World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

Cardiac AL amyloidosis with multisystem manifestations: Case report and brief literature review

Alireza Izadian Bidgoli [1], Nimra Khan[2], Nelson Chang Tsang [2], Thomas Saliba [1], Corey Steinman [1], Jessica Jahoda [3, 4, *], Yehuda Wenger [3] and Mohamed Aziz [4, 5]

[1] *American University of the Caribbean, AUC, St. Maarten.*
[2] *Ross University School of Medicine, Barbados.*
[3] *Memorial Healthcare System, Pembroke Pines, FL, USA.*
[4] *Research Writing & Publication (RWP), LLC, NY, USA.*
[5] *Saint Vincent's Medical Center, New York City, NY, USA.*

World Journal of Advanced Research and Reviews, 2026, 30(01), 191-198

Publication history: Received on 24 February 2026; revised on 01 April 2026; accepted on 03 April 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.30.1.0840

Abstract

Systemic light-chain (AL) amyloidosis is a rare but lethal plasma cell disorder characterized by amyloid deposition from misfolded immunoglobulin light chains, often leading to multi-organ dysfunction. Cardiac involvement is the predominant driver of prognosis and survival.

A 66-year-old man presented with an eight-month history of progressive exertional dyspnea, fatigue, and bilateral leg edema. His past medical history included controlled hypertension and diabetes mellitus. Physical examination revealed signs of advanced heart failure, including elevated jugular venous pressure (12 cm H_2O), bibasilar crackles, peripheral edema, and neuropathy. Electrocardiography showed low-voltage QRS complexes in the limb leads, and echocardiography demonstrated concentric left ventricular hypertrophy (septal thickness 16 mm) with preserved systolic function. Strain imaging displayed an apical-sparing "cherry-on-top" pattern, and cardiac MRI revealed markedly increased extracellular volume, suggesting amyloid infiltration.

Endomyocardial biopsy confirmed amyloid deposition, exhibiting Congo red positivity with apple-green birefringence and lambda light chain restriction. The patient was diagnosed with systemic AL amyloidosis and advanced cardiac involvement. He was treated with bortezomib, cyclophosphamide, and dexamethasone, and later escalated to daratumumab due to limited hematologic response. Despite aggressive therapy, he developed refractory heart failure and died 29 months after diagnosis.

This case highlights the subtle early signs of cardiac amyloidosis and underscores the critical importance of recognizing and treating it promptly before irreversible organ damage develops.

Keywords: Light-chain amyloidosis; Cardiac; Misfolded immunoglobulin; Congo red; Refractory heart failure

1. Introduction

Systemic light-chain (AL) amyloidosis is a rare but life-threatening plasma cell dyscrasia characterized by the widespread extracellular deposition of misfolded monoclonal immunoglobulin light chains as insoluble amyloid fibrils in vital organs. [1] With an estimated annual incidence of 9 to 14 cases per million person-years in the United States and a median age at diagnosis of 64 years, the disease predominantly affects older adults and carries a slight male

* Corresponding author: Jessica Jahoda

predominance. [2] Its clinical presentation is notoriously heterogeneous and insidious, frequently resulting in significant diagnostic delays, as the structural changes in the heart, proteinuria, and neuropathy are often misattributed to more common conditions such as hypertension or diabetes mellitus. [2]

Among all affected organs, the heart is the most critical determinant of prognosis, typically manifesting as a rapidly progressive restrictive cardiomyopathy with diastolic dysfunction. [3] Multisystem involvement, encompassing the kidneys, liver, peripheral and autonomic nervous systems, is present in the majority of patients at the time of diagnosis. [4] Despite recent therapeutic advances, including the incorporation of daratumumab-based frontline regimens, advanced cardiac AL amyloidosis continues to carry high early mortality. [5] Herein, we present a challenging case of a 66-year-old man with advanced cardiac AL amyloidosis and extensive multisystem manifestations, underscoring the diagnostic complexities and the urgent necessity for prompt, multidisciplinary management.

