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Fulminant Cardiac Light-Chain (AL) Amyloidosis: A Case of Rapid Multiorgan Collapse and the Diagnostic Urgency of the Difference Between Involved and Uninvolved Free Light Chains (dFLC)

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Abstract

Light-chain (AL) amyloidosis is a rare systemic disease that can affect multiple organs. Cardiac involvement is common and frequently determines prognosis and survival. Cardiac AL amyloidosis is a progressive infiltrative cardiomyopathy. We report an unusually rapid course of cardiac AL amyloidosis resulting in severe multiorgan failure. This case illustrates the diagnostic and therapeutic challenges associated with a rapidly progressive disease course. We present the case of a 54-year-old man admitted with decompensated heart failure. Physical examination demonstrated significant volume overload. Laboratory evaluation of blood and urine was indicative of an active monoclonal plasma cell disorder. Echocardiography revealed a left ventricular ejection fraction of 10-15% and severe left ventricular hypertrophy. Review of prior echocardiograms demonstrated a preserved ejection fraction of 50-55% four months earlier. Nuclear medicine single-photon emission computed tomography combined with computed tomography (NM SPECT-CT) demonstrated cardiomegaly and findings inconsistent with transthyretin amyloidosis, with a heart-to-contralateral lung (H/CL) uptake ratio of 1.17. CT of the abdomen revealed enlargement of multiple organs, including the heart, liver, spleen, and kidneys. The patient underwent multidisciplinary evaluation by cardiology, nephrology, gastroenterology, and hematology-oncology. A bone marrow biopsy was performed, and combination chemotherapy was planned. However, the patient developed recurrent tachyarrhythmias and bradyarrhythmias followed by cardiac arrest and died before treatment could be initiated. The patient progressed from preserved ejection fraction to death within four months. Extremely elevated immunoglobulin light chain levels likely contributed to rapid cardiac deterioration through both amyloid deposition and direct cytotoxic effects. Disease staging using biomarkers including brain natriuretic peptide, troponins, and the difference in free light chain (dFLC) values is helpful in recognizing aggressive AL amyloidosis biology and treatment urgency. The recurrent arrhythmias likely reflected advanced amyloid infiltration of the cardiac conduction system. This fatal and complex case underscores the importance of interdisciplinary management and prompt initiation of plasma cell-directed therapy.

Categories: Cardiology
Keywords: al cardiac amyloidosis, cardiology imaging, heart failure with reduced ejection fraction, infiltrative cardiomyopathy, left ventricular hypertrophy (lvh)

Introduction

Amyloid disease can be classified into three common subtypes: light-chain (AL) amyloidosis, transthyretin (ATTR) amyloidosis, and serum amyloid A (AA) amyloidosis. AL amyloidosis is rare and affects about 10 per million/year [1]. All three subtypes are classified as systemic amyloidosis and can involve several organ systems. The heart is a frequently affected organ in AL amyloidosis [2]. Cardiac AL amyloidosis is a progressive infiltrative cardiomyopathy. Cardiac involvement contributes significantly to the overall prognosis. It is one of the most important determinants of survival and the leading cause of death in systemic AL amyloidosis [3]. Recognizing this disease and its cardiac involvement early is crucial, particularly in patients with left ventricular wall thickening or unexplained heart failure. We report an unusually rapid course of cardiac amyloidosis with severe multiorgan failure. This case illustrates the diagnostic and therapeutic challenges encountered in the setting of a very rapidly progressive disease course. This case underscores how swiftly cardiac AL amyloidosis can evolve from initial presentation to catastrophic multiorgan failure, highlighting the critical need for early recognition and timely intervention.

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Case Presentation

A 54-year-old man with atrial fibrillation, stage 3 chronic kidney disease (CKD), pulmonary sarcoidosis,

heart failure with reduced ejection fraction, and liver cirrhosis presented with profound bradycardia around 20-30 beats per minute and hypoxia with a peripheral oxygen saturation (SpO_2) of 60% on room air. He had severe volume overload with large ascites, pleural effusions, and a moderate pericardial effusion.

Laboratory workup revealed moderate anemia, an elevated brain natriuretic peptide (BNP), elevated troponins, and kidney function impairment. A serum protein electrophoresis (SPEP) and serum immunofixation electrophoresis (SIFE) revealed an M-spike pattern with an elevated immunoglobulin G fraction. Further quantitative analysis revealed markedly increased free lambda light chains and a profoundly suppressed kappa/lambda ratio with a large proportion of free lambda light chains (Table *1*). Urine protein electrophoresis confirmed an M-spike. These abnormalities were strongly suggestive of an active monoclonal plasma cell disorder.

