Cardiac Amyloidosis: The Importance of Echocardiography for Early Diagnosis

Amiloidose Cardíaca: A Importância do Ecocardiograma para o Diagnóstico Precoce

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Abstract:

Cardiac amyloidosis is an infiltrative and progressive disease that often causes restrictive cardiomyopathy and heart failure with preserved ejection fraction. Two types of protein precursors are responsible for most cardiac amyloidosis cases: transthyretin amyloid (ATTR) and monoclonal immunoglobulin light chains (AL). We present the case of a 75-year-old man admitted in the urgency department with signs and symptoms of heart failure. After performing a transthoracic echocardiogram, the hypothesis of cardiac amyloidosis was raised. The investigation of monoclonal light chains of immunoglobins continued to assess the possibility of AL amyloidosis. Given the negative results, ^{99m}Tc di-phosphonate scintigraphy and genetic tests were performed, confirming wild-type ATTR amyloidosis.

We discuss the clinical and diagnostic features of cardiac amyloidosis, with the aim of increasing awareness of this systemic disease so that it can be recognized, diagnosed and treated promptly. We also aim to demonstrate the potential of transthoracic echo-cardiography as a first-line examination.

Keywords: Amyloidosis/diagnostic imaging; Cardiomyopathies/diagnostic imaging; Cardiomyopathy, Restrictive/diagnostic imaging; Echocardiography.

Resumo:

A amiloidose cardíaca é uma doença infiltrativa que causa frequentemente miocardiopatia restritiva e insuficiência cardíaca com fração de ejeção preservada. Os tipos de proteínas precursoras responsáveis pela maioria dos casos são a transtirretina (ATTR) e as cadeias leves de imunoglobulinas (AL).

Apresentamos o caso de um homem, 75 anos, admitido no serviço de urgência com sinais e sintomas de insuficiência cardíaca. Após a realização de um ecocardiograma transtorácico, colocou-se a hipótese de amiloidose cardíaca. Foi realizada a investigação de cadeias leves monoclonais de imunoglobulina para avaliar a possibilidade de amiloidose AL. Diante dos resultados negativos, foram realizadas cintigrafia óssea com difosfonato de ^{99m}Tc e testes genéticos, confirmando-se amiloidose ATTR *wild-type*.

Neste caso discutimos as características clínicas e diagnósticas desta doença, com o objetivo de aumentar a consciencialização sobre a mesma, para que seja reconhecida, diagnosticada e tratada prontamente. Também pretendemos demonstrar a importância da ecocardiografia como exame de primeira linha.

Palavras-chave: Amiloidose/diagnóstico por imagem; Cardiomiopatias/diagnóstico por imagem; Cardiomiopatia Restritiva/diagnóstico por imagem; Ecocardiografia.

Introduction

Amyloidosis is a systemic infiltrative disease characterized by the extracellular deposition of insoluble proteins in different organs and tissues.[1-3] The different systemic manifestations and the variety of symptoms make it one of the most underdiagnosed entities.[1,2,4] It is classified according to the precursor protein that forms the amyloid deposit.[1,4,5] While more than 30 proteins are known, more than 98% of currently diagnosed amyloidosis results from fibrils composed of monoclonal immunoglobulin light chains (AL) or transthyretin (ATTR).[1,4,6] Cardiac amyloidosis (CA) refers to amyloid fibril deposition in the heart and has been reported in both variants of amyloidosis.[1,2,4,5] Cardiac involvement is one of the leading causes of restrictive cardiomyopathy and the main cause of mortality in patients with systemic amyloidosis.[1,3] The classic clinical presentation of CA is exertional dyspnoea and orthopnoea, fatigue and weakness.[7]

In this case report, we discuss a 75-year-old man who presented to the emergency department with symptoms and signs of heart failure (HF). After a suspicious echocardiogram, additional tests were performed and ATTR amyloidosis was diagnosed.