2. Case Presentation

2.1. Clinical Presentation

A 66-year-old man presented to the cardiology clinic with progressive dyspnea and fatigue that had developed over eight months. Initially, his symptoms occurred with moderate exertion, but they gradually progressed to the point that he experienced breathlessness at rest. Over the preceding three months, he also noticed bilateral lower extremity edema and reported intermittent episodes of palpitations. He denied chest pain but described an unintentional weight loss of approximately 12 pounds during the same period.

His past medical history was significant for long-standing hypertension and hyperlipidemia, both treated with medications, as well as type 2 diabetes mellitus diagnosed five years earlier and controlled with metformin. There was no family history of heart failure or amyloidosis. The patient had worked as an accountant, denied tobacco use, and reported only occasional alcohol consumption. He also noted increasing difficulty performing physical activities that he had previously tolerated without limitation.

2.2. Evaluation: Examination, History, and Labs

On physical examination, the patient appeared chronically ill. Vital signs revealed a blood pressure of 105/70 mmHg, a heart rate of 92 beats per minute with an irregular rhythm, a respiratory rate of 20 breaths per minute, and an oxygen saturation of 93% on room air. Jugular venous pressure was elevated at approximately 12 cm of water. Cardiac auscultation revealed distant heart sounds, a fourth heart sound (S4), and a gallop, but no audible murmurs. Pulmonary examination demonstrated bibasilar crackles. Abdominal examination showed mild hepatomegaly, with the liver edge palpable approximately 3 cm below the right costal margin. Bilateral pitting edema extended to the mid-calves. Neurological examination revealed decreased sensation in a stocking–glove distribution consistent with peripheral neuropathy.

Initial laboratory evaluation demonstrated mild normocytic anemia with a hemoglobin level of 11.2 g/dL, an elevated serum creatinine of 1.8 mg/dL, hypoalbuminemia with a serum albumin of 3.0 g/dL, and a markedly elevated B-type natriuretic peptide (BNP) level of 850 pg/mL. Troponin-I was mildly elevated at 0.08 ng/mL. Electrocardiography revealed atrial fibrillation with low-voltage QRS complexes in the limb leads. Notably, these low voltages were observed despite increased left ventricular wall thickness seen on bedside echocardiography, raising concern for an infiltrative cardiomyopathy.

2.3. Radiographic Studies and Differential Diagnosis

Comprehensive transthoracic echocardiography demonstrated concentric left ventricular hypertrophy with an interventricular septal thickness of 16 mm and posterior wall thickness of 15 mm, along with a small left ventricular cavity and preserved left ventricular ejection fraction of approximately 55%. Severe diastolic dysfunction (grade III) was present. The myocardium exhibited a characteristic granular or “sparkling” appearance, and biatrial enlargement was noted. Speckle-tracking strain imaging demonstrated markedly reduced global longitudinal strain of approximately 10% with relative preservation of apical strain, producing the classic “cherry-on-top” pattern frequently associated with cardiac amyloidosis.

Cardiac magnetic resonance imaging further supported an infiltrative process, demonstrating diffuse subendocardial late gadolinium enhancement with difficulty nulling the myocardium, a pattern suggestive of extensive amyloid infiltration. Native T1 mapping values were markedly elevated at approximately 1150 ms (normal <1000 ms), and extracellular volume fraction was significantly increased to 45% (normal <28%). Nuclear scintigraphy with technetium-

99m pyrophosphate (PYP) showed grade 1 myocardial uptake, less than that of the adjacent ribs, a finding more consistent with AL amyloidosis rather than transthyretin-related amyloidosis. Chest radiography revealed mild cardiomegaly with pulmonary vascular congestion.

Based on these findings, several diagnostic possibilities were considered. The leading consideration was cardiac amyloidosis, including AL and transthyretin (ATTR) subtypes. Other potential diagnoses included hypertrophic cardiomyopathy, hypertensive heart disease with secondary left ventricular hypertrophy, and restrictive cardiomyopathy due to other infiltrative or storage disorders such as sarcoidosis or hemochromatosis. Constrictive pericarditis and metabolic disorders such as Fabry disease were also considered in the differential diagnosis. However, the presence of disproportionately low electrocardiographic voltages despite markedly increased ventricular wall thickness strongly suggested an infiltrative cardiomyopathy, particularly amyloidosis.