Laboratory Test	Result	Reference Range
Hemoglobin	10.6 g/dL	14.0-18.0 g/dL
Brain natriuretic peptide (BNP)	2,349 pg/mL	2-100 pg/mL
High-sensitivity troponin I	613 and 673 ng/L	≤57 ng/L
Creatinine	3.5 mg/dL	0.50-1.40 mg/dL
Estimated glomerular filtration rate (GFR)	20 mL/min/1.73 m²	≥60 mL/min/1.73 m^2
Serum protein electrophoresis (SPEP)	M spike of 1.7 g/dL and a second small M-spike of 0.2 g/dL	No M-spike detected
Serum immunofixation electrophoresis (SIFE)	Biclonal IgG protein with lambda specificity	No monoclonal protein detected
Immunoglobulin G fraction	3,609 mg/dL	603-1,613 mg/dL
Free lambda light chains	1,260 mg/L	5.7-26.3 mg/L
Kappa/lambda ratio	0.02	0.26-1.65
Urine protein electrophoresis (UPEP)	M spike of 22.5 mg/dL	No M-spike detected

TABLE 1: Results of laboratory tests suggesting an active monoclonal plasma cell disorder

A review of serial echocardiography imaging studies revealed a rapid decline in left ventricular ejection fraction (LVEF). An echocardiogram four months prior showed an LVEF of 50-55%. An echocardiogram performed two months ago demonstrated an LVEF of 35%. The echocardiogram at presentation revealed an LVEF of 10-15% with massive concentric LV hypertrophy (LVH). His diastolic left ventricular posterior wall diameter (LVPW) was 1.99 cm (Figure *1*).

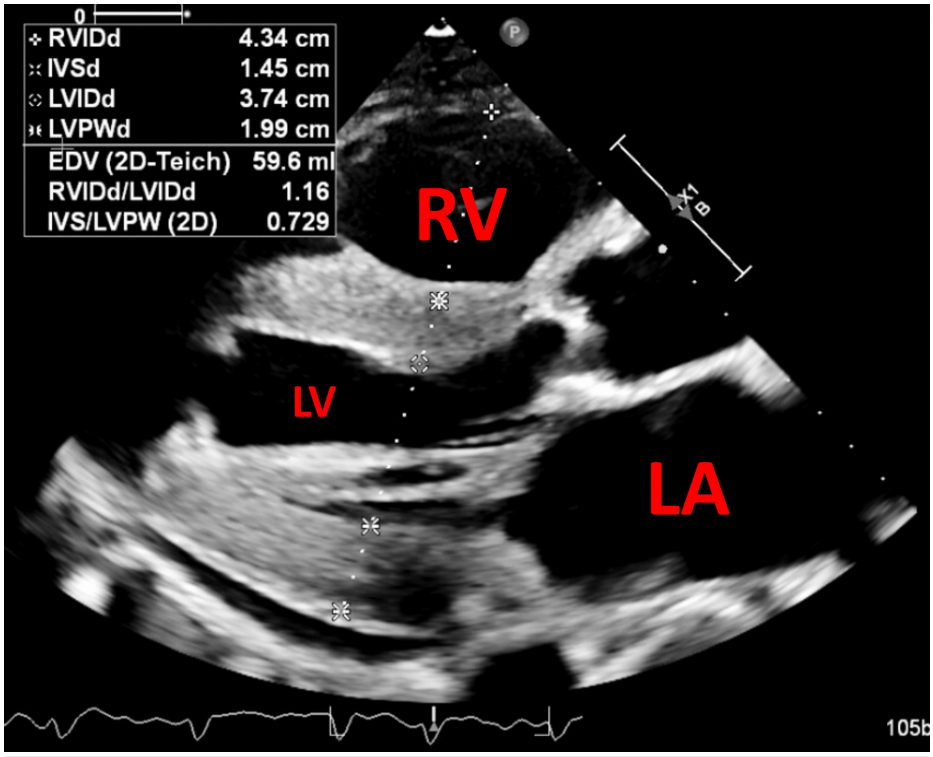


FIGURE 1: Parasternal long axis view of transthoracic echocardiogram (TTE) showing severe hypertrophy with diastolic left ventricular posterior wall thickness (LVPWd) of 1.99 cm.

LA: Left atrium, LV: Left ventricle, RV: Right ventricle, EDV (2D-Teich): End-diastolic volume calculated by Teichholz formula, IVSd: Interventricular septal thickness at end-diastole, IVS/LVPW (2D): Interventricular septum to left ventricular posterior wall thickness ratio, LVIDd: Left ventricular internal diameter at end-diastole, RVIDd: Right ventricular internal diameter at end-diastole

Nuclear medicine single-photon emission computed tomography combined with computed tomography (NM SPECT-CT) imaging demonstrated cardiomegaly with a heart-to-contralateral lung (H/CL) uptake ratio of 1.17. This was not consistent with transthyretin amyloidosis (Figure *2*).

