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Renal Amyloidosis Presenting as Nephrotic Syndrome and Hypotension

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ABSTRACT

Background: Renal amyloidosis is a rare cause of nephrotic syndrome, often presenting with heavy proteinuria, hypoalbuminemia, and progressive renal dysfunction. Systemic involvement may lead to autonomic dysfunction and persistent hypotension, posing diagnostic and therapeutic challenges. ***Case Presentation:*** We report a 52-year-old male who presented with frothy urine, edema, and persistent low blood pressure. Laboratory investigations revealed nephrotic-range proteinuria (urine microalbumin 5799 mg/L), severe hypoalbuminemia (1.9 g/dL), hypercholesterolemia, and preserved renal function (serum creatinine 1.2 mg/dL). Ultrasound showed normal-sized kidneys with preserved parenchyma. Renal biopsy confirmed primary renal amyloidosis. Despite corticosteroids, midodrine, diuretics, and albumin infusion, the patient continued to experience symptomatic hypotension, likely secondary to autonomic dysfunction. ***Conclusion:*** This case highlights the importance of early renal biopsy in unexplained nephrotic syndrome and underscores the role of autonomic dysfunction in renal amyloidosis. Multidisciplinary management is essential for improved outcomes.

Keywords: Renal Amyloidosis; Nephrotic Syndrome; Hypotension; Membranous Glomerulonephritis; Autonomic Dysreflexia.

BACKGROUND

Renal amyloidosis is a rare but serious cause of nephrotic syndrome, resulting from extracellular deposition of misfolded protein fibrils, typically light chains (AL type) or serum amyloid A (AA type) within renal tissue [1,2]. The disease commonly presents with heavy proteinuria, hypoalbuminemia, and edema, and may progress to renal failure [1]. Autonomic dysfunction is a recognized complication of systemic amyloidosis and often manifests as persistent hypotension or orthostatic dizziness, especially in advanced stages [3]. Diagnosis relies on histopathological confirmation via kidney biopsy, with Congo red staining demonstrating amyloid deposits under polarized light [4,5].

CLINICAL PRESENTATION

History of Presenting Illness

A 52-year-old male patient reported frothy urine for 6–8 months and bilateral pedal edema for the past 5–6 months. He also experienced reduced urine output, chronic low blood pressure (as low as 76/46 mmHg), dizziness upon walking, and generalized fatigue. There was no history of hematuria. Abdominal and back pain were also noted.

Examination Findings

- *General:* Alert, oriented, hypotensive, mild pallor
- *Edema:* Pedal edema +4, scrotal edema +4
- *Chest:* Clear on auscultation
- *Systemic:* Unremarkable findings

Medical and Surgical History

- Cholecystectomy (September 2024)
- No prior autoimmune disease, diabetes, or hypertension

INVESTIGATIONS

Laboratory Results

- *18/12/2024:*
  - Urine microalbumin: 2302
  - Urine protein: 2+, RBC: 2–4/HPF, WBC: 0–2
  - Hgb: 12.6, WBC: 9700, Platelets: 227
  - Serum albumin: 1.9, Total protein: 4.1
  - Cholesterol: 340, Triglycerides: 168, LDL: 246, HDL: 280
  - Serum creatinine: 1.2 mg/dL

- *20/12/2024:*
  - Urine microalbumin: 5799
  - Urine microalbumin/creatinine: 2806
  - Albumin: 1.9, Total protein: 4.36
  - SPEP: No M band

Radiology

- *Ultrasound KUB:*
  - Right kidney: 10.9x4.6 cm; Left kidney: 9.1x4.1 cm
  - Parenchymal thickness preserved; Mildly enlarged prostate
  - No hydronephrosis or renal stones

- *Renal Biopsy:*
  - Histopathology: Light microscopy and immunofluorescence consistent with primary renal amyloidosis
  - No immediate post-procedure complications

DIAGNOSIS

- Primary Renal Amyloidosis
- Nephrotic Syndrome with Diffuse Membranous Glomerulonephritis
- Hypotension is likely secondary to autonomic dysreflexia
- Rule out: Multiple Myeloma (pending bone marrow biopsy)

OUTCOME

The patient was empirically started on prednisolone (1 mg/kg/day) and managed for hypotension with midodrine 5 mg BID, later increased to 10 mg. Apixaban 5 mg BID was given until April 7th, then tapered to 2.5 mg BID. IV albumin and IV Lasix were administered when systolic BP reached ≥90 mmHg.

Additional medications included pantoprazole, vitamin D, calcium supplements, and symptomatic management with Buscopan and Flatuna. Fluid intake was restricted to 1L/day, and daily vitals, weight, and I/O were monitored.

Upon stabilization, the patient was discharged with prescriptions and advised to seek oncology/ hematology consultation for suspected plasma cell dyscrasia.

DISCHARGE MEDICATIONS

- *Pantozol 40 mg:* 1 tab/day for 15 days
- *Vitamin D 50,000 IU:* 1 cap/week
- *Flatuna:* BID for 15 days
- *Xtracal:* BID for 15 days
- *Buscopan 10 mg:* TID for 5 days
- *Eliquis 2.5 mg:* BID for 15 days
- *Lasix 40 mg:* BID for 15 days
- *Midodrine 10 mg:* OD for 15 days

FOLLOW-UP AND OUTCOME

The patient continued to experience dizziness and fatigue on standing, likely due to persistent autonomic dysfunction. He reported bony pains and was advised to reduce prednisolone to 40 mg/day in preparation for chemotherapy using dexamethasone. Further care was advised under a hematologist with the plan for bone marrow biopsy and initiation of chemotherapy for suspected plasma cell dyscrasia.

CONCLUSION

This case highlights the diagnostic and therapeutic challenges in managing nephrotic syndrome with an underlying infiltrative etiology. Early renal biopsy and a multidisciplinary approach are crucial. Persistent hypotension in the setting of amyloidosis should raise suspicion for autonomic involvement.

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