Cardiac Amyloidosis: Why Cardiac Magnetic Resonance?

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Introduction

Amyloidosis is a clinical disorder characterized by extracellular deposition of misfolded protein fibrils in various organs. These fibrils arise from proteins with unstable tertiary structures that misfold into insoluble β-pleated sheets resistant to proteolysis, leading to progressive accumulation within the extracellular matrix. Organ dysfunction occurs when these deposits disrupt tissue architecture and function [1]. Cardiac involvement is the leading cause of morbidity and mortality in systemic amyloidosis [2–4].

Cardiac amyloidosis (CA) results from myocardial deposition of amyloid fibrils, which leads to increased ventricular stiffness, impaired diastolic function, and eventually systolic dysfunction. The vast majority (~95%) of cases are due to either immunoglobulin light-chain amyloidosis (AL) or transthyretin amyloidosis (ATTR) [2, 3]. Differentiating between AL and ATTR amyloidosis is critical due to major therapeutic and prognostic implications. Transthyretin (TTR) is a transport protein synthesized primarily in the liver. Disease occurs when TTR tetramers dissociate into unstable monomers that misfold and aggregate into amyloid fibrils. This process may result from inherited mutations (ATTRv) or age-related instability of the wild-type protein (ATTRwt). TTR variants are endemic to certain areas with the p.Val50Met variant being most common in Europe and the p.Val142Ile variant thought to be carried by up to 3% to 4% of African Americans [5, 6]. AL amyloidosis is caused by deposition of misfolded monoclonal light chains produced by clonal plasma cells.

ATTRwt predominantly affects older men, whereas ATTRv presents with variable phenotypes, which depend on the mutation and include cardiac, neurologic, or mixed involvement. The prognosis for AL amyloidosis depends heavily on the extent of cardiac involvement, with a median survival of nine months in advanced cardiac stages. Compared with AL amyloidosis, ATTR CA generally progresses more slowly, with a median survival of 3–5 years if untreated [5–7]. A recent meta-analysis [8] grouped the screening studies performed up to January 2022 and found that ATTR cardiomyopathy (CM) was present in 12% of patients with heart failure with preserved ejection fraction (HFpEF), in 10%–15% of elderly patients with severe aortic stenosis undergoing valve replacement, and in 7% of patients with left ventricular (LV) wall thickness ≥15 mm. Screening studies in other less explored scenarios, such as heart failure with reduced ejection fraction or cardiac conduction disorders [9–14] have also shown that ATTR CM is not uncommon and is certainly far from being a rare disease for the practicing cardiologist. Recent therapeutic advances — particularly TTR stabilizers and gene-silencing therapies, and emerging therapies such as CRISPR-based editing or amyloid-depleting antibodies — have transformed the management of ATTR amyloidosis. These findings underscore the importance of systematic evaluation and early diagnosis.

A better understanding of the underlying pathophysiology of TTR amyloid formation [8], advances in imaging techniques that have facilitated a more precise epidemiology of the disease [15–17], and momentum from new specific and highly effective therapies [18–22] have revolutionized the field, transforming what used to be a rare disease with a devastating prognosis into a more common disease with several therapeutic and diagnostic options.

In this evolving landscape, cardiac magnetic resonance (CMR) has emerged as a pivotal imaging modality that is uniquely capable of combining functional assessment with advanced tissue characterization.

Why CMR in cardiac amyloidosis?

CMR provides a noninvasive, multiparametric, and highly reproducible evaluation of cardiac structure, function, and tissue composition within a single examination. This comprehensive approach is particularly valuable in CA, where myocardial infiltration is diffuse and often difficult to quantify using conventional imaging.

1. Tissue characterization: The core strength of CMR

- Late gadolinium enhancement (LGE) imaging: LGE reveals the characteristic amyloid infiltration pattern, which is typically diffuse subendocardial or patchy/diffuse transmural enhancement. The pattern reflects expansion of the interstitial space due to amyloid deposition and has important diagnostic and prognostic implications [23].
- Parametric mapping techniques (linking imaging to biology): Recent advances in mapping techniques have significantly enhanced the role of CMR. Native T1 mapping detects early myocardial involvement, even in the absence of contrast, and shows high diagnostic accuracy in suspected CA in patients with high pretest probability [24]. Extracellular volume (ECV) quantification provides a direct measure of interstitial expansion and serves as a robust surrogate for amyloid burden. T2 mapping identifies myocardial edema, particularly relevant in AL amyloidosis, where direct light-chain toxicity contributes to myocardial injury [25]. Notably, ECV elevation may precede LGE and structural changes, allowing detection of disease at earlier stages.

