GMR
The Original

MULTIPLE MYELOMA ASSOCIATED WITH AL AMYLOIDOSIS PRESENTING AS NEPHROTIC SYNDROME: A RARE CASE REPORT

Hensha H.S.[1], Hepsiba Selva Kumari K[2], Parameshwar S M[3], Shathika J[4], Keerthiga J[5], Dr. K. Karthickeyan[6], Dr. K. Bharathi Priya[*7]

[1]Pharm D Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID : hensha2003@gmail.com , Orchid ID : 0009-0007-8253-2148
[2]Pharm D. Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID : hepsibakrishnamoorthy@gmail.com , Orchid ID: 0009-0003-9664-4889
[3]Pharm D. Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID: parameshwararun007@gmail.com , Orchid ID: 0009-0009-1656-7726
[4]Pharm D. Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID : shathikaj2001@gmail.com , Orchid ID: 0009-0008-3361-8778
[5]Pharm D. Intern, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID : keerthigajaisankar@gmail.com , Orchid ID : 0009-0006-1329-2905
[6]Professor and Head, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID: hodppractice@vistas.ac.in , Orchid ID : 0000-0002-1709-730X
[7*]Professor, Department of Pharmacy Practice, School of Pharmaceutical Sciences, Vels Institute of Science, Technology & Advanced Studies, Chennai, Tamil Nadu, India. Email ID: bharathipriya.sps@vistas.ac.in , Orchid ID: 0000-0002-5022-6636

ABSTRACT

Background: AL amyloidosis is an uncommon systemic condition due to deposits of monoclonal immunoglobulin light chains in different organs, predominantly kidneys. Myeloma is a plasma cell tumor which can occur together with AL amyloidosis and can result in organ dysfunction and poor prognosis. Renal involvement is often manifested as nephrotic syndrome and can be the first presentation of the disease.

Case Presentation : A 62-year-old man had come with progressive bilateral pedal edema, facial swelling, foamy urine, generalised weakness, and anorexia for 3 months. His investigations were suggestive of nephrotic range proteinuria (8.4 g/day), hypoalbuminemia, hyperlipidemia, anemia, raised erythrocyte sedimentation rate, and impaired renal function. His serum protein electrophoresis showed a monoclonal M-spike while immunofixation electrophoresis was suggestive of monoclonal lambda light chains. The bone marrow biopsy showed approximately 30% clonal plasma cells suggesting multiple myeloma. The renal biopsy was positive for Congo red staining with apple green birefringence in polarised light suggesting amyloidosis. He was started on a chemotherapy combination consisting of bortezomib, cyclophosphamide, and dexamethasone along with treatment for nephrotic syndrome. There was marked improvement in his proteinuria, serum albumin, renal function, and other hematological parameters.

Conclusion: This case underscores the necessity to include AL amyloidosis and plasma cell dyscrasia in the differential diagnosis in cases where there is an atypical presentation of nephrotic syndrome. It is crucial to make an early diagnosis through appropriate investigations such as laboratory studies, bone marrow study, and kidney biopsy for better prognosis.

KEYWORDS: Multiple myeloma; AL amyloidosis; Nephrotic syndrome; Plasma cell dyscrasia; Renal amyloidosis; Proteinuria.

1. INTRODUCTION

Multiple myeloma is a type of hematological cancer that is marked by the proliferation of clonal plasma cells in the bone marrow, leading to the increased production of immunoglobulins or light chains. This form of cancer represents about 10 percent of all hematology cancers. It occurs mostly in elderly people whose mean age at the time of diagnosis is estimated at 65 years. The disease presents a number of complications such as anemia, bone pain, high calcium concentration, frequent infections, and renal dysfunction. Of these complications, renal dysfunction is important since it plays a great part in morbidity and mortality. Diagnosis of multiple myeloma is not easy owing to its varied symptoms and their resemblance to other chronic illnesses in old age.[1,2]

Amyloidosis describes a set of conditions that involve deposition of insoluble protein fibrils outside the cells causing damage and dysfunction. AL amyloidosis is the most frequent type of systemic amyloidosis that arises in conjunction with plasma cell disorders, such as multiple myeloma. In AL amyloidosis, there is an excessive production of light chains in abnormal plasma cells that leads to misfolded light chains being deposited as amyloid fibrils. Amyloid deposits are usually found in multiple organs, especially kidney, heart, liver, gastrointestinal tract, and peripheral nerves. Clinical picture of this disorder varies according to which organs are involved and how extensive the deposits are. Since the signs and symptoms of the disease are quite unspecific, diagnosis is often made only when organs begin malfunctioning.[3,4]

