Journal of Cardiovascular Emergencies 2026;12(3):140-146
DOI: 10.2478/jce-2026-0009

CASE REPORT

When Chronic Inflammation Reaches the Heart: Systemic AA Amyloidosis with Cardiac Involvement in Long-Standing Ankylosing Spondylitis

Nicoleta Crăciun-Ciorba[1,2], Adina Rotari[3], Marius Ciorba[2,4]

[1] Department of Internal Medicine II, Faculty of Medicine, George Emil Palade University of Medicine, Pharmacy, Science and Technology, Târgu Mureș, Romania
[2] County Emergency Clinical Hospital, Târgu Mureș, Romania
[3] Department of Cardiology, Institute for Cardiovascular Diseases and Heart Transplantation, Târgu Mureș, Romania
[4] Department of Internal Medicine I, Faculty of Medicine, George Emil Palade University of Medicine, Pharmacy, Science and Technology, Târgu Mureș, Romania

ABSTRACT

Introduction: Systemic amyloidosis (SA) comprises a heterogeneous group of disorders characterized by extracellular deposition of misfolded protein fibrils, leading to progressive organ dysfunction. Amyloid A (AA) amyloidosis is a systemic disorder that may involve multiple organs, including the gastrointestinal tract, heart, and serosal surfaces. **Case presentation:** We report a middle-aged man with long-standing ankylosing spondylitis who presented with progressive dyspnea and marked functional decline. Laboratory evaluation demonstrated severe hypoalbuminemia, chronic kidney disease with severe albuminuria, and markedly elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) despite only mild high-sensitivity troponin elevation. Electrocardiography showed low QRS voltage with a pseudoinfarction pattern. Transthoracic echocardiography revealed concentric left ventricular hypertrophy, mildly reduced ejection fraction, an infiltrative myocardial appearance, and severely impaired global longitudinal strain disproportionate to the degree of systolic dysfunction. Gastrointestinal biopsies demonstrated Congo red-positive amyloid deposits with positive amyloid A immunostaining, confirming systemic AA amyloidosis. **Conclusions:** This case illustrates systemic AA amyloidosis as a rare but devastating complication of long-standing, poorly controlled ankylosing spondylitis. The combination of dyspnea with large serosal effusions, markedly elevated NT-proBNP disproportionate to troponin elevation, low-voltage electrocardiography, and left ventricular hypertrophy with severely impaired global longitudinal strain should raise suspicion for cardiac amyloidosis in patients with chronic inflammatory diseases and prompt timely diagnostic evaluation.

Keywords: systemic amyloidosis, ankylosing spondylitis, AA amyloidosis, amyloid transthyretin amyloidosis

ARTICLE HISTORY

Received: February 28, 2026
Accepted: July 6, 2026

CORRESPONDENCE

Nicoleta Crăciun-Ciorba
Email: nicoleta.craciun-ciorba@umfst.ro

INTRODUCTION

Systemic amyloidosis comprises a heterogeneous group of disorders characterized by extracellular deposition of misfolded protein fibrils, leading to progressive organ dysfunction and increased mortality. Serum amyloid A (AA) amyloidosis, also known as secondary amyloidosis, results from the deposition of fibrils derived from serum amyloid A (SAA), an acute-phase reactant synthesized by the liver in response to chronic inflammatory stimuli. Sustained elevation of circulating SAA is essential for the development of AA amyloidosis. However, only a subset of patients with long-standing inflammatory diseases ultimately develops clinically significant amyloid deposition, suggesting the involvement of additional genetic and disease-related modifying factors.[1–4]

AA amyloidosis most frequently complicates chronic inflammatory conditions such as rheumatoid arthritis, spondyloarthritis, autoinflammatory syndromes, chronic infections, and inflammatory bowel disease. Although its incidence has declined with the widespread use of effective anti-inflammatory and biologic therapies, AA amyloidosis remains a severe and potentially life-threatening complication in patients with prolonged or inadequately controlled inflammation.[1,2,5] Renal involvement is the most common manifestation and often dominates the clinical presentation. However, AA amyloidosis is a systemic disease that may affect multiple organs, including the gastrointestinal tract, heart, and serosal surfaces.[6–8]