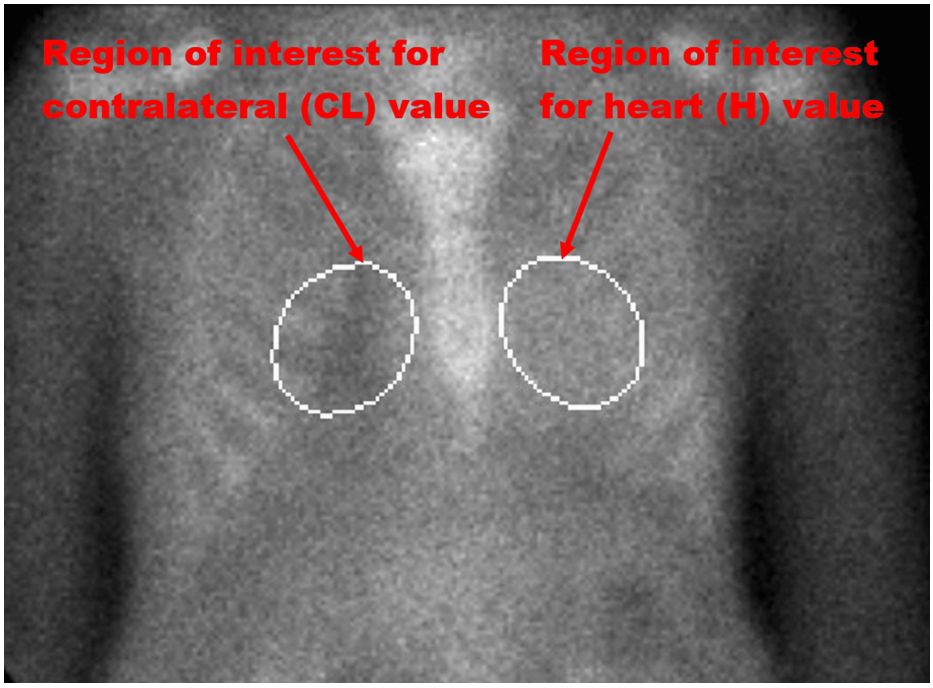


FIGURE 2: Planar scintigram derived from nuclear medicine single-photon emission computed tomography combined with computed tomography (NM SPECT-CT) imaging. The oval regions of interest over the heart (right circle) and the contralateral lung (left circle) are demonstrating a heart-to-contralateral lung (H/CL) uptake ratio of 1.17.

Further review of prior imaging included a cardiac magnetic resonance imaging (MRI) with and without gadolinium contrast. This study was performed four years prior to evaluate for cardiac sarcoidosis. The study was prompted by new-onset atrial fibrillation with rapid ventricular response in the setting of a long-standing history of pulmonary sarcoidosis with persistent mediastinal lymphadenopathy. The diagnosis of sarcoidosis had been established 15 years prior to this cardiac MRI, when bronchoscopy with transbronchial biopsies revealed noncaseating granulomatous disease consistent with sarcoidosis. The patient had been treated with systemic corticosteroids during the course of the disease. Given the known association between cardiac sarcoidosis and atrial arrhythmias, the consulting cardiologist pursued advanced cardiac imaging to assess for myocardial involvement. Cardiac MRI demonstrated mild concentric left ventricular hypertrophy with a maximum wall thickness of 1.2 cm and a preserved LVEF of 61%. Late gadolinium enhancement sequences revealed no evidence of myocardial fibrosis or infiltrative disease. Overall, there was no MRI evidence of cardiac sarcoidosis at that time.

The patient was diagnosed with new-onset liver cirrhosis approximately four months prior to this presentation. This was revealed by abdominal ultrasonography performed in the setting of abdominal distension, new-onset ascites, and mild liver function test abnormalities. Laboratory evaluation demonstrated elevated liver injury markers, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase, along with a mildly decreased albumin and prolonged coagulation markers (Table *2*). Ammonia was within normal limits. An extensive laboratory workup was performed to evaluate for alternative etiologies of liver cirrhosis. Viral hepatitis serologies were all negative. Autoimmune markers, including antinuclear antibody (ANA), double-stranded DNA (dsDNA) antibody, smooth muscle antibody (SMA), and anti-mitochondrial antibody (AMA), were all negative, arguing against autoimmune hepatitis and primary biliary cholangitis. Ceruloplasmin was normal, excluding Wilson disease. Iron studies with a low serum iron, low iron saturation, and low-normal ferritin revealed a pattern inconsistent with hereditary hemochromatosis and instead suggestive of iron deficiency (Table *2*).