The involvement of the kidney is one of the most frequent and significant findings of AL amyloidosis. Deposits of amyloid within the glomeruli impair their filtration function, causing proteinuria and nephrotic syndrome. Manifestations include generalized edema, low levels of blood albumin, high cholesterol levels, and impaired kidney function. In a number of

patients, nephrotic syndrome may represent an initial and the only finding before plasma cell dyscrasia is identified. Nephrotic syndrome may be caused by a variety of conditions such as diabetes mellitus, membranous nephropathy, focal segmental glomerulosclerosis, and minimal change disease. Thus, the diagnosis may be difficult to establish without specific lab tests and renal biopsy.[5,6]

The presence of both conditions of multiple myeloma and AL amyloidosis in one patient denotes a highly aggressive disease process with a higher degree of organ involvement and worse clinical outcome. In around 10-15% of cases of multiple myeloma, AL amyloidosis occurs. On the other hand, in a considerable number of cases of AL amyloidosis, there is an existing plasma cell clone. Among the organs affected by amyloidosis, the kidney and the heart are the most common. The diagnosis can become late enough to cause permanent damage to the organs, making treatment difficult and causing death. Thus, early recognition of amyloidosis symptoms in patients with plasma cell disorder is very important.[7,8]

The current case represents a very unusual case of multiple myeloma with associated AL amyloidosis with a predominant manifestation of nephrotic syndrome in a senior male. The diagnosis was confirmed using a number of methods of clinical examination and investigations, including serum protein electrophoresis, immunofixation, bone marrow examination, and renal biopsy with positive Congo red staining. The patient demonstrated a substantial improvement both clinically and biochemically under treatment by a regimen containing bortezomib and nephrological support. This case illustrates that consideration of plasma cell disorders and systemic amyloidosis should be considered in patients with idiopathic nephrotic-range proteinuria.[9,10]

CASE PRESENTATION

Patient Demographics

A 62-year-old male patient reported to the Department of General Medicine in a tertiary care teaching hospital with complaints of increasing swelling in both lower limbs, puffy face, generalized weakness, and frothy urine for the last three months. The patient came from a rural background and was a farmer by profession for more than 35 years. The patient was married and belonged to the middle socioeconomic stratum. There was no history of any travel recently, industrial exposure, or environmental hazards. There was no history of any familial predisposition to hematological cancers, kidney diseases, or amyloidosis.

Chief Complaints

The patient presented with the following complaints:

- Bilateral lower limb swelling for 3 months
- Facial puffiness for 2 months
- Frothy urine for 3 months
- Generalized weakness and easy fatigability for 2 months
- Decreased appetite for 1 month
- Unintentional weight loss of approximately 5 kg over the preceding 4 months

History of Present Illness

The patient appeared to be well until three months before admission when he developed swelling in both his ankle areas. This initial swelling started mildly and gradually worsened becoming more pronounced, extending to the knees. With time, he developed facial puffiness especially in the mornings along with general body weakness and intolerance to activity. He stated that he had passing of foamy urine which suggested severe proteinuria. He did not have complaints of burning during urination, frequency, urgency, blood-streaked urine, flank pain, and decrease in volume of urine production. He did not have complaints of fever, chills, skin rashes, joint pains, oral ulcers, sensitivity to light, and symptoms suggestive of collagen diseases. One month prior to admission, he had developed loss of appetite and weakness. Additionally, he had complained of mild breathlessness on exertion and did not have complaints of chest pain, palpitations, difficulty breathing while lying flat, and nocturnal breathlessness.

Past Medical History

The patient had a history of hypertension diagnosed eight years earlier and was receiving regular antihypertensive treatment. Blood pressure had remained reasonably controlled during follow-up visits.

Medication History

- At the time of presentation, the patient was taking:
- Tablet Amlodipine 5 mg once daily for hypertension
- Occasional Tablet Paracetamol 500 mg for generalized body aches

Family History

There was no known family history of any disease.

Social History

The patient was married and lived with his family. He maintained an active lifestyle before the onset of symptoms, Non-smoker and Non-Alcoholic. His nutritional intake had decreased during the month preceding hospitalization because of poor appetite.