Cardiac involvement in AA amyloidosis is considerably less frequent than in amyloid light-chain (AL) amyloidosis or amyloid transthyretin (ATTR) amyloidosis but is associated with adverse prognosis when present. It typically manifests as restrictive cardiomyopathy with preserved ejection fraction, myocardial wall thickening, infiltrative echocardiographic features, low-voltage electrocardiographic patterns, and pericardial effusion.[4,6,7,9] Gastrointestinal involvement may present with nonspecific symptoms such as chronic diarrhea, malabsorption, weight loss, and cachexia, frequently contributing to diagnostic delay.[8,10,11] Serosal involvement, including pleural effusions and ascites, further reflects advanced systemic disease.

Definitive diagnosis of AA amyloidosis requires histological confirmation of amyloid deposits with Congo red staining and accurate amyloid typing. Identification and sustained control of the underlying inflammatory driver represent the cornerstone of management and may stabilize organ function or slow disease progression.[7,12–14]

CASE PRESENTATION

INITIAL PRESENTATION

A 60-year-old male patient was admitted to the emergency department with progressive dyspnea, epigastric pain, nausea, recurrent vomiting, chronic diarrhea and severe unintentional weight loss of approximately 30 kg over the preceding year. His medical history was notable for long-standing ankylosing spondylitis with extra-articular manifestations, including recurrent uveitis, diagnosed in adolescence. Disease-modifying antirheumatic therapy had been discontinued 2 years prior to admission because of poor treatment adherence. Additional comorbidities included paroxysmal atrial fibrillation, chronic kidney disease, and long-standing gastrointestinal symptoms with multiple hospitalizations.

On presentation, the patient appeared severely cachectic, dehydrated, and pale. Physical examination demonstrated diminished pulmonary sounds at both lung bases, systolic hypotension (95/60 mmHg), and abdominal distension due to ascites, with the liver palpable 3 cm below the costal margin. Marked musculoskeletal rigidity and muscle wasting were noted, consistent with advanced ankylosing spondylitis and severe malnutrition.

Laboratory evaluation (Table 1) revealed leukocytosis with neutrophilia, normocytic normochromic anemia, iron (13.00 µg/dl; reference 33–193 µg/dl) and folic acid (1.56 ng/ml; reference 5.6–45.8 ng/ml) deficiency, marked systemic inflammation, hypoalbuminemia, and renal dysfunction. Cardiac biomarkers showed elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels (6,940 pg/ml), while high-sensitivity troponin values were only mildly increased (19.6 ng/L).

Chest X-rays demonstrated bilateral basal opacities, vascular congestion, and pleural effusions. Computed tomography of the chest and abdomen confirmed large bilateral pleural effusions with near-complete lower lobe atelectasis.

A right-sided thoracentesis evacuated 650 ml of serous fluid, consistent with a transudative process. Microscopic examination revealed the absence of leukocytes, and the cultures showed no aerobic microbial growth and no anaerobic flora detected, even after enrichment on culture media.

HISTOLOGICAL CONFIRMATION

We note that during the patient's most recent hospitalization in another institution 7 months earlier, where he presented for cardiology follow-up, he also underwent upper gastrointestinal endoscopy. Histopathological examina-

TABLE 1. Bloodwork evolution during inpatient stay

Parameter	On admission	Midway evaluation	On discharge	Reference
WBC, $\times 10^3/\mu l$	14.2	11.2	9.3	4–10
Hemoglobin, g/L	70	94	97.2	130–170
Hematocrit, %	21.1	29.4	35.6	40–52
Platelets, $\times 10^3/\mu l$	564	558	489	150–400
INR	1.04	0.95	0.98	0.8–1.2
Serum iron, µg/dl	13.4	19.2	34	33–193
ESR, mm/h	75	98	56	0–15
CRP, mg/L	156.4	180.5	65.4	0–5
Fibrinogen	635	480	445	200–400
Serum proteins, g/L	51.8	53.4	50.2	64–83
Albumin, g/L	26.8	25.4	29.8	35–50
ASAT, U/L	18.4	15.7	19.3	0–40
ALAT, U/L	14.6	15.4	24.5	0–41
HCO_3, mmol/L	15.4	18.5	22.4	22–28
Creatinine, mg/dl	1.78	1.67	1.19	0.72–1.25
eGFR, ml/min/1.73 m^2	40.43	46.7	65.79	>90