2. Diagnostic and differential value

CMR plays a key role in differentiating CA from other causes of increased LV wall thickness, such as hypertensive heart disease, Anderson–Fabry disease, or hypertrophic cardiomyopathy. While certain imaging features may suggest ATTR versus AL (e.g., higher LV mass, higher ECV, and asymmetric myocardial thickening in ATTR), there is considerable overlap. CMR alone therefore cannot reliably distinguish subtypes, and CMR findings should always be interpreted within a multimodality diagnostic framework [26].

3. CMR in the era of disease-modifying therapies

Therapeutic advances in both AL and ATTR amyloidosis have significantly improved patient outcomes. In particular, TTR stabilizers and gene-silencing therapies have transformed the management of ATTR CA.

In this context, CMR has become an essential tool for longitudinal assessment: ECV quantification enables tracking of amyloid burden over time, detection of disease progression, or stabilization and early identification of treatment response, often before changes in ventricular function occur. The high reproducibility of CMR makes it ideally suited for serial evaluation in both clinical practice and clinical trials.

4. Prognostic stratification

CMR-derived biomarkers provide powerful prognostic information:

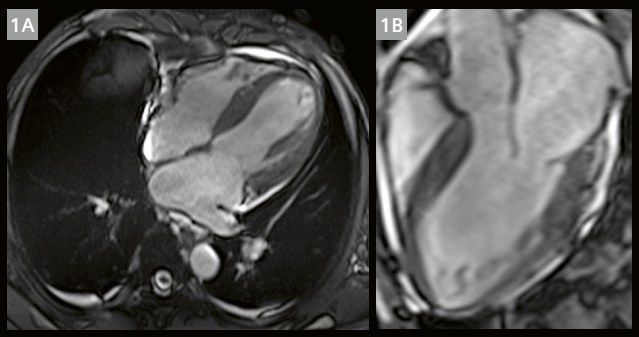
- The extent and pattern of LGE correlate with survival [23].
- Elevated native T1 and ECV reflect higher disease burden and worse outcomes.
- Myocardial edema (T2 elevation) in AL-CA is associated with adverse prognosis [25].

These parameters allow more precise risk stratification and complement clinical and biomarker-based staging systems.