ATTR results from the misfolding and deposition of transthyretin.[1,2,8] In normal conditions, it is a stable tetramer that is produced by the liver, and it is responsible for the transport of thyroid hormone and retinol.[2,7-9] The pathogenesis of ATTR amyloidosis is caused by destabilisation of transthyretin protein tetramers.[2,7,8] ATTR amyloidosis can be hereditary

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(ATTRh) or acquired/wild-type (ATTRwt).[1,2,4,5,8,9] ATTRh amyloidosis is a rare disease and, besides the heart, it can also involve the peripheral and autonomic nervous systems.[1,2,8] ATTRwt amyloidosis is relatively common and accounts for a significant number (13%) of patients with heart failure with preserved ejection fraction (HFpEF).[10] So, it is recognized as a cause of HFpEF in the elderly.[2,8] ATTRwt amyloidosis is more prevalent in people over 60 years of age and in the male sex and mainly involves the heart and soft tissues, which can manifest as bilateral carpal tunnel syndrome, spontaneous biceps tendon rupture and spinal stenosis.[1,2,5,8]

The prognosis of cardiac amyloidosis varies based on the type and stage of the disease, the degree of cardiac involvement, early diagnosis, and the response to treatment.[11] Overall, ATTR amyloidosis has a better prognosis than AL amyloidosis, because it progresses more slowly.[1,11] CA has a median survival from diagnosis ranging from less than 6 months to 5 years for AL amyloidosis, and from 3 to 5 years for ATTR amyloidosis. Advances in treatments are improving life expectancy for many patients.[11] In the case of ATTR amyloidosis, treatment options include transthyretin stabilizers, such as tafamidis.[1,2,12,13]

Case Report

We present the case of a 75-year-old-man admitted to the emergency department with fatigue on minimal exertion, shortness of breath and nonspecific chest pain that resolved spontaneously. On physical examination, the patient was cooperative, acyanotic, afebrile, well hydrated, with no evidence of edema or signs of deep vein thrombosis. His systemic blood pressure was 102/49 mmHg.

The patient had a history of chronic renal failure (with a serum creatinine of 1.46 mg/dL), atrial fibrillation and pacemaker implantation four months before. He presented to the emergency department on four separate occasions during the last three months. Laboratory testing revealed elevated N-Terminal pro-B-type natriuretic peptide (NT-proBNP) at 3162 pg/mL, and cardiac troponin T was also increased to 86.70 ng/L. Point-of-care ultrasound (POCUS) was not performed during any of the patient's presentations to the emergency department.

The initial suspected diagnosis was acute HF, and it was then decided to hospitalize the patient.

To assess cardiac structure and function, determine the specific type of HF (systolic/diastolic) and identify underlying causes of HF (valvular disease, ischemic heart disease or cardiomyopathies), a transthoracic echocardiography (TTE) was requested. It revealed severe left ventricular wall thickness with “granular sparkling” appearance (Fig. 1A-B). The left ventricular ejection fraction (LVEF) was at the lower limit of normality (Fig. 1C). Regional strain values were greatly reduced in the basal and mid-wall regions in comparison to the apex, where the strain is relatively preserved (*apical sparing*) and global longitudinal strain (GLS) was diminished (Fig. 1D). In imaging exams performed with GE equipment, a GLS more negative than -18% is considered normal.

The low septal and lateral Doppler e´ values and the elevated septal E/e´ ratio were diastolic dysfunction indicators with elevated filling pressures (Fig. 1E). Biatrial enlargement and a moderate pericardial effusion were also seen.

In view of these echocardiographic findings, the hypothesis of CA was raised, prompting a search for other "*red flags*" to support this diagnosis. Notably, the patient had also undergone treatment for carpal tunnel syndrome 14 years prior and, as previously mentioned, had a pacemaker implanted and a history of atrial fibrillation. Additionally, the prior electrocardiogram (ECG) before pacemaker implantation was reviewed and showed low QRS voltage.

Therefore, the patient proceeded with diagnostic testing. To assess the possibility of AL the investigation of monoclonal light chains of immunoglobins was carried out by serum and urine protein electrophoresis and immunofixation. No free light chains were found in either the patient’s blood or urine. Given the negative results, ^{99m}Tc diphosphonate scintigraphy was performed and it revealed a myocardial uptake greater than bone with reduced bone uptake (grade 3), confirming ATTR amyloidosis (Fig. 1F). The patient started treatment with tafamidis 61 mg orally once daily. Genetic tests were negative, making it possible to diagnose ATTRwt.