ALAT, alanine aminotransferase; ASAT, aspartate aminotransferase; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; ESR, erythrocyte sedimentation rate; HCO3, bicarbonate; INR, international normalized rate; WBC, white blood cells

tion of the gastric biopsy revealed antro-corporeal junctional mucosa with reactive foveolar changes. The lamina propria contained eosinophilic, acellular, homogeneous deposits within a polymorphous inflammatory infiltrate. These deposits were Congo red-positive, Masson's trichrome-negative, and showed positive immunostaining for amyloid A, with expression of IgM and both kappa and lambda light chains. Unfortunately, the histopathological images were not available because the examination had been performed at another institution.

CARDIOVASCULAR ASSESSMENT

Cardiac evaluation revealed low-voltage QRS complexes in limb leads and a pseudoinfarction pattern on electrocardiography.

Transthoracic echocardiography revealed concentric left ventricular hypertrophy with preserved cavity dimensions and mildly reduced ejection fraction (50%). It also demonstrated marked hypertrophy involving all cardiac chambers and valves, with a hyperechogenic myocardium displaying an infiltrative appearance associated with circumferential pericardial effusion (Figure 1). Global longitudinal strain was severely impaired (-10.7%), markedly disproportionate to the ejection fraction, supporting advanced longitudinal systolic dysfunction. Transmitral Doppler demonstrated an impaired relaxation pattern (E/A 0.59) (Figure 2), with markedly reduced septal (Figure 3) and lateral e′ velocities (4 cm/s and 5 cm/s, respectively) (Figure 4) and a normal E/e′ ratio (8.56), consistent with grade I diastolic dysfunction without overt evidence of elevated left ventricular filling pressures. The Bull's eye graph of the left ventricle displaying the magnitude and homogeneity of longitudinal strain in a color-coded map showed relative preservation of longitudinal strain at the apex resulting in a 'cherry on top' or 'apical sparing' pattern (Figure 5). These diastolic indices should, however, be interpreted cautiously in the clinical context of relative intravascular volume depletion. Also, right ventricular free wall hypertrophy with borderline longitudinal systolic function was observed. A circumferential pericardial effusion (maximum thickness 12 mm anterior to the right ventricle) with fibrin deposits was present, without echocardiographic signs of hemodynamic compromise. The inferior vena cava measured 9 mm, with preserved collapsibility.

CLINICAL COURSE

Abdominal ultrasonography demonstrated hyperechoic atrophic kidneys, suggesting chronic kidney disease (CKD), and moderate ascites, which was confirmed by computed tomography. Laboratory evaluation showed impaired renal function (eGFR 40.43 ml/min/1.73 m^2; reference >90 ml/min/1.73 m^2), hypoalbuminemia (26.8 g/L; reference 35–50

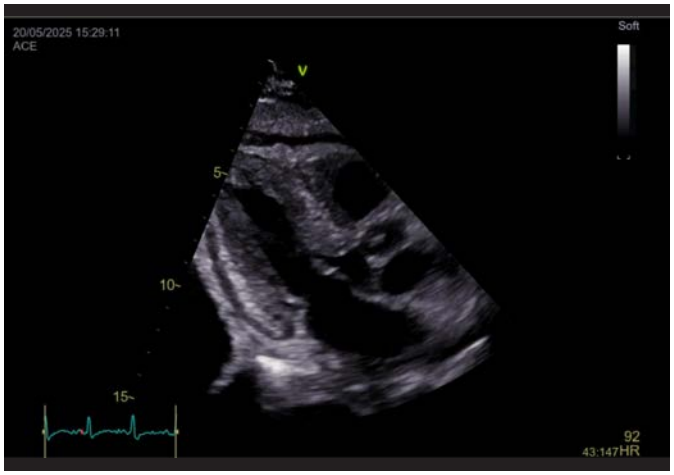

FIGURE 1. Transthoracic echocardiography demonstrating marked hypertrophy involving all cardiac chambers and valves, with a hyperechogenic myocardium displaying an infiltrative appearance, associated with circumferential pericardial effusion.