Test	Result	Reference Range
Liver Injury Markers		
Aspartate aminotransferase (AST)	87 U/L (High)	13-39 U/L
Alanine aminotransferase (ALT)	136 U/L (High)	7-52 U/L
Alkaline phosphatase	133 U/L (High)	30-105 U/L
Liver Synthetic Function		
Albumin	3.2 g/dL (Low)	3.5-5.7 g/dL
Bilirubin, total	1.0 mg/dL	0.3-1.0 mg/dL
Prothrombin time (PT)	14.0 sec (High)	9.3-11.5 sec
International normalized ratio (INR)	1.37 (High)	0.90-1.10
Ammonia	51 µmol/L	16-54 µmol/L
Viral Hepatitis Serologies		
Hepatitis A IgM antibody	Nonreactive	Nonreactive
Hepatitis B surface antigen	Nonreactive	Nonreactive
Hepatitis B core IgM antibody	Nonreactive	Nonreactive
Hepatitis C antibody	Nonreactive	Nonreactive
Autoimmune Markers		
Antinuclear antibody (ANA) ratio	0.40	<0.70
Double-stranded DNA (dsDNA) antibody	2.5 IU/mL	0.0-9.9 IU/mL
Smooth muscle antibody (SMA)	Negative	Negative
Anti-mitochondrial antibody (AMA)	Negative	Negative
Anti-mitochondrial antibody level	12.6	≤20 (Negative)
Metabolic Markers		
Ceruloplasmin	43.0 mg/dL	18.0-58.0 mg/dL
Iron Studies		
Iron	30 µg/dL (Low)	65-165 µg/dL
Total iron-binding capacity (TIBC)	405 µg/dL	246-419 µg/dL
Iron saturation (%)	7% (Low)	20-50%
Unsaturated iron-binding capacity (UIBC)	375 µg/dL	150-375 µg/dL
Ferritin	16.4 ng/mL	10.0-300.0 ng/mL

TABLE 2: Laboratory evaluation for liver cirrhosis etiology

Ascitic fluid analysis demonstrated a serum-ascites albumin gradient (SAAG) of 2.0 g/dL (serum albumin 3.4 g/dL minus ascitic fluid albumin 1.4 g/dL), indicating portal hypertension. Notably, the ascitic fluid total protein was elevated at 3.5 g/dL. This combination of a high SAAG with high ascitic fluid protein is characteristic of a cardiac etiology for ascites, as hepatic venous congestion from elevated right-sided filling pressures produces portal hypertension while preserving sinusoidal permeability to protein. This pattern favored a cardiac contribution to the patient's ascites. The fluid white blood cell count was elevated at 613/µL with a mononuclear cell predominance (90.9%), while the absolute polymorphonuclear (PMN) cell count was 60/µL, below the threshold of 250/µL typically used to diagnose spontaneous bacterial peritonitis. Cytology revealed no malignant cells (Table 3). A liver biopsy was not performed.

Test	Result	Reference Range
Serum albumin (day of paracentesis)	3.4 g/dL	3.1-4.7 g/dL
Ascitic fluid albumin	1.4 g/dL	Transudate: >1.1 g/dL; Exudate: <1.1 g/dL
Serum-ascites albumin gradient (SAAG)	2.0 g/dL	≥1.1 g/dL: portal hypertension
Ascitic fluid total protein	3.5 g/dL	Transudate: <3.0 g/dL; Exudate: >3.0 g/dL
WBC in ascitic fluid	613 /µL	≤10 /µL
RBC in ascitic fluid	2,000 /µL	≤0 /µL
Nucleated cells in ascitic fluid	661 /µL	≤10 /µL
Mononuclear cells (%) in ascitic fluid	90.9%	Not reported
Mononuclear cells (absolute count) in ascitic fluid	560.00 /µL	≤0.00 /µL
Polymorphonuclear (PMN) cells (%) in ascitic fluid	9.1%	0.0%
PMN cells (absolute count) in ascitic fluid	60.00 /µL	≤0.00 /µL
Cytology	No malignant cells identified	No malignant cells identified

TABLE 3: Ascitic fluid analysis with serum-ascites albumin gradient (SAAG)

The patient had undergone esophagogastroduodenoscopy (EGD) one month prior to the current presentation in the setting of liver cirrhosis and iron deficiency anemia. Gastric biopsies from the gastric antrum and gastric body obtained during this procedure both confirmed amyloid deposition. A current CT of the abdomen and pelvis without contrast revealed hepatomegaly, splenomegaly, bilaterally enlarged kidneys, cardiomegaly, and volume overload with anasarca, bilateral pleural effusions, and ascites (Figures *3*-*6*). Further review of imaging revealed that all of these findings were already present on a CT abdomen four months prior to the current presentation.