Discussion

The initial diagnostic evaluation of a patient with suspected amyloidosis includes recognition of cardiac and extracardiac signs and symptoms that are termed *“red flags”*.[1,12] Cardiac signs and symptoms include HFpEF or other cardiac conditions, such as increasing left ventricular wall thickness, atrial fibrillation, atrioventricular conduction abnormalities, intolerance to conventional HF therapies, discordance between QRS voltage on ECG and an increase in left ventricular wall thickness.[2,5,10,12,14] Extracardiac signs and symptoms, such as bilateral carpal tunnel syndrome, spontaneous biceps tendon rupture, peripheral polyneuropathy, and hypotension in previously hypertensive patients, are extremely useful to suspect the disease in the presence of compatible cardiac imaging findings.[1,8,12]

CA remains an underdiagnosed condition, largely due to its nonspecific clinical presentation.[1,3,12] In the case under analysis, the patient presented some important clues, such as pacemaker implantation, atrial fibrillation and a previous carpal tunnel surgery, which further raised suspicion for CA. However, only after the suspicion of HF and the echo-cardiographic evaluation was the puzzle completed by identifying these additional *“red flags”*.

So, although an echocardiogram is not sufficient for a definitive diagnosis or to differentiate between types of CA, it remains the primary imaging modality.[1,5] The main findings include moderate to severe left ventricular hypertrophy,