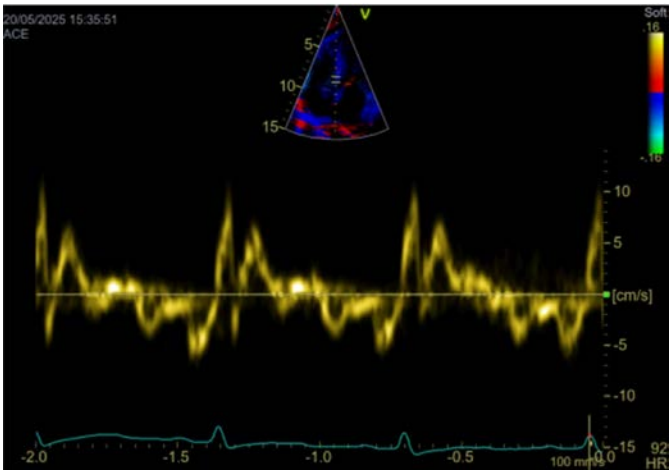

FIGURE 2. Pulsed-wave Doppler assessment of transmitral inflow demonstrating an E/A ratio of 0.59.

g/L), proteinuria (435.6 mg/24 h; reference 0–299 mg/24 h), and a markedly elevated urine albumin-to-creatinine ratio (539.68 mg/g), consistent with severely increased albuminuria (A3). Together with the presence of pleural effusions and ascites in the absence of diabetes, these findings raised suspicion for an underlying protein-losing nephropathy, including renal amyloidosis (AA or AL).

Given the persistent gastrointestinal symptoms, profound cachexia, and consumptive syndrome, an underlying malignancy was initially suspected. To rule out possible malignancies we screened alpha-fetoprotein, 19-9 cancer antigen, carcinoembryonic antigen, and prostate specific antigen, all being within normal ranges. Antinuclear antibodies (ANA) were also within the normal range (17.6 AU/ml; reference 0–39.9 AU/ml).

Given that upper gastrointestinal endoscopy was previously performed at another institution, we performed a colonoscopy, which revealed a friable mucosa in the left colon and multiple rectal ulcerations. Multiple biopsies were obtained, and histopathological examination revealed a chronic inflammatory infiltrate, with foci of activity characterized by epithelial neutrophils, findings suggestive of ulcerative colitis.

Further etiological evaluation included hematological assessment with bone marrow biopsy, peripheral blood smear, and serum and urine protein electrophoresis, which excluded plasma cell dyscrasia and AL amyloidosis. Serum protein electrophoresis demonstrated decreased albumin (45.9%; reference 55.8–66.1%), increased α1-globulins (6.9%; reference 2.9–4.9%), increased α2-globulins (15.4%; reference 7.1–11.8%), normal β1-globulins (6.1%;

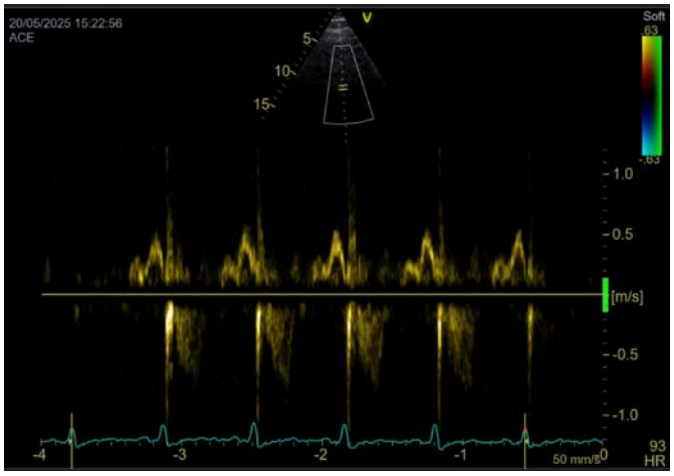

FIGURE 3. Tissue Doppler imaging of the mitral annulus demonstrating reduced septal e′ velocity (4 cm/s).