2.4. Pathological Studies and Final Diagnosis

To establish a definitive diagnosis, an endomyocardial biopsy was obtained from the right ventricular septum. Histopathological examination demonstrated extensive amorphous eosinophilic extracellular deposits between myocardial fibers, resulting in compression and atrophy of adjacent cardiomyocytes. Congo red staining of the specimen revealed characteristic apple-green birefringence under polarized light, confirming the presence of amyloid deposits. Immunohistochemical analysis demonstrated that the deposits were composed of lambda light chains, establishing a diagnosis of AL amyloidosis. Staining was negative for transthyretin, kappa light chains, amyloid A, and other amyloid subtypes.

Subsequent mass spectrometry analysis confirmed that the amyloid fibrils consisted of lambda light chains. Serum and urine immunofixation electrophoresis detected monoclonal lambda free light chains, and serum free light chain analysis revealed a markedly abnormal kappa-to-lambda ratio of 0.01 (reference range 0.26–1.65), with markedly elevated lambda light chains measuring 450 mg/L. A bone marrow biopsy demonstrated approximately 15% clonal plasma cell infiltration, with lambda light-chain restriction confirmed on flow cytometry. Collectively, these findings established the diagnosis of systemic AL (lambda) amyloidosis with cardiac involvement secondary to an underlying plasma cell dyscrasia.

2.5. Multisystem Involvement

Further evaluation revealed evidence of multisystem involvement. Renal assessment demonstrated nephrotic-range proteinuria measuring 4.2 g per 24 hours, predominantly albumin. Renal biopsy confirmed amyloid deposition within the glomeruli with Congo red positivity and lambda light-chain restriction, consistent with AL amyloid nephropathy. The patient's estimated glomerular filtration rate was 42 mL/min/1.73 m^2, corresponding to stage 3 chronic kidney disease. Neurological evaluation revealed progressive paresthesias and numbness in a stocking–glove distribution, with diminished ankle reflexes. Nerve conduction studies demonstrated an axonal sensorimotor polyneuropathy predominantly affecting the lower extremities.

A sural nerve biopsy showed amyloid deposits within the endoneurium and epineurium, as demonstrated by lambda immunostaining, confirming the diagnosis of amyloid-related peripheral neuropathy. Autonomic testing revealed orthostatic hypotension characterized by a blood pressure drop of approximately 35/20 mmHg upon standing, along with abnormal heart rate variability and impaired sudomotor function.

Clinically, the patient reported early satiety, alternating constipation and diarrhea, and erectile dysfunction, findings consistent with autonomic nervous system involvement. Hepatic involvement was also suspected. Laboratory testing demonstrated a mildly elevated alkaline phosphatase level of 180 U/L, and abdominal ultrasound revealed hepatomegaly with a liver span of approximately 18 cm. Although a liver biopsy was not performed because the diagnosis of systemic AL amyloidosis had already been established through other tissue samples, hepatic infiltration by amyloid was considered highly likely based on the clinical and laboratory findings. Soft-tissue manifestations were also observed, including mild periorbital purpura, a characteristic feature of systemic AL amyloidosis, although macroglossia was absent.

2.6. Multidisciplinary Tumor Board Discussion

The patient’s case was subsequently discussed in a multidisciplinary conference that included cardiologists, hematologists/oncologists, nephrologists, radiologists, pathologists, heart failure specialists, and a palliative care physician. During the discussion, the patient was determined to have advanced cardiac involvement consistent with Mayo Clinic cardiac stage III disease, based on the elevation of both cardiac troponin and natriuretic peptide levels.

According to the Boston University staging system, the patient was classified as stage IIIb due to elevated cardiac biomarkers in the setting of impaired renal function with an estimated glomerular filtration rate below 50 mL/min.

The presence of extensive multi-organ involvement, particularly cardiac and renal disease, was recognized as an important adverse prognostic factor. The team emphasized the urgency of initiating therapy given the severity of cardiac involvement while also carefully evaluating the patient's functional status and potential treatment tolerability. The primary therapeutic goal was rapid suppression of the underlying plasma cell clone to halt further amyloid production and preserve organ function.