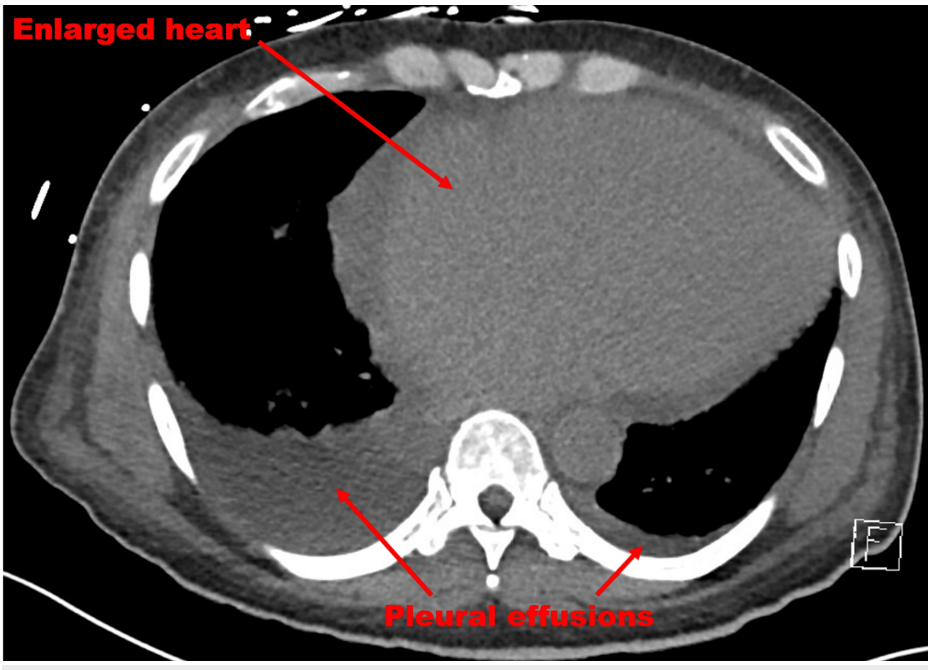


FIGURE 3: Axial non-contrast computed tomography (CT) of the abdomen and pelvis demonstrating significant cardiomegaly with bilateral pleural effusions.

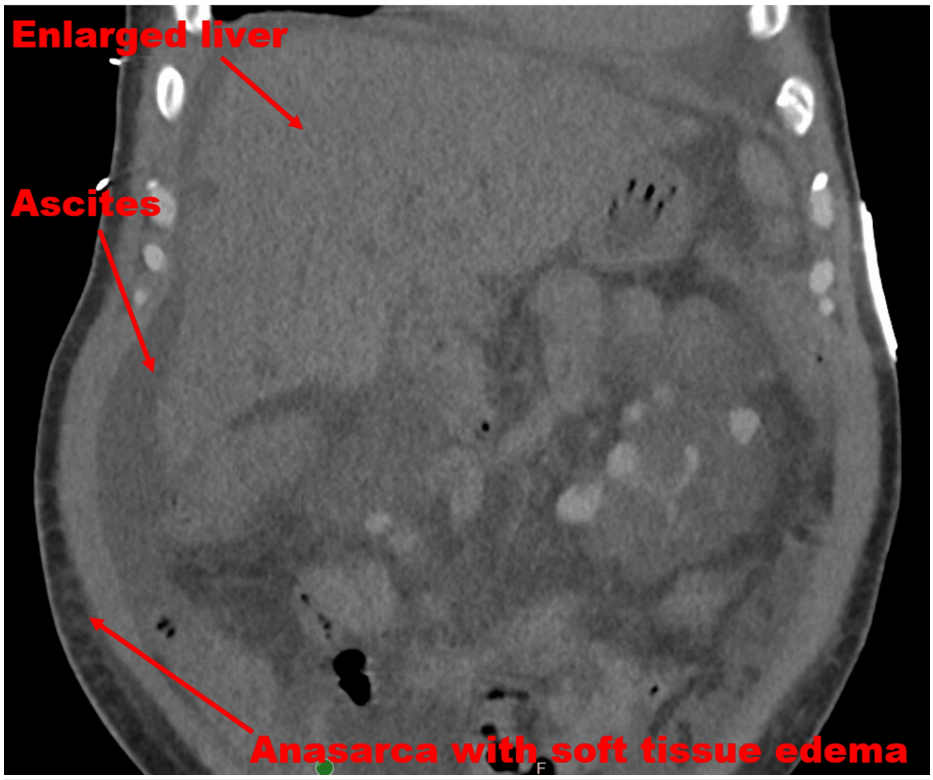


FIGURE 4: Coronal non-contrast computed tomography (CT) of the abdomen and pelvis demonstrating marked hepatomegaly with diffuse ascites and subcutaneous edema consistent with anasarca.