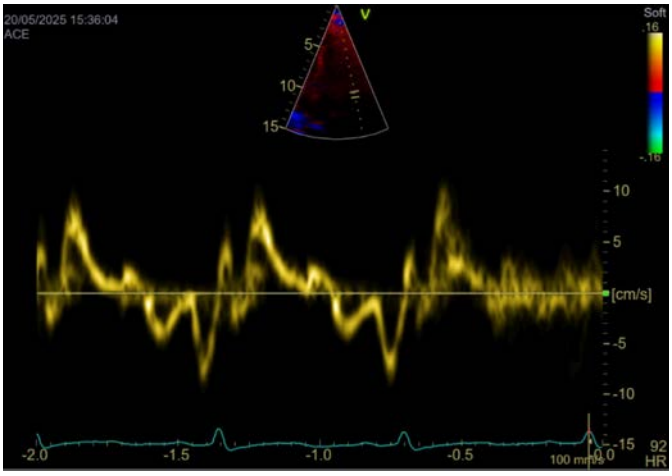

FIGURE 4. Tissue Doppler imaging of the mitral annulus demonstrating reduced lateral e′ velocity (5 cm/s).

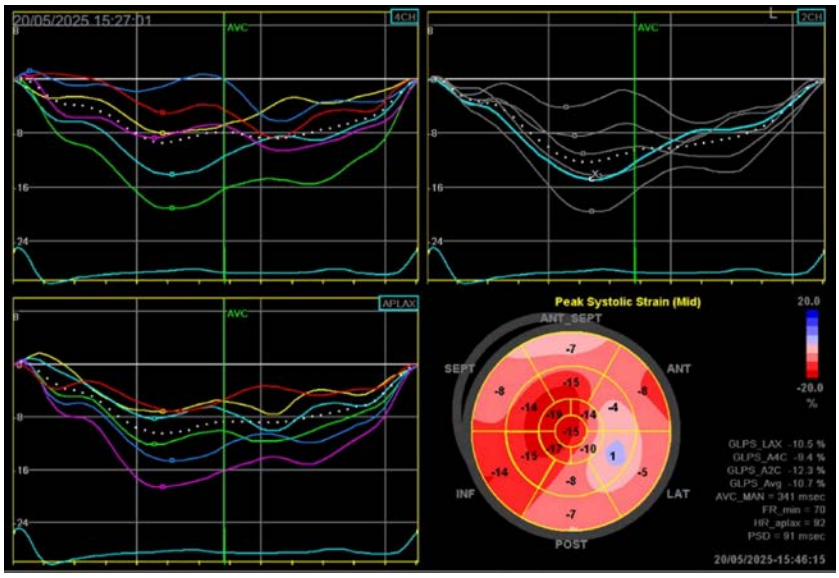

FIGURE 5. Bull's-eye plot of left ventricular longitudinal strain demonstrating a diffuse reduction in strain with relative apical sparing, displayed as a color-coded map.

reference 4.7–7.2%) and β2-globulins (4.6%; reference 3.2–6.5%), and mildly increased γ-globulins (19.7%; reference 11.1–18.8%), a pattern consistent with a chronic inflammatory response.

Serum immunoglobulin profiling showed elevated IgA (514 mg/dl; laboratory upper limit of normal (ULN) 400 mg/dl) and IgG (1,990 mg/dl; ULN 1,600 mg/dl), whereas IgM was within the normal range (168 mg/dl). Serum free light chain analysis revealed markedly increased kappa (278.00 mg/L; ULN 20.73 mg/L) and lambda (133.00 mg/L; ULN 27.69 mg/L) concentrations.

Bone marrow examination revealed hemophagocytosis without fulfilling criteria for hemophagocytic lymphohistiocytosis. Genetic testing for transthyretin amyloidosis was negative. In the context of long-standing, poorly controlled ankylosing spondylitis and chronic systemic inflammation, a diagnosis of systemic AA amyloidosis with cardiac, renal, gastrointestinal, and serosal involvement was established.