2.7. Treatment

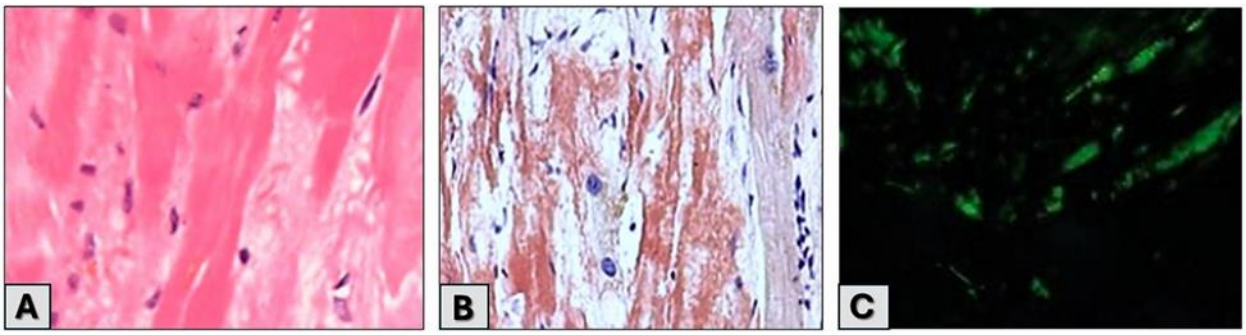
Treatment was initiated with a bortezomib-based chemotherapy regimen consisting of cyclophosphamide, bortezomib, and dexamethasone (CyBorD). Cyclophosphamide was administered orally at 300 mg/m^2 on days 1, 8, and 15; bortezomib was administered subcutaneously at 1.3 mg/m^2 on days 1, 8, and 15; and dexamethasone was given orally at 40 mg on days 1, 8, 15, and 22 of each 28-day cycle. Dose adjustments were implemented to account for renal dysfunction, and the patient was closely monitored for potential worsening of peripheral neuropathy given his baseline neurologic involvement.

Cardiac management focused on careful volume control. Treatment with furosemide, a loop diuretic, was started at 40 mg twice daily and carefully adjusted to maintain normal fluid balance without causing hypotension. Angiotensin-converting enzyme inhibitors were avoided because of significant orthostatic hypotension. Low-dose carvedilol (3.125 mg twice daily) was prescribed for rate control of atrial fibrillation. Anticoagulation was initiated with apixaban at a reduced dose of 2.5 mg twice daily because of renal dysfunction and the elevated thromboembolic risk associated with atrial fibrillation and cardiac amyloidosis. Supportive management included treating orthostatic hypotension with midodrine, using compression stockings, and counseling on salt and fluid balance. Nephrology services were involved in the ongoing management of progressive renal dysfunction.

2.8. Follow-up and Outcome

The patient was closely monitored throughout therapy. Laboratory evaluation included monthly assessment of serum free light chains and the difference in involved versus uninvolved free light chains, as well as measurement of cardiac biomarkers such as troponin and NT-proBNP. Routine laboratory testing, including complete blood count and comprehensive metabolic panel, was also performed to assess treatment tolerance and organ function. Clinical evaluation at each visit focused on symptoms of treatment toxicity, functional status, and assessment of volume status. Hematologic response was evaluated after cycles 2, 4, and 6 of therapy using established criteria. Cardiac response was evaluated after three and six months of therapy.

An adequate response was not achieved, leading to escalation to a daratumumab-based treatment regimen. Surveillance for disease showed rising free light chain levels and worsening cardiac biomarkers. The patient expired 29 months after treatment initiation due to progressive refractory heart failure and multiple organ failure.



1A: High power view showing extensive amorphous eosinophilic extracellular deposits between myocardial fibers, resulting in compression and atrophy of adjacent cardiomyocytes (H&E X40).; 1B: Cardiac biopsy positive for Cong Red stain.; 1C: Cardiac biopsy revealing characteristic apple-green birefringence positive Congo Red under polarized light

Figure 1 Microscopic examination of cardiac biopsy with amyloidosis

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification):