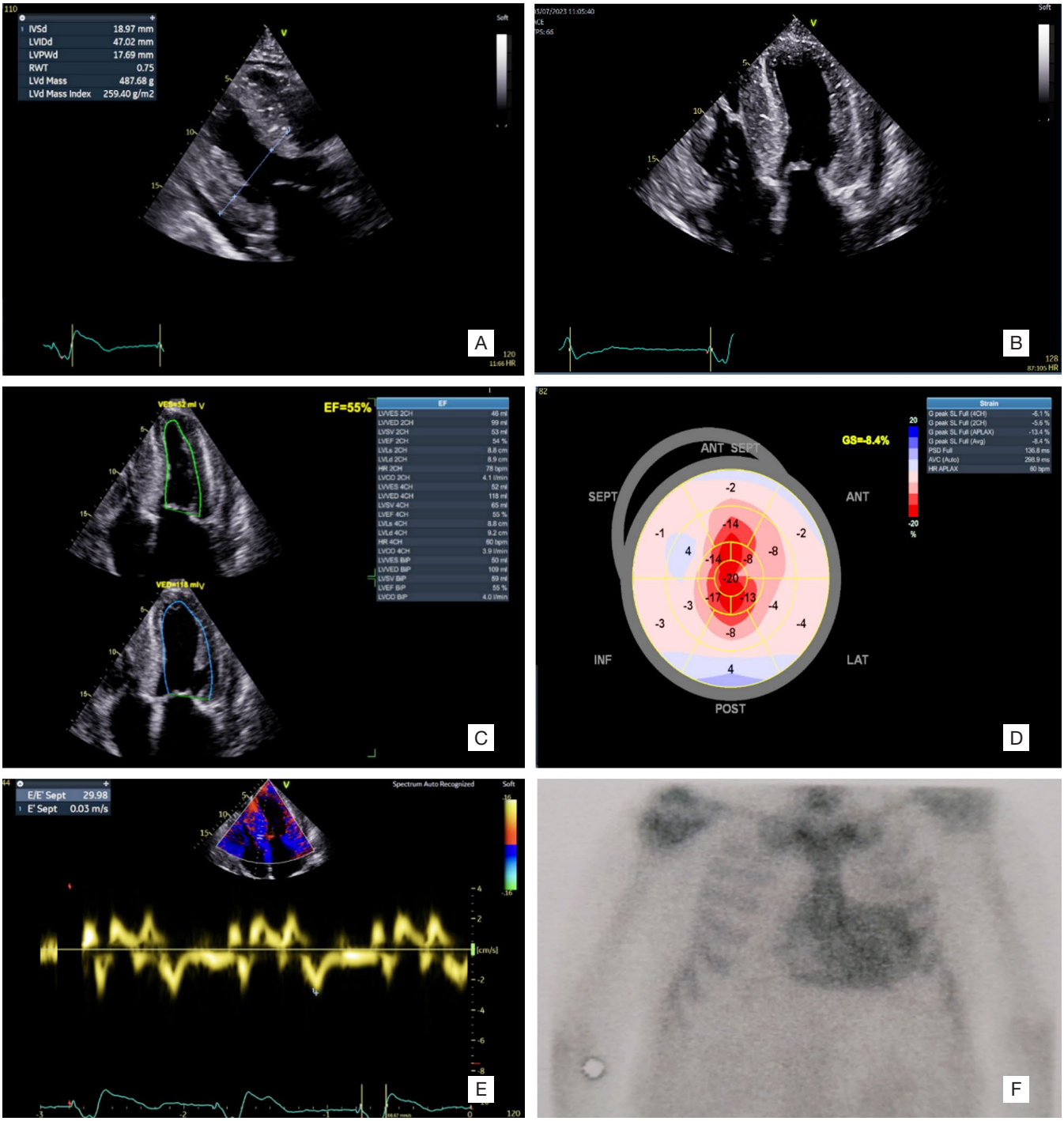

Figure 1: (**A** and **B**) TTE revealed severe thickness of the ventricular walls, with "granular sparkling". (**C**) Preserved global left ventricular ejection fraction (biplane LVEF=55%). (**D**) Myocardial deformation analysis showed a basal-to-apical gradient ("apical sparing") and decrease in global longitudinal strain (GLS=-8.4%) (**E**) Left ventricular diastolic measurements were abnormal, demonstrating diastolic dysfunction with elevated filling pressures (E/e´ septal=29.98) (**F**) ^{99m}Tc diphosphonate scintigraphy confirmed ATTR.

diastolic dysfunction with a restrictive filling pattern, biatrial enlargement, thickening of atrioventricular valves and interatrial septum and presence of pericardial effusion.[1,4,5,14,15] However, reduction in GLS with a relative *apical sparing* pattern is a key feature of CA.[1-6,12,15] GLS is a measure of longitudinal deformation with more negative values indicating greater deformation and, in imaging exams performed with GE equipment, a GLS more negative than -18% is considered normal.[4,5,15] Although a normal base-to-apex gradient in longitudinal strain exists, this gradient is typically more pronounced in patients with CA, corresponding to the distinctive apical sparing pattern.[1,3-5,15]

All of these echocardiographic *"red flags"* were identified in the present case. Echocardiographic changes in CA, similar to

clinical manifestations, present insidiously and become more evident as the degree of amyloid infiltration increases.

It is important to mention that CA is relatively common and should enter the differential diagnosis for any patient presenting with increased left ventricular wall thickness, including those with hypertrophic cardiomyopathy.[3,12] Its poorer prognosis compared with other causes of LV wall thickening makes the early detection and differential diagnosis of CA extremely important.[3,12,16] Myocardial infiltration in CA may cause HFpEF in early stages and reduced ejection fraction later.[16] Amyloid protein deposition in the heart muscle impairs relaxation and filling during the diastolic phase, increasing left ventricular filling pressures.[16] The resulting diastolic dysfunction can manifest as dyspnoea and fatigue, symptoms that were present in the case we report. Signs of diastolic dysfunction with increased ventricular filling pressures were detected on echocardiography by a high E/e' ratio.[12,16] Bidimensional strain echocardiography and E/e' ratio are often abnormal in early disease, aiding in the differential diagnosis.[12]

The regional *apical sparing* pattern mentioned above has a specificity of 82%.[3] On the other hand, other distinguishing features include the presence of other intracardiac or extracardiac signs.[3,12]

After diagnostic suspicion of CA, serum and urine protein electrophoresis and immunofixation to identify monoclonal proteins are mandatory.[1,4,12,17] Exclusion of monoclonal proteins helps support the diagnosis of ATTR amyloidosis and the condition can be diagnosed noninvasively with nuclear scintigraphy (in this case, ^{99m}Tc diphosphonate scintigraphy).[1,2,12] Cardiac bone tracer scintigraphy detects cardiac transthyretin and may help identify and diagnose ATTR cardiac amyloidosis.[1,2,4,12,17] The Perugini staging system is applied based on visual scoring: grade 0, no cardiac uptake and normal bone uptake; grade 1, myocardial uptake in a lower degree than at the bone level; grade 2, similar myocardial and bone uptake; grade 3, myocardial uptake greater than bone with reduced/absent bone uptake (Fig. 2).[1,17]

In all patients with ATTR amyloidosis, regardless of age, genetic testing is important to determine whether the patient has the ATTRh or ATTRwt, because the results may have implications for family members and may also help predict the sites of organ involvement.[1,17]

ATTR CA is diagnosed with grade 2 or 3, in contrast with AL amyloidosis, in which usually there is absent or grade 1 uptake.[1,13] If the bone scintigraphy result is negative or inconclusive, an invasive method, endomyocardial biopsy should be considered.[1,13]