The patient was managed in an acute care setting with close hemodynamic and renal monitoring. Given the combination of large pleural effusions, dyspnea, hypoalbuminemia, and renal dysfunction, a cautious decongestive strategy was implemented with individualized loop diuretic therapy and strict fluid balance, while avoiding excessive preload reduction. Intravenous furosemide was started via continuous infusion (100 mg/24 h), then transitioned to oral furosemide 40 mg daily after clinical stabilization, with strict fluid balance and serial renal function assessment. Spironolactone 50 mg orally was added for persistent congestion. Intravenous albumin was administered to address the marked hypoalbuminemia and suspected low effective circulating volume, with reassessment of blood pressure, urine output, and renal function. Therapeutic thoracentesis was performed for symptomatic relief and diagnostic clarification. Atrial fibrillation was managed with rate/rhythm control as clinically indicated, and anticoagulation was initiated with low-molecular-weight heparin (40 mg twice daily) and subsequently switched to apixaban 2.5 mg twice daily after stabilization and reassessment of renal function and bleeding risk. The circumferential pericardial effusion (12 mm) showed no echocardiographic signs of tamponade, therefore conservative management with surveillance was pursued.

Supportive management, including replacement therapy with packed red blood cells, iron, and intravenous albumin, careful volume control, diuretic therapy, anticoagulation adjustment, antiarrhythmic treatment, nutritional support, and targeted management of gastrointestinal complications led to gradual clinical stabilization and resolution of diarrhea. The patient was discharged in stable condition with close multidisciplinary follow-up and referral for reinitiation of disease-modifying therapy for ankylosing spondylitis.

DISCUSSION

AA amyloidosis is a severe systemic complication of chronic inflammatory disorders, resulting from sustained overproduction of SAA in the setting of persistent inflammation. Although its incidence has declined with the introduction of effective anti-inflammatory and bio-

logic therapies, AA amyloidosis remains a life-threatening condition in patients with long-standing or inadequately controlled inflammatory disease.[2,7,13] Ankylosing spondylitis, although less frequently associated with AA amyloidosis than rheumatoid arthritis, is a recognized etiological factor, particularly in cases with early disease onset, prolonged inflammatory burden, extra-articular manifestations, and poor treatment adherence.[7,14,15]

This case illustrates the varied manifestations of advanced AA amyloidosis, with simultaneous cardiac, renal, gastrointestinal, and serosal involvement. Although renal disease is the most common and often earliest manifestation, extrarenal involvement may dominate the clinical picture and substantially delay diagnosis.[1-3,7,17] In our patient, chronic diarrhea, profound weight loss, hypoalbuminemia, and severe cachexia preceded the diagnosis of systemic amyloidosis by several years, initially raising suspicion of malignancy or primary gastrointestinal disease.

Gastrointestinal involvement in AA amyloidosis is frequently underrecognized because of its nonspecific clinical presentation and the patchy distribution of amyloid deposits within the gastrointestinal tract. Amyloid deposition predominantly affects the lamina propria and submucosa and may be focal, leading to false-negative results despite multiple biopsy samples.[8,12,18] In the present case, although several gastric and colonic biopsies were obtained, amyloid deposition was demonstrated only in selected specimens, significantly complicating and delaying the diagnostic process. This observation underscores the importance of repeated and adequately targeted biopsies, as well as close clinicopathological correlation, when AA amyloidosis is clinically suspected.

Cardiac involvement in AA amyloidosis is considerably less common than in AL or ATTR amyloidosis but is associated with adverse prognosis when present.[4,6,9] In the case of this patient, the coexistence of heart failure with mildly reduced ejection fraction (HFmrEF), microvolted QRS complexes with pseudoinfarction pattern, markedly elevated NT-proBNP levels, and circumferential pericardial effusion strongly supported clinically relevant cardiac involvement, further contributing to disease severity.

An additional factor contributing to the diagnostic complexity was the presence of hemophagocytosis on bone marrow examination. Although nonspecific and insufficient to establish hemophagocytic lymphohistiocytosis, this finding likely reflected chronic immune activation and cytokine dysregulation in the context of sustained systemic inflammation,[19] reinforcing the inflammatory environment driving persistent SAA elevation and amyloidogenesis.