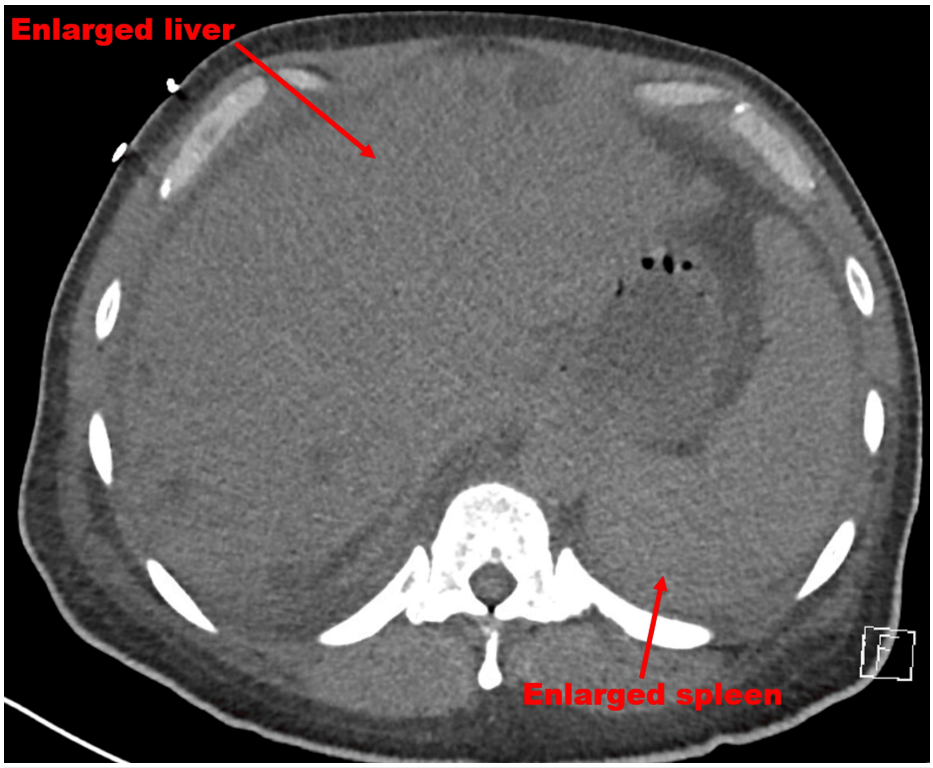


FIGURE 5: Axial non-contrast computed tomography (CT) of the abdomen and pelvis demonstrating hepatomegaly and splenomegaly.

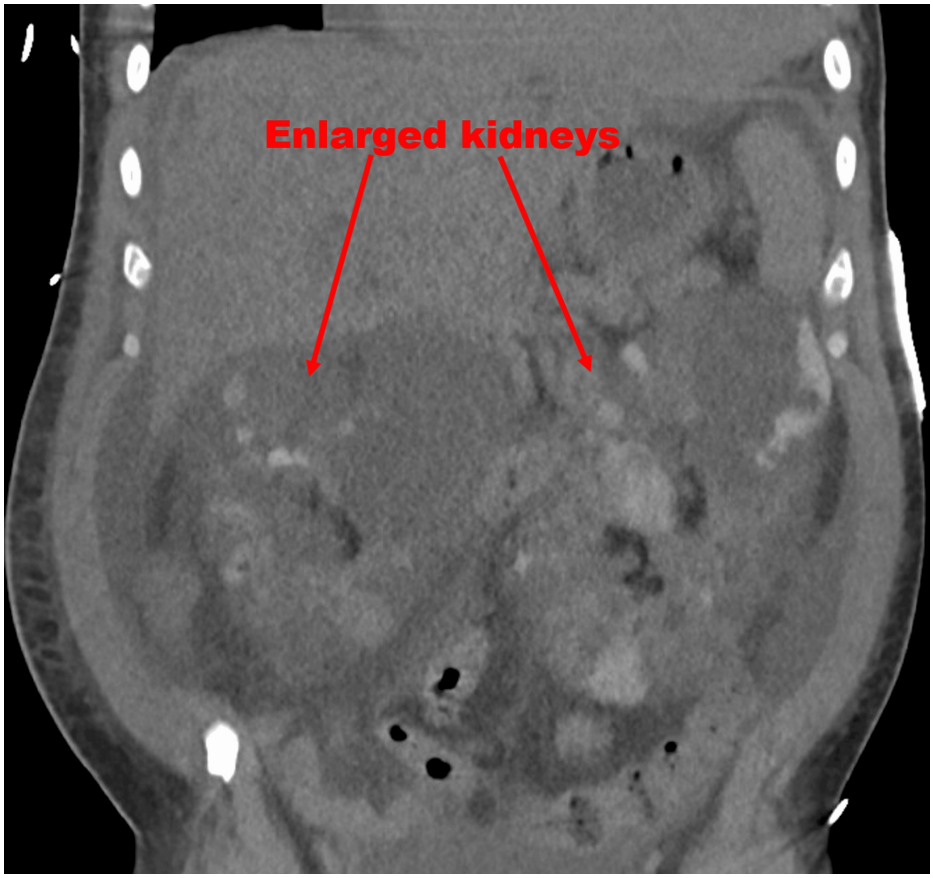


FIGURE 6: Axial non-contrast computed tomography (CT) of the abdomen and pelvis demonstrating bilateral renal enlargement.