Amyloidosis, or rather the descriptions of patients consistent with amyloidosis, can be described as early as 1639 by Nicolaus Fontanus in an autopsy of a young patient who had a spleen filled with white stones. The term amyloid was first coined in 1838 by Matthias Schleiden, though in a manner suited for botanical purposes. It was not until 1854 that Rudolf Virchow officially introduced it to medicine. The first case of primary amyloidosis was likely to be reported in 1856 by Samuel Wilks, describing a patient with "lardaceous viscera" in the absence of syphilis, osteomyelitis, or tuberculosis. [6, 7, 8]

The WHO classifies systemic AL Amyloidosis as a clonal plasma cell disorder distinct from multiple myeloma, though both are derived from clonal plasma cell proliferation. [9] It can be further classified as either a clonal plasma cell dyscrasia (the most common) or a lymphoid dyscrasia (much rarer), depending on the underlying bone marrow disease. It is caused by clonal plasma cells or, rarely, by B cells that produce immunoglobulin light chains that are prone to misfolding. These proteins are then deposited into various peripheral tissues as amyloid fibrils, leading to organ damage and/or failure. [10,11] Organs involved typically include the heart (70-80%), kidneys (60-70%), with the nervous system, liver, GI system, and other soft tissues being less common (~20%). Although it remains unclear, risk factors can include preexisting monoclonal gammopathy (such as MGUS), myeloma, and exposure to certain chemical agents (such as Agent Orange used in the Vietnam War). [12]

AL amyloidosis is rare, though it has been increasing, with an incidence rate of 10-17 cases per million person-years, typically occurring in the 6th or 7th decade of life, with a slight male predominance. [12,13] In most cases, AL amyloidosis is a rapidly progressing disease that can cause organ damage/dysfunction. However, symptoms can be non-specific, such as fatigue and weight loss, and are often difficult to diagnose because clinicians give little diagnostic consideration to the disease. Organ-specific symptoms often prompt further evaluation and are what usually lead to the eventual diagnosis of the disease. [11] The prognosis depends on the stage of the disease. Several staging systems have been developed, though most focus on cardiac function (notably the Mayo Clinic 2004 Staging System), with some also evaluating renal function at the time of diagnosis. [12]

3.2. Pathogenesis, Pathophysiology

Amyloidosis is characterized by the uncontrolled proliferation and synthesis of monoclonal immunoglobulin light chains, which commonly deposit in various organs, including the heart, gastrointestinal tract, liver, kidneys, and peripheral nerves. [14,15]

The pathogenesis of AL amyloidosis originates from a small, often indolent, clonal expansion of plasma cells in the bone marrow that overproduce unstable monoclonal free light chains, most frequently of the lambda isotype. [16, 18] These amyloidogenic light chains undergo a cascade of protein unfolding and misfolding, driven by specific variable-region gene mutations, most notably the IGLV1-44 germline segment, which confers a fivefold increase in the odds of dominant cardiac involvement, and by permissive local tissue microenvironments that facilitate nucleation and fibril elongation. [14]

Misfolded monomers form soluble oligomers, which then polymerize into amyloid fibrils with cross-β-sheet structures that accumulate in the extracellular spaces of organs. [16] Organ damage in the heart is mediated by a dual mechanism. The first is mechanical: the progressive accumulation of amyloid fibrils expands the extracellular volume, stiffens the myocardium, and produces the characteristic severe diastolic dysfunction and restrictive physiology. [17,18] The second is direct cardiotoxicity: pre-fibrillar light chain oligomers independently induce high levels of reactive oxygen species, cause mitochondrial dysfunction, and trigger cardiomyocyte apoptosis via p38-MAPK autophosphorylation. [14] This dual mechanism explains why patients with AL amyloidosis exhibit disproportionately elevated cardiac biomarkers and worse survival compared to those with transthyretin amyloidosis, despite similar degrees of myocardial hypertrophy. [11,14]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical, radiology, pathology, Lab, diagnosis, management, and outcome)

The clinical trajectory of our patient aligns closely with the established literature on advanced AL amyloidosis, while simultaneously underscoring the profound challenges of multisystem disease. Consistent with published cohort data, our patient presented with a constellation of non-specific symptoms, including progressive dyspnea, fatigue, bilateral edema, and unintentional weight loss, that evolved over months before a definitive diagnosis was established. [2,4,11]