The management of AA amyloidosis relies primarily on durable suppression of the underlying inflammatory process with the aim of reducing circulating SAA levels.[3,10,20] Early recognition is crucial, as effective control of inflammation may stabilize organ function and, in some cases, induce partial regression of amyloid deposits. This case highlights the need for heightened clinical suspicion, awareness of diagnostic limitations related to focal amyloid deposition, and close interdisciplinary collaboration among cardiology, internal medicine, gastroenterology, nephrology, hematology, and rheumatology specialists.

CONCLUSIONS

In patients with chronic inflammatory disorders presenting emergently with dyspnea, serous effusions, renal dysfunction, and biochemical evidence of heart failure, clinicians should maintain a high index of suspicion for systemic AA amyloidosis with cardiac involvement. The combination of low-voltage ECG, concentric LV hypertrophy with 'infiltrative' myocardial texture, and severely reduced global longitudinal strain disproportionate to ejection fraction, particularly with apical sparing, represents a practical bedside constellation that can rapidly narrow the differential in the emergency and acute care setting. Prompt multidisciplinary evaluation (cardiology, rheumatology, nephrology/hematology, and pathology) and rapid control of the underlying inflammatory driver are crucial to limiting progression and improving prognosis.

This case illustrates systemic AA amyloidosis as a rare but devastating complication of long-standing, poorly controlled ankylosing spondylitis, characterized by extensive multiorgan involvement and clinically significant cardiac manifestations. Although cardiac involvement is uncommon in AA amyloidosis, it substantially worsens prognosis.

The presence of chronic kidney disease with severe albuminuria suggests significant glomerular involvement. Marked hypoalbuminemia was likely multifactorial, reflecting chronic inflammation together with renal and possibly gastrointestinal protein losses, and likely contributed to the development of pleural effusions and ascites.

Nonspecific gastrointestinal symptoms, severe cachexia, serosal effusions, and the focal distribution of amyloid deposits may substantially delay the diagnosis, even when multiple biopsy specimens are obtained. Gastrointestinal biopsy with appropriate amyloid typing remains a valuable diagnostic tool; however, negative colonic biopsy results do not exclude the disease and should prompt repeat evaluation with systematic biopsy sampling. Sustained suppression of systemic inflammation remains the cornerstone of

therapy, underscoring the importance of early diagnosis, treatment adherence, and a coordinated multidisciplinary approach in patients with chronic inflammatory diseases and otherwise unexplained multisystem involvement.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

ETHICS APPROVAL

The publication of this case report was approved by the Hospital Commission for Medical Ethics of Studies of the County Emergency Clinical Hospital of Târgu Mureș (approval no. 34798/04.02.2026).

CONSENT TO PARTICIPATE

Written informed consent for publication was obtained from the patient.

FUNDING

This research received no external funding.

ACKNOWLEDGMENT

The authors thank the patient for consenting to publication of this case report.

AUTHOR CONTRIBUTIONS

N.C.C. conceived and supervised the study, conducted the investigation, curated the data, and prepared the original draft of the manuscript. M.C. contributed to the study conceptualization, participated in the investigation, and critically reviewed and edited the manuscript. A.R. participated in the investigation and performed the imaging acquisition and interpretation. All authors have read and agreed to the published version of the manuscript.