Serologic and urine testing was consistent with monoclonal gammopathy. The differential diagnosis included AL amyloidosis and multiple myeloma. Suspicion for amyloidosis secondary to plasma cell dyscrasia was high. Nephrology was concerned about advanced kidney involvement secondary to renal amyloidosis with CKD.

The patient was seen by cardiology, who referred him to an advanced heart failure center. The patient was also evaluated by hematology-oncology. They ordered a bone marrow biopsy and planned to initiate treatment with a combination chemotherapy regimen of daratumumab plus cyclophosphamide, bortezomib, and dexamethasone (daratumumab-CyBorD) for his primary monoclonal gammopathy. They were considering referral for possible stem cell transplant in the future. A CT-guided bone marrow biopsy was performed six days prior to his death. While the biopsy results were still pending, the patient developed alternating tachyarrhythmic atrial fibrillation and episodes of profound bradycardia, culminating in cardiac arrest. After successful return of spontaneous circulation (ROSC), he required escalating vasopressors. Despite receiving treatment, the patient's condition deteriorated with ongoing multiorgan failure. Cardiology, nephrology, and gastroenterology recommended supportive care due to poor prognosis. The patient was transitioned to comfort care and passed away shortly thereafter. A timeline of the patient's presentation and diagnostic workup is provided in the figure below (Figure 7). A current cardiac MRI was scheduled. However, the patient passed away before it could be performed.

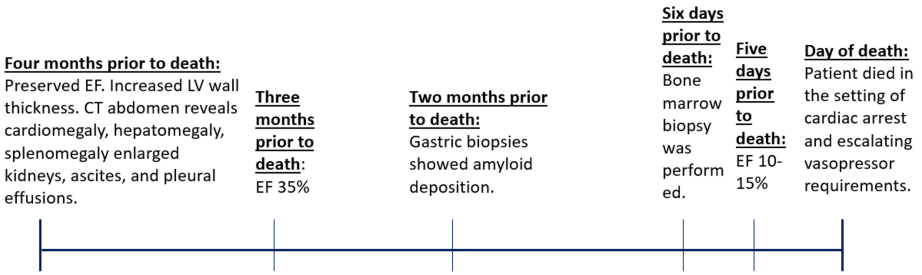


FIGURE 7: Timeline summarizing the patient's presentation and diagnostic workup

CT: Computed tomography, EF: Ejection fraction, LV: Left ventricular

Discussion

Pathophysiology of AL amyloidosis

AL amyloidosis is the most aggressive type of amyloidosis. The pathomechanism involves the deposition of immunoglobulin light chains in the extracellular space. The light chains misfold and form oligomers and insoluble amyloid fibrils, which are deposited in various organs. Organ dysfunction is mediated by architectural disruption from the amyloid deposits and direct cytotoxic effects. The deposits commonly occur in the kidneys, heart, peripheral nervous system, and liver [3]. Prior research has shown that circulating soluble amyloidogenic light chains are directly toxic to cardiomyocytes independent of amyloid fibril deposition [4].

Diagnostic considerations and missed opportunities

The diagnosis of amyloidosis is often delayed. It is crucial to recognize that hypertrophy and organomegalies may be a sign of amyloidosis caused by extracellular deposits. Increased left ventricular wall thickness by amyloid infiltration is a hallmark feature of cardiac amyloidosis [2]. Hepatomegaly, which was observed in our patient, is the most frequent clinical feature of hepatic amyloidosis [5]. Further review of the patient chart showed that his transthoracic echocardiogram four months prior to diagnosis demonstrated an LVPWd of 1.7 cm. The 2022 American Heart Association/American College of Cardiology/Heart Failure Society of America (AHA/ACC/HFSA) Heart Failure Guidelines suggest that a left ventricular wall thickness of 14 mm or above in conjunction with fatigue, dyspnea, edema, or heart failure with preserved ejection fraction (HFpEF) should trigger consideration of workup for amyloidosis [6]. The hepatomegaly, cardiomegaly, renal enlargement, and splenomegaly were already present on a CT scan three months prior to his death. The multiorgan involvement is consistent with systemic amyloid infiltration rather than isolated cardiac disease, and gastrointestinal biopsy confirmation of amyloid deposition provided definitive histopathological evidence of systemic involvement.

Staging, biomarkers, and the role of difference in free light chain (dFLC)

The elevation of BNP and troponin further suggested myocardial involvement. Troponin elevation is an indicator of myocardial injury from the direct toxic effect of light chains, microvascular ischemia mediated by amyloid infiltrates, and diastolic dysfunction [7]. The dFLC value was dFLC = 1260.5 - 28.8 = 1231.7 mg/L. A dFLC value of at least 180 mg/L places a patient in the high-risk AL amyloidosis category [8]. A dFLC of >1000 mg/L indicates an extremely high clonal light chain production and aggressive disease biology. Based on the Mayo 2004 Staging System with European Modifications [9], the patient was in stage IIIB, and according to the Mayo 2012 Staging System [8], the patient was in stage IV - the most severe stage in both systems. These staging systems incorporate troponin, BNP, and dFLC values. This indicates a very high tumor burden and aggressive AL biology, especially when cardiac involvement is present.