Occupational History
The patient had been working as a farmer for more than three decades. His work involved moderate physical activity and routine exposure to agricultural environments.

Allergic History
The patient denied any history of allergies
- Physical Examination
- General Examination

The patient was conscious, alert, and oriented to time, place, and person.
Vital Signs

Parameter	Value
Temperature	98.4°F
Pulse Rate	88 beats/min
Blood Pressure	148/94 mmHg
Respiratory Rate	18 breaths/min
Oxygen Saturation	98% on room air
Weight	72 kg
Height	168 cm
BMI	25.5 kg/m²

Systemic Examination
Cardiovascular System
- S1 and S2 heard normally
- No murmurs
- No gallops
- Jugular venous pressure not elevated
- Respiratory System
- Bilateral air entry equal
- No wheeze or crackles
- Normal vesicular breath sounds

Gastrointestinal System
- Mild hepatomegaly palpable 2 cm below right costal margin
- No splenomegaly
- No ascites
- Bowel sounds normal

Nervous System
- Higher mental functions intact
- Cranial nerves normal
- Motor examination normal
- Sensory examination normal
- No peripheral neuropathy

Musculoskeletal System
- No bone tenderness
- No joint swelling
- No deformities
- Laboratory Investigations

Complete Blood Count

Investigation	Result	Reference Range
Hemoglobin	9.6 g/dL	13–17 g/dL
RBC Count	3.2 ×10⁶/µL	4.5–5.9 ×10⁶/µL
Total Leukocyte Count	7,800 cells/mm³	4,000–11,000
Platelet Count	248,000/mm³	150,000–450,000
ESR	96 mm/hr	<20 mm/hr

Renal Function Tests

Investigation	Result
Blood Urea Nitrogen	56 mg/dL
Serum Creatinine	2.3 mg/dL
eGFR	38 mL/min/1.73m²
Uric Acid	7.8 mg/dL

Liver Function Tests

Investigation	Result
Total Protein	5.7 g/dL
Serum Albumin	2.1 g/dL
Globulin	3.6 g/dL
Total Bilirubin	0.8 mg/dL
AST	32 U/L
ALT	28 U/L
ALP	124 U/L

Lipid Profile

Investigation	Result
Total Cholesterol	338 mg/dL
Triglycerides	248 mg/dL
LDL Cholesterol	196 mg/dL
HDL Cholesterol	38 mg/dL

Serum Electrolytes

Investigation	Result
Sodium	137 mmol/L
Potassium	4.5 mmol/L
Chloride	102 mmol/L
Bicarbonate	23 mmol/L

Urinalysis

Parameter	Result
Appearance	Pale yellow
Specific Gravity	1.020
Protein	4+
Glucose	Negative
RBCs	2–3/HPF
Pus Cells	1–2/HPF
Casts	Occasional hyaline casts
24-Hour Urinary Protein Excretion	8.4 g/day

erum Protein Electrophoresis (SPEP)

- Presence of monoclonal spike (M-band) in gamma region
- M-protein concentration: 2.9 g/dL
- Immunofixation Electrophoresis
- Monoclonal Lambda Light Chain detected
- Consistent with plasma cell dyscrasia

Serum Free Light Chain Assay

Parameter	Result
Free Kappa Light Chain	18 mg/L
Free Lambda Light Chain	425 mg/L
Kappa/Lambda Ratio	0.04

Bone Marrow Aspiration and Biopsy

- Hypercellular marrow
- Plasma cells constituted approximately 30% of nucleated cells
- Sheets and clusters of atypical plasma cells observed

Immunohistochemistry

Marker	Result
CD138	Positive
CD38	Positive
Lambda Light Chain	Positive
Kappa Light Chain	Negative

Radiological Investigations
Skeletal Survey

- Multiple punched-out lytic lesions involving skull vault
- Osteolytic lesions involving thoracic vertebrae
- Ultrasonography Abdomen
- Bilateral kidneys normal in size
- Increased cortical echogenicity
- Mild hepatomegaly

Renal Biopsy
Light Microscopy : Extensive amorphous eosinophilic deposits in glomeruli and vascular walls and Mesangial expansion noted
Congo Red Staining : Strongly positive
Polarized Light Microscopy : Apple-green birefringence observed
Electron Microscopy : Randomly arranged non-branching fibrils measuring 8–12 nm
Histopathological Diagnosis : Renal AL (Light Chain) Amyloidosis