REFERENCES

1. Kaptein FHJ, Kroft LJM, Been HD, et al. Pulmonary infarction Savadogo B, Fahed H, Sellam J, Georgin-Lavialle S, Fautrel B, Mitrovic S. AA amyloidosis in inflammatory joint diseases: a systematic review. Semin Arthritis Rheum. 2025;74:152762. doi: 10.1016/j. semarthrit.2025.152762
2. Obici L, Merlini G. AA amyloidosis: basic knowledge, unmet needs and future treatments. Swiss Med Wkly. 2012;142:w13580. doi: 10.4414/smw.2012.13580
3. Mirioglu S, Uludag O, Hurdogan O, et al. AA Amyloidosis: A Contemporary View. Curr Rheumatol Rep. 2024 Jul;26(7):248-259. doi: 10.1007/s11926-024-01147-8
4. Muchtar E, Dispenzieri A, Magen H, et al. Systemic amyloidosis from A (AA) to T (ATTR): a review. J Intern Med. 2021;289(3):268-292. doi: 10.1111/joim.13169
5. Brunger AF, Nienhuis HLA, Bijzet J, Hazenberg BPC. Causes of AA amyloidosis: a systematic review. Amyloid. 2020;27(1):1-12. doi: 10.1080/13506129.2019.1693359
6. Liu L, Wang R, Strocchi S, et al. Serum amyloid A in HFpEF and cardiometabolic diseases. Basic Res Cardiol. 2026;121:1-21. doi: 10.1007/s00395-025-01150-9
7. Lachmann HJ, Goodman HJB, Gilbertson JA, et al. Natural history and outcome in systemic AA amyloidosis. N Engl J Med. 2007;356(23):2361-2371. doi: 10.1056/NEJMoa070265
8. Cowan AJ, Skinner M, Seldin DC, et al. Amyloidosis of the gastrointestinal tract: a 13-year, single-center, referral experience. Haematologica. 2013;98(1):141-146. doi: 10.3324/haematol.2012.068155
9. Kittleson MM, Maurer MS, Ambardekar AV, et al. 2023 ACC Expert Consensus Decision Pathway on Comprehensive Multidisciplinary Care for the Patient With Cardiac Amyloidosis. J Am Coll Cardiol. 2023;81(11):1076-1126. doi: 10.1016 /j.jacc. 2022.11.022
10. Petre S, Shah IA, Gilani N. Review article: gastrointestinal amyloidosis—clinical features, diagnosis and therapy. Aliment Pharmacol Ther. 2008;27(11):1006-1016. doi: 10.1111/j.1365-2036.2008.03682.x
11. Talar-Wojnarowska R, Jamroziak K. Intestinal amyloidosis: clinical manifestations and diagnostic challenge. Adv Clin Exp Med. 2021;30(5):563-570. doi: 10.17219/acem/133521
12. Wisniowski B, Wechalekar A. Confirming the diagnosis of amyloidosis. Acta Haematol. 2020;143(4):312-321. doi: 10.1159/000508022
13. Hazenberg BPC, van Gameren II, Bijzet J, Jager PL, van Rijswijk MH. Diagnostic and therapeutic approach of systemic amyloidosis. Neth J Med. 2004;62(4):121-128.
14. Picken MM. The pathology of amyloidosis in classification: a review. Acta Haematol. 2020;143(4):322-334. doi: 10.1159/000506696
15. Pamuk ÖN, Kalyoncu U, Aksu K, et al. A multicenter report of biologic agents for the treatment of secondary amyloidosis in Turkish rheumatoid arthritis and ankylosing spondylitis patients. Rheumatol Int. 2016;36(7):945-953. doi: 10.1007/s00296-016-3500-9
16. Nakamura T. Amyloid A amyloidosis secondary to rheumatoid arthritis: pathophysiology and treatments. Clin Exp Rheumatol. 2011;29(5):850-857.
17. Georgin-Lavialle S, Grateau G. [Clinical aspects of systemic amyloidosis in 2024]. Ann Pathol. 2024;44(6):407-413. doi: 10.1016/ j.annpat. 2024.09.015
18. Ebert EC, Nagar M. Gastrointestinal manifestations of amyloidosis. Am J Gastroenterol. 2008;103(3):776-787. doi: 10.1111/j.1572-0241.2007.01669.x
19. Wu Y, Sun X, Kang K, et al. Hemophagocytic lymphohistiocytosis: current treatment advances, emerging targeted therapy and underlying mechanisms. J Hematol Oncol. 2024;17(1):106. doi: 10.1186/s13045-024-01621-x
20. Lane T, Gillmore JD, Wechalekar AD, Hawkins PN, Lachmann HJ. Therapeutic blockade of interleukin-6 by tocilizumab in the management of AA amyloidosis and chronic inflammatory disorders: a case series and review of the literature. Clin Exp Rheumatol. 2015;33(6 Suppl 94):S46-S53.