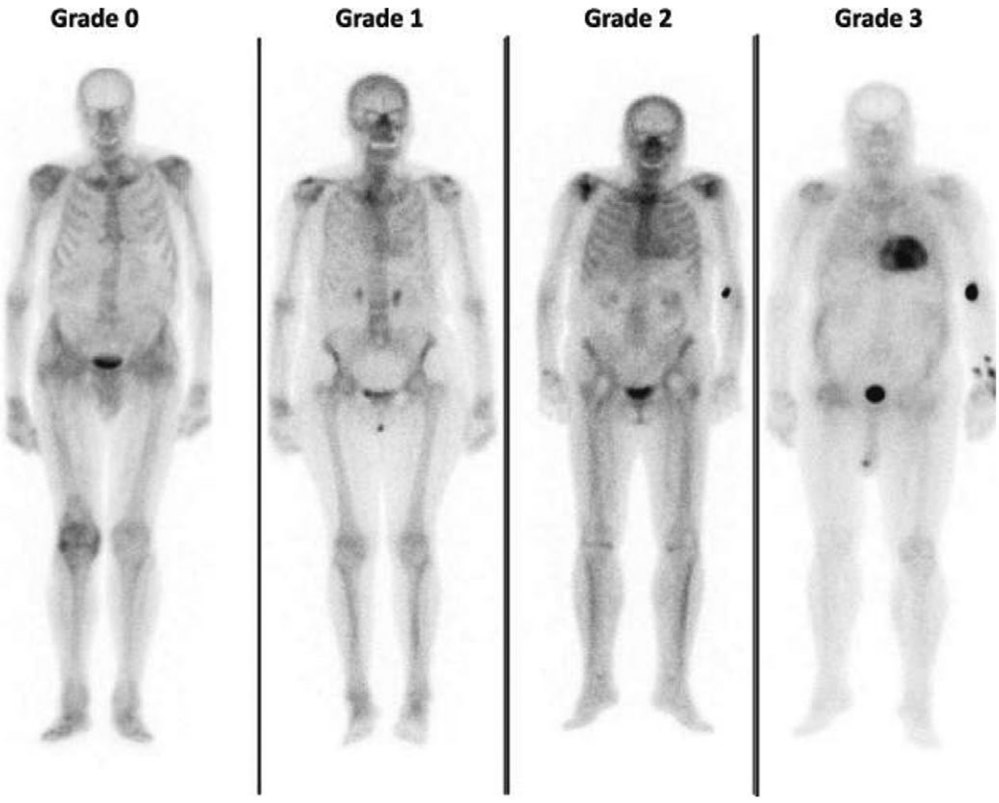

Figure 2: Examples of 99mTc-diphosphonate bone scintigraphy illustrating the different grades of the Perugini classification system for ATTR amyloidosis.
Source: Garcia-Pavia P, *et al*. Diagnosis and treatment of cardiac amyloidosis. Eur Heart J. 2021;42:1554-68.[1]

In the case under analysis, it was possible to establish the diagnosis of ATTRwt through a suspected TTE, clinical re-evaluation, analysis of monoclonal proteins, bone scintigraphy, and, finally, genetic testing.

It is important to note that the validation of bone tracer scintigraphy as a diagnostic modality for CA has significantly advanced the ability to establish a definitive, non-invasive diagnosis of ATTR in a substantial number of patients.[12] However, the diagnosis of AL amyloidosis still relies on histopathological confirmation of amyloid deposits. Nevertheless, positron emission tomography (PET) radiotracers are showing considerable promise as innovative, non-invasive tools for the detection of cardiac amyloid deposition. These radiotracers provide valuable insights into the amyloid type and the extent of myocardial involvement.[12]

On the other hand, artificial intelligence (AI) is emerging as a valuable tool for the diagnosis of CA, using data from ECG, echocardiography, cardiac magnetic resonance, and nuclear imaging to detect disease-specific patterns with high accuracy. AI can identify early or subtle signs of CA.[12] For instance, AI-enhanced ECG models have been shown to detect early and even preclinical manifestations of CA, identifying signal abnormalities that may go unnoticed by human interpretation.[12] Similarly, AI applied to echocardiographic data, both still images and video clips, can extract complex structural and functional features that facilitate differentiation between CA and other causes of left ventricular hypertrophy.[12] These technologies, which continue to evolve, support earlier diagnosis, risk stratification, and personalized treatment planning, potentially reducing invasive testing and improving patient outcomes.[12]