FINAL DIAGNOSIS
Diagnosis of Multiple Myeloma with AL (Light Chain) Amyloidosis presenting as Nephrotic Syndrome was made by the combination of the patient's clinical features, laboratory tests, and histopathological analysis. Clinically, the patient had progressive edema of feet, puffiness of face, foamy appearance of urine, and weakness, indicative of nephrotic syndrome. Lab tests showed presence of nephrotic range proteinuria (8.4 g/day), hypoproteinemia, hyperlipidemia, anemia, high serum creatinine, and M protein spike on serum protein electrophoresis. Bone marrow analysis showed 30% clonal plasma cell proliferation with lambda light chain restriction. Congo red stained biopsy with apple green birefringence suggested AL amyloidosis.

DIFFERENTIAL DIAGNOSIS
Differential diagnoses that considered were primary nephrotic syndrome, diabetic nephropathy, membranous nephropathy, Focal Segmental Glomerulosclerosis (FSGS), minimal change disease, and secondary amyloidosis (AA amyloidosis). Diabetic nephropathy was ruled out since there was no history of diabetes mellitus or its complications. Both membranous nephropathy and FSGS were possible considering the heavy proteinuria; however, they were eventually ruled out using renal biopsy. Possibility of secondary amyloidosis was considered unlikely since there was no history of chronic inflammatory or infectious diseases. Anemia, increased ESR, monoclonal gammopathy, abnormal serum free light chain ratio, plasma cell infiltrate in the bone marrow, and Congo red positivity in the kidney biopsy confirmed multiple myeloma AL amyloidosis.

TREATMENT AND MANAGEMENT
After admission, the patient was subjected to conservative management aimed at controlling the features of nephrotic syndrome and preserving renal function. Fluid and sodium restriction were suggested for reduction of edema. Furosemide 40 mg orally two times per day was prescribed initially, then titrated depending on urine volume and clinical response. In order to improve diuresis and preserve potassium balance, Spironolactone 25 mg orally one time per day was administered. Ramipril 2.5 mg orally one time per day was prescribed with gradual increase in dosage as a renoprotective agent and a means to reduce proteinuria.
The patient showed features of hypoalbuminemia and hyperlipidemia due to nephrotic syndrome. The patient was counseled about nutritional therapy with guidelines regarding the consumption of moderate amounts of protein considering renal function. The use of atorvastatin 20 mg orally at bedtime was indicated to control dyslipidemia. Considering that nephrotic syndrome and AL amyloidosis increase the risk of thromboembolic events, the patient was carefully assessed for any sign of venous thrombosis.

Anti-Myeloma and Anti-Amyloid Therapy
After confirmation of multiple myeloma with AL amyloidosis, the patient was initiated on a Bortezomib-Cyclophosphamide-Dexamethasone (VCd) regimen, which is considered a standard first-line treatment for AL amyloidosis associated with plasma cell dyscrasias. The regimen consisted of:

- Bortezomib 1.3 mg/m² subcutaneously on Days 1, 8, 15, and 22
- Cyclophosphamide 300 mg/m² orally once weekly
- Dexamethasone 40 mg orally once weekly

Each treatment cycle was repeated every 28 days. The objective of therapy was rapid suppression of monoclonal light-chain production, reduction of amyloid deposition, and prevention of further organ damage.
To minimize treatment-related complications, the patient received comprehensive supportive care. Acyclovir 400 mg orally twice daily was administered as antiviral prophylaxis against herpes zoster reactivation associated with bortezomib therapy. Pantoprazole 40 mg orally once daily was prescribed for gastrointestinal protection during corticosteroid treatment. Antiemetic prophylaxis with Ondansetron 8 mg orally as needed was provided for chemotherapy-induced nausea. Complete blood counts, renal parameters, liver function tests, and serum free light chains were monitored regularly to assess treatment response and detect adverse effects promptly.

Chemotherapy Schedule (VCd Regimen)

Day	Bortezomib	Cyclophosphamide	Dexamethasone
Day 1	1.3 mg/m² SC	300 mg/m² PO	40 mg PO
Day 8	1.3 mg/m² SC	300 mg/m² PO	40 mg PO
Day 15	1.3 mg/m² SC	300 mg/m² PO	40 mg PO
Day 22	1.3 mg/m² SC	300 mg/m² PO	40 mg PO

LONG-TERM FOLLOW-UP AND RESPONSE ASSESSMENT

The patient received four cycles of VCd therapy with acceptable tolerability without experiencing any serious side effects. The patient was followed up, and it was found that there was a significant decrease in the edema in the limbs, normalization of the serum albumin level, decrease in proteinuria, and stabilization of renal function. There was a significant reduction in the levels of serum free lambda light chain, suggesting hematologic response to treatment.

