022;23(1):370.
9. Karam S, Haidous M, Royal V, Leung N. Renal AA amyloidosis: presentation, diagnosis, and current therapeutic options: a review. Kidney Int. 2023;103(3):473-84.
10. Gurung R, Li T. Renal Amyloidosis: Presentation, Diagnosis, and Management. Am J Med. 2022;135:S38-43.
11. Xu J, Wang M, Shen Y, Yan M, Xie W, Wang B, et al. Effects of Amyloid Light-Chain Amyloidosis on Clinical Characteristics and Prognosis in Multiple Myeloma: A Single-Center Retrospective Study. Cancer Manag Res. 2021;13:1343-56.

Cite this article as: Barysenka T, Kokwewa RBSWMPM, Gawarammana MMSP. A clinical case of multiple myeloma complicated by renal amyloidosis. Int J Res Med Sci 2025;13:5498-501.