His echocardiographic findings of severe concentric left ventricular hypertrophy, grade III diastolic dysfunction, and the characteristic "apical sparing" longitudinal strain pattern, coupled with low-voltage electrocardiography in the setting of increased wall thickness, represent the hallmark multimodality imaging phenotype of cardiac amyloidosis consistently described in the literature. [3] The additional finding of grade 1 technetium-99m pyrophosphate uptake appropriately directed the diagnosis toward AL rather than transthyretin amyloidosis, reflecting the established scintigraphic distinction between these two subtypes. [16]

The breadth of extracardiac involvement in our case, nephrotic-range proteinuria with biopsy-confirmed glomerular amyloid deposition, axonal sensorimotor polyneuropathy, autonomic dysfunction, hepatomegaly, and periorbital purpura, mirrors the multisystem nature of AL amyloidosis, which is reported in up to 69% of patients at the time of diagnosis. [14] Because our patient had both severe heart and kidney disease, they were classified as high-risk, matching Mayo Stage III and Boston University Stage IIIb. This subgroup typically has a median survival of just 6 months unless effective treatment is provided. [2]

In contrast to more favorable outcomes reported in early-stage disease, our patient's failure to achieve an adequate hematologic response to CyBorD, followed by disease progression despite daratumumab-based escalation, reflects the therapeutic limitations well-documented in advanced-stage AL amyloidosis. Recent data from the ANDROMEDA trial demonstrated that Dara-CyBorD significantly improves hematologic and organ response rates compared to CyBorD alone; however, patients with Mayo Stage IIIb disease were excluded from that trial, and real-world outcomes in this subgroup remain substantially worse. [5,11]

Our patient's clinical course thus aligns with the literature documenting that once extensive amyloid deposition and direct light-chain cardiotoxicity have occurred, reversal of organ failure remains exceedingly difficult, and survival is markedly shortened. [17]

4. What Have We Learned from This Case?

This case demonstrates several important clinical insights regarding the recognition and management of cardiac AL amyloidosis. One of the most instructive diagnostic lessons is the importance of recognizing discordance between ECG findings and cardiac imaging. In this patient, low-voltage QRS complexes on ECG despite increased left ventricular wall thickness represented a key red flag for infiltrative cardiomyopathy. This should prompt consideration of cardiac amyloidosis early in the process. Similarly, the presence of progressive heart failure symptoms with preserved ejection fraction, arterial arrhythmias, peripheral neuropathy, nephrotic disease, and orthostatic hypotension shows the multisystem pattern that should raise suspicion for systemic amyloidosis disease.

This case reinforces the diagnostic value of multimodal imaging and tissue confirmation. Findings such as a characteristic "apical sparing" pattern on imaging and diffuse late gadolinium enhancement on cardiac MRI provided strong noninvasive evidence of amyloid infiltration. At the same time, endomyocardial biopsy with Congo red stain and mass spectrometry remained essential for definitive subtype identification.

Accurate typing is critical because management strategies differ substantially between AL and transthyretin amyloidosis. The case highlights the need for quick hematologic control of plasma cell clones to reduce amyloid buildup. Despite early initiation of bortezomib therapy, the patient developed progressive disease, reflecting the aggressive condition and the prognostic significance of cardiac biomarker elevation.

Additionally, this case highlights the importance of early recognition of subtle systemic clues and of prompt multidisciplinary evaluation in improving outcomes. For clinicians, maintaining a high index of suspicion in patients with unexplained restrictive cardiomyopathy and multisystem manifestations may allow earlier diagnosis, earlier therapy, and potentially improved survival in this otherwise rapidly progressive disease.

5. Conclusion

This case of advanced systemic AL amyloidosis with severe cardiac, renal, hepatic, and neurological involvement illustrates the devastating natural history of the disease when diagnosis is delayed, and organ damage is already extensive at presentation. We report this case to emphasize to the medical community that a high index of clinical suspicion is paramount when encountering unexplained heart failure with preserved ejection fraction, nephrotic syndrome, peripheral neuropathy, or autonomic dysfunction, particularly in older adults with a background of plasma cell dyscrasia.