DISCUSSION

This current case emphasizes the need to consider AL amyloidosis in cases where there is nephrotic syndrome without a cause. Pedal swelling, puffiness of face, frothy urine, and massive proteinuria are some of the features associated with nephrotic syndrome. Nevertheless, the patient also had anemia, high ESR, renal impairment, and hypoalbuminemia. This led to a diagnosis of a systemic disease such as AL amyloidosis. A similar presentation was recorded by Meyskens et al., where patients presented with AL amyloidosis and primary nephrotic syndrome. On the other hand, Kilic et al. recorded a case of AL amyloidosis complicated by multiple myeloma, who presented with nephrotic-range proteinuria.[11,12]

In this particular case, the diagnosis was made using serum protein electrophoresis, serum free light chain analysis, bone marrow examination, and renal biopsy. Monoclonal lambda light chains and 30% plasma cells infiltrates in bone marrow confirmed the diagnosis of multiple myeloma, whereas Congo red staining showing apple green birefringent deposits helped to diagnose AL amyloidosis. Renal biopsy is the gold standard test in establishing amyloid deposits and ruling out other conditions presenting with nephrotic syndrome. In one case report by Fuah and Lim, a similar presentation was managed using renal biopsy for confirming renal-limited AL amyloidosis. Moreover, in another literature review about renal-limited AL amyloidosis, tissue biopsy still plays an important role in diagnosis and management.[13,14]

The involvement of kidneys is among the most common organ complications seen in AL amyloidosis and contributes greatly to the morbidity of the patients. In the present patient, proteinuria and impaired renal function were the most prominent findings. The deposition of amyloid in the glomeruli had caused the disturbance of the filtration mechanism and led to the development of nephrotic syndrome. In their study, Innocenti et al. demonstrated that renal manifestations are common among patients suffering from multiple myeloma and negatively impact the prognosis in cases when the diagnosis is made late. In addition, Katagiri et al. described a case of a patient with systemic AL amyloidosis and nephrotic syndrome where the progression of renal disease was the most prominent complication.[15,16]

Our patient demonstrated a positive response to chemotherapy involving bortezomib along with supportive therapy for nephrotic syndrome. There have been significant decreases in the levels of proteinuria and serum free light chains in follow-up observations, which suggest hematological and renal responses. It is important to begin the treatment early enough since further amyloid accumulation might cause permanent damage. According to J Vela et al., patients with multiple myeloma and amyloidosis have worse outcomes and need an aggressive treatment approach. In addition, there are several clinical observations which show that chemotherapy regimens with bortezomib successfully control pathological clones of plasma cells and normalize organ function in AL amyloidosis patients.[17,18]

CONCLUSION

This case highlights the rare coexistence of multiple myeloma and AL (light-chain) amyloidosis presenting as nephrotic syndrome, emphasizing the diagnostic challenges associated with plasma cell dyscrasias. The patient's initial presentation with progressive edema, nephrotic-range proteinuria, hypoalbuminemia, and renal dysfunction necessitated extensive evaluation, ultimately leading to the diagnosis through serum monoclonal protein studies, bone marrow examination, and renal biopsy. Early recognition of AL amyloidosis is crucial because delayed diagnosis may result in irreversible organ damage and poor clinical outcomes. Prompt initiation of a bortezomib-based chemotherapy regimen along with supportive nephrological management resulted in significant hematological and renal improvement. This case underscores the importance of considering underlying hematological malignancies in patients with unexplained nephrotic syndrome and demonstrates the value of a multidisciplinary approach in achieving favorable outcomes.