The patient had CKD, which increases both absolute kappa and lambda values due to decreased renal elimination. Interpretation of the reported absolute values is difficult in such patients, as they can have a high baseline light chain level. Similarly, CKD can elevate troponin and BNP levels independent of cardiac injury or volume overload. However, the dFLC is still relatively robust compared to those absolute values and isolates the tumor-related monoclonal excess, making it a measure of true clonal plasma cell activity [10].

Clinical course and arrhythmic complications

This case represents an unusually fulminant variant of AL amyloidosis with catastrophic cardiac decline. AL

amyloidosis is known for rapid cardiac deterioration, but an LVEF drop from 55% to 10-15% within four months is exceptionally rare [11]. This atypically rapid deterioration was likely associated with the severe elevation of cardiotoxic lambda light chains.

The patient experienced recurrent tachyarrhythmias and bradyarrhythmias culminating in cardiac arrest, vasopressor-dependent shock, and irreversible multiorgan failure. The severe bradyarrhythmias alternating with atrial fibrillation are associated with advanced cardiac infiltration of the conduction system with amyloid deposits. Cardiac amyloid deposits may distort the anatomy of the sinoatrial (SA) node, atrioventricular (AV) node, and bundle branches. Atrial fibrillation is the most common arrhythmia in cardiac amyloidosis [12]. Infranodal conduction system disease as evidenced by His-ventricular (HV) interval prolongation is a common conduction abnormality in cardiac amyloidosis [13]. Previous ECGs of this patient showed a first-degree AV block and intraventricular conduction delay with a QRS of 162 ms. It is thought that cytotoxic effects result in diastolic dysfunction, which contributes to left atrial enlargement and atrial fibrillation. The disruption of the myocardial architecture and infiltration of the heart's sympathetic fibers may precipitate atrial and ventricular arrhythmias [12].

Treatment implications and key takeaways

In AL amyloidosis, the key element of treatment is to terminate the production of further toxic light chains [2]. Prior to initiation of daratumumab-CyBorD therapy, this patient deteriorated into refractory cardiogenic shock. This case underscores the need for early recognition and rapid initiation of plasma cell-directed therapy, particularly when free lambda chain levels exceed 1,000 mg/L and cardiac involvement is present. It demonstrates how delays in treatment can result in rapid progression of cardiac disease and culminate in death within a few months.

This case report underscores that dFLC, alongside cardiac biomarkers such as troponin and BNP, can help gauge the urgency of treatment initiation. These are immediately actionable metrics in patients with CKD. They enable members of a multidisciplinary team - including hematologists, cardiologists, nephrologists, and intensivists - to more readily recognize high-risk disease and facilitate close collaboration with cardiac specialists when dFLC values signal rapidly progressive AL amyloidosis. This case suggests that patients with dFLC values exceeding 1000 mg/L may experience fulminant cardiac deterioration and death within weeks to months. Clinicians should be aware that in such cases, plasma cell-directed therapy must be initiated as expeditiously as possible to mitigate further cardiotoxic light chain exposure and prevent cardiac death.

Conclusions

This case represents an exceptionally aggressive and rapidly progressive form of systemic amyloidosis with severe cardiac involvement and multiorgan failure. The patient's dFLC of 1,231.7 mg/L reflected an exceptionally high clonal burden. It illustrates the importance of maintaining a high index of suspicion for amyloidosis in patients with unexplained organomegaly, progressive heart failure, or left ventricular hypertrophy of unclear etiology.

This case reinforces that AL amyloidosis with dFLC values exceeding 1,000 mg/L and cardiac involvement is a medical emergency in which time to treatment initiation is a key determinant of outcome. It illustrates that delays of even weeks may prove fatal. Routine measurement of dFLC, troponin, and BNP values alongside absolute free light chain values would enhance interpretation of monoclonal light chain testing and facilitate quick risk stratification with staging. It would alert multidisciplinary teams to the urgency of treatment initiation and the need for close collaboration with cardiac specialists. Early recognition, prompt staging with cardiac biomarkers and dFLC, and immediate initiation of plasma cell-directed therapy are crucial in patients with advanced cardiac amyloidosis.

Cardiac AL amyloidosis can be a rapidly lethal diagnosis in medicine when recognition is delayed. This case serves as a stark reminder that the window for meaningful intervention is narrow, and that a heightened clinical awareness, combined with swift multidisciplinary action, may be the difference between life and death.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Jasmin K. Bhullar

Acquisition, analysis, or interpretation of data: Jasmin K. Bhullar

Drafting of the manuscript: Jasmin K. Bhullar

Critical review of the manuscript for important intellectual content: Jasmin K. Bhullar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received 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 have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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