The value of this report lies in its reminder that the diagnostic "red flags" of AL amyloidosis are often present long before a definitive diagnosis is made. Routine screening for monoclonal proteins in atypical heart failure, prompt tissue biopsy with amyloid typing, and early multidisciplinary intervention remain the most critical determinants of outcome in this complex and frequently fatal systemic disease.

Compliance with ethical standards

Acknowledgments

Special thanks to Angela Zavaro and Armando Piedra for their assistance in reviewing the final manuscript. Additionally, we appreciate the assistance of Grammarly's language editor and the Claude Sonnet 4.6 program, which provided valuable writing support by identifying and correcting errors in grammar, spelling, punctuation, and writing style, ultimately enhancing the manuscript.

Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

References

[1] Merlini, G., Dispenzieri, A., Sanchorawala, V., et al. Systemic immunoglobulin light chain amyloidosis. Nature Reviews Disease Primers, 4, 38 (2018).

[2] Baker, K. R. Light Chain Amyloidosis: Epidemiology, Staging, and Prognostication. Methodist DeBakey Cardiovascular Journal, 18(2), 27–35 (2022).

[3] Llerena-Velastegui, J., et al. Advances in the Diagnosis and Management of Cardiac Amyloidosis: A Literature Review. Cureus, 16(8), e67568 (2024).

[4] Adrogue, H. E. Amyloidosis of the Heart and Kidney. Methodist DeBakey Cardiovascular Journal, 18(4), 27–33 (2022).

[5] Kastritis, E., Palladini, G., Minnema, M. C., et al. Daratumumab-Based Treatment for Immunoglobulin Light-Chain Amyloidosis (ANDROMEDA). New England Journal of Medicine, 385(1), 46–58 (2021).

[6] Kyle RA. Amyloidosis: a brief history. Amyloid. 2011 Jun 1;18(sup1):6-7.

[7] Muchtar E, Buadi FK, Dispenzieri A, Gertz MA. Immunoglobulin light-chain amyloidosis: from basics to new developments in diagnosis, prognosis and therapy. Acta haematologica. 2016 Mar 1;135(3):172-90.

[8] Giorgetti A, Pucci A, Aimo A. A brief history of amyloidosis. In Cardiac Amyloidosis: Diagnosis and Treatment 2024 Mar 28 (pp. 13-21). Cham: Springer Nature Switzerland.

[9] Fend F, Dogan A, Cook JR. Plasma cell neoplasms and related entities—evolution in diagnosis and classification. Virchows Archiv. 2023 Jan;482(1):163-77.

[10] Minnema M, Schönland S. Systemic Light Chain Amyloidosis. The EBMT Handbook: Hematopoietic Cell Transplantation and Cellular Therapies. 2024 Apr 11:751-5.

[11] Shams P, Ahmed I, Cardiac Amyloidosis. StatPearls. Accessed 3/25/2026. From https://www.statpearls.com/point-of-care/144241

[12] Sanchorawala V. Systemic light chain amyloidosis. New England Journal of Medicine. 2024 Jun 27;390(24):2295-307

[13] Laires PA, Fang S, Evans J, Thompson J, Gaur A, Staruk B, Gupta A, Manwani R. Incidence and prevalence of light chain amyloidosis in the United States in 2019–2021 using Optum EHR data. Scientific Reports. 2025 Jul 11;15(1):25149.

[14] Ikura, H., Endo, J., Kitakata, H., et al. Molecular Mechanism of Pathogenesis and Treatment Strategies for AL Amyloidosis. International Journal of Molecular Sciences, 23(11), 6336 (2022).

[15] Juneja R, Pati HP. Approach to the diagnosis of amyloidosis. Indian Journal of Hematology and Blood Transfusion. 2020 Apr;36(2):246-53.

[16] Morè, S., Manieri, V. M., Corvatta, L., et al. AL amyloidosis: an overview on diagnosis, staging system, and treatment. Frontiers in Hematology, 3, 1378451 (2024).

[17] Morfino, P., Aimo, A., Franzini, M., et al. Pathophysiology of cardiac amyloidosis. Heart Failure Clinics, 20(2), 145–155 (2024).

[18] Bansal M. Primary amyloidosis. PathologyOutlines.com website. https://www.pathologyoutlines.com/topic/lymphomaprimaryamyloid.html. Accessed March 25th, 2026.