REFERENCES

1. Nandra TK, Devi A, Jones JR. Multiple myeloma: what a non-haematologist should know. Clin Med (Lond). 2022 May;22(3):230-233
2. Faiman BM, Mangan P, Spong J, Tariman JD; The International Myeloma Foundation Nurse Leadership Board. Renal complications in multiple myeloma and related disorders: survivorship care plan of the International Myeloma Foundation Nurse Leadership Board. Clin J Oncol Nurs. 2011 Aug;15 Suppl(Suppl):66-76.
3. Blancas-Mejía LM, Ramirez-Alvarado M. Systemic amyloidoses. Annu Rev Biochem. 2013;82:745-74.
4. Baker KR, Rice L. The amyloidoses: clinical features, diagnosis and treatment. Methodist Debakey Cardiovasc J. 2012 Jul-Sep;8(3):3-7
5. Wang AA, Kanwar YS, Aggarwal V, Srivastava A. AL Amyloidosis Presenting With Crescentic Glomerulonephritis. Kidney Med. 2021 Apr 20;3(4):644-648.

6. Wendt R, Sobhani A, Diefenhardt P, Trappe M, Völker LA. An Updated Comprehensive Review on Diseases Associated with Nephrotic Syndromes. Biomedicines. 2024 Oct 4;12(10):2259
7. Ríos-Tamayo R, Krsnik I, Gómez-Bueno M, Garcia-Pavia P, Segovia-Cubero J, Huerta A, Salas C, Silvestre RÁ, Sánchez A, Manso M, Delgado L, Lahuerta JJ, Martínez-López J, Duarte RF. AL Amyloidosis and Multiple Myeloma: A Complex Scenario in Which Cardiac Involvement Remains the Key Prognostic Factor. Life (Basel). 2023 Jul 6;13(7):1518.
8. Gioeva Z, Mikhaleva L, Tsutsaev A, Tebenkova A, Gutyrchik N, Shakhpazyan N, Ilyichev A, Kakturskij L. Multiple Myeloma Concomitant with AL Amyloidosis: Histopathological Aspects of the Common Plasma Cell Spectrum. International Journal of Molecular Sciences. 2026; 27(11):5120.
9. Kogler W, Canha C, Makary R, Omman R, Isache CL. Multiple myeloma with extensive AL amyloidosis presenting as chronic diarrhoea. BMJ Case Rep. 2020 Jan 8;13(1):e232934
10. Beyler Kilic O, Oguz AK, Ergun I, Baydar DE, Ayli M. Nonsecretory Multiple Myeloma and AL Amyloidosis Presenting with Nephrotic Range Proteinuria. Case Rep Nephrol. 2015;2015:635974.
11. Meyskens, T. & Lerut, E. & Arnouts, Paul & Rycke, A.. (2015). AL amyloidosis presenting as nephrotic syndrome: Case reports and literature survey. Tijdschrift voor Geneeskunde. 71. 769-780
12. Beyler Kilic O, Oguz AK, Ergun I, Baydar DE, Ayli M. Nonsecretory Multiple Myeloma and AL Amyloidosis Presenting with Nephrotic Range Proteinuria. Case Rep Nephrol. 2015;2015:635974.
13. Fuah KW, Lim CTS. Renal-limited AL amyloidosis - a diagnostic and management dilemma. BMC Nephrol. 2018 Nov 6;19(1):307.
14. Velayati S, Belkin A, Sidhu Kumar G, Tharayil ZJ, Kumar N, Patel S. Kidney-limited AL amyloidosis: a case report and review of the literature. J Community Hosp Intern Med Perspect. 2021 Sep 20;11(5):698-702
15. Innocenti S, Bacchi B, Allinovi M, Perfetto F, Antonioli E, Marchionni N, Di Mario C, Caroti L, Cappelli F, Stefàno P. A multidisciplinary case report of multiple myeloma with renal and cardiac involvement: a look beyond amyloidosis. BMC Nephrol. 2022 Nov 17;23(1):370.
16. Katagiri T, Miyazawa K, Yahata N, Gotoh A, Serizawa H, Ohyashiki K. A long-survival case of systemic AL amyloidosis with nephrotic syndrome. Intern Med. 2002 Nov;41(11):1052-5
17. Crosthwaite A, Skene A, Mount P. Rapidly progressive glomerulonephritis complicating primary AL amyloidosis and multiple myeloma. Nephrol Dial Transplant. 2010 Aug;25(8):2786-9.
18. Vela-Ojeda J, García-Ruiz Esparza MA, Padilla-González Y, Sánchez-Cortes E, García-Chávez J, Montiel-Cervantes L, Reyes-Maldonado E, Majluf-Cruz A, Mayani H. Multiple myeloma-associated amyloidosis is an independent high-risk prognostic factor. Ann Hematol. 2009 Jan;88(1):59-66.