In terms of prognosis, two staging systems are commonly used in ATTR amyloidosis.[18] The Mayo Clinic system incorporates NT-proBNP (>3000 pg/mL), cardiac troponin T (>50 ng/L), and estimated glomerular filtration rate (eGFR) (<45 mL/min/1.73 m²), with Stage III (all three criteria) associated with the poorest outcomes.[18] The National Amyloidosis Centre (NAC) system uses NT-proBNP and eGFR, with Stage III defined when both parameters are abnormal.[18]

In this case, the patient showed elevated NT-proBNP (3162 pg/mL), cardiac troponin T (86.70 ng/L), and impaired renal function (serum creatinine 1.46 mg/dL; eGFR <45 mL/min/1.73 m²). He therefore meets Stage III criteria in both models, highlighting the advanced stage of the disease and its associated poor prognosis

So, initiation of tafamidis, a transthyretin (TTR) stabilizer, is clearly indicated in this context. At the time of diagnosis, Tafamidis is the only medication approved in Europe for ATTR amyloidosis with cardiac involvement.[12,13] The choice of a 61 mg once-daily dose of tafamidis is supported by clinical data demonstrating superior tetrameric stabilization of TTR, along with a favourable safety profile. This dosage proved more effective than lower doses without a significant increase in adverse events, as evidenced in the ATTR-ACT trial.[19] More recently, in February 2025, acoramidis was also approved by the European Medicines Agency (EMA) for the treatment of ATTR amyloidosis.[20] Acoramidis is also a TTR stabilizer that demonstrates higher affinity and selectivity, with the potential for superior clinical efficacy.[12,20] It may surpass or complement tafamidis, particularly in patients with more advanced symptoms or suboptimal responses to existing therapies.[12,13,20]

The treatment of ATTR has advanced significantly over recent decades, shifting from exclusively palliative approaches to therapies targeting the underlying disease mechanisms.[12] Previously, ATTR was treated solely through liver transplantation; however, recent developments have introduced a range of pharmacological therapies that act at various stages of the amyloidogenic process, from TTR stabilization to gene editing.[12] With advancements in biotechnology, therapies aimed at reducing hepatic TTR production have been developed, including gene silencers and antisense oligonucleotides. More recently, gene editing strategies, particularly those based on CRISPR-Cas[9] technology, have garnered attention for their potential to induce long-lasting or permanent suppression of mutant TTR production in the liver.[12] Early clinical studies in humans have demonstrated significant reductions in serum TTR levels following a single administration, suggesting the possibility of a functional cure.[12]

This therapeutic evolution, from TTR stabilizers to direct genomic modification, signals a promising future for patients with ATTR.[12]

Conclusion

Cardiac involvement of amyloidosis carries prognostic value and is the most critical determinant of survival in this disease.[1] Amyloidosis is a systemic infiltrative disorder that can cause restrictive cardiomyopathy and transthyretin amyloid cardiomyopathy is an increasingly recognized cause of heart failure with HFpEF.[2,10,17] CA remains underdiagnosed, so increased awareness of this disease is essential to achieve a timely and definitive diagnosis and therefore prevent the patient from reaching a poor clinical outcome.[12,17] CA has historically been difficult to diagnose due to the absence of specific clinical manifestations.[7] However, nowadays it is possible to recognize this disease in a non-invasive manner,[1,2,7,10] as this case highlights. No single imaging modality is sufficient for the diagnosis of cardiac amyloidosis, but strain echocardiography can play a critical role in the diagnosis due to its wide availability and portability.[4,6,12] It is also important for the prognosis and follow-up of these patients.[1,2,4,6] Thus, with advances in imaging techniques, conditions that were previously considered uncommon and difficult to diagnose, such as amyloidosis, are now more easily identified.[4,7,10] In parallel, therapeutic strategies for ATTR amyloidosis have advanced noticeably in recent years, with disease-modifying agents demonstrating the capacity to alter the natural course of the disease. These developments highlight the urgent need for heightened clinical awareness and the

implementation of systematic diagnostic approaches to facilitate earlier detection and optimize treatment outcomes.[12] Early detection of CA is essential.[1,2,6,12] ■

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AF - Image acquisition and manuscript drafting
JA - Supervision, editing, and validation
All authors approved the final version to be published.

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