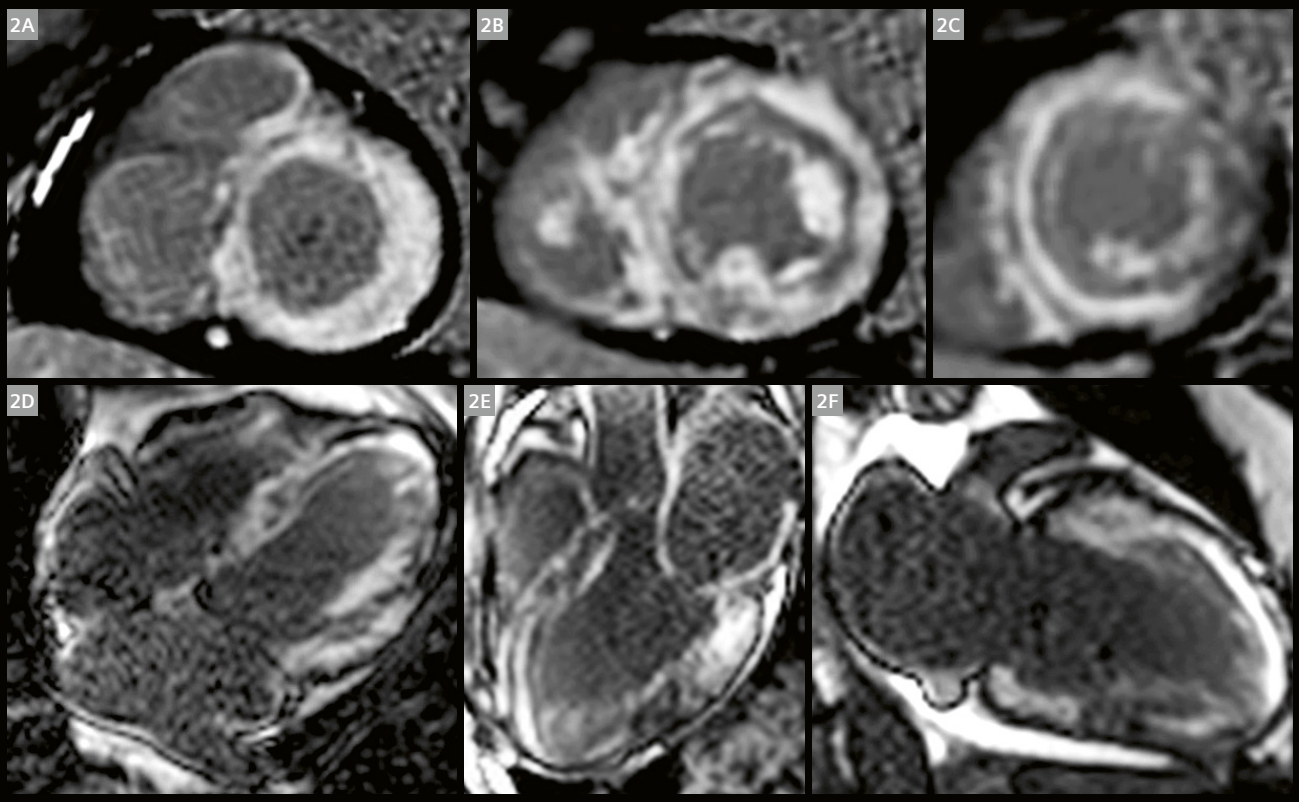
Cardiac amyloidosis should be considered in patients with increased wall thickness in the presence of cardiac or extracardiac amyloidosis red flags and/or in specific clinical situations. Modern diagnostic algorithms allow non-invasive confirmation of ATTR CA when a Perugini grade 2 or 3 bone scintigraphy is combined with negative monoclonal protein testing (in serum and urine) and positive echocardiographic or CMR criteria for CA. AL-CA still typically requires histological confirmation. CMR plays a key supportive role in these algorithms by providing characteristic tissue signatures and helping rule out alternative diagnoses.

CMR findings in cardiac amyloidosis

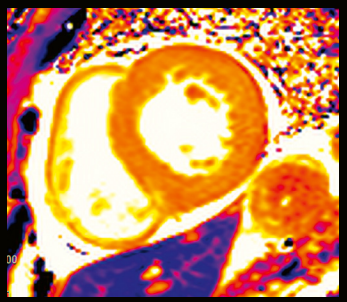
1. Diffuse myocardial thickening (≥ 12 mm): Increased LV wall thickness, typically without significant chamber dilatation. Usually concentric and homogeneous, mimicking hypertrophy. Frequent involvement of the right ventricle and interatrial septum.
2. Typical LGE pattern (a key diagnostic feature): Diffuse circumferential subendocardial LGE or transmural LGE. Does not follow a coronary artery distribution, unlike ischemic disease.
3. Difficulty in appropriately nulling the myocardium on inversion recovery sequences: Abnormal gadolinium kinetics: The myocardium often nulls earlier than, or simultaneously with, the blood pool (rapid contrast wash-in and wash-out due to expanded extracellular space).
4. T1 mapping and extracellular volume (ECV) expansion
5. Additional features: Restrictive filling pattern. Thickened atrioventricular (AV) valves and frequent pericardial and pleural effusions. Marked bi-atrial enlargement. Atrial wall thickening and fibrosis (atrial LGE).



1 **Cine CMR images: Morphological and functional assessment.** **(1A)** 4-chamber view, **(1B)** 2-chamber view. CMR demonstrates diffuse myocardial thickening of the left ventricle with a maximum wall thickness of 16 mm at the basal septum, accompanied by involvement of the right ventricular free wall and the interatrial septum. There is associated bi-atrial enlargement, more pronounced in the left atrium. Small pericardial and bilateral pleural effusions are also present. Biventricular ejection fractions are within normal limits, with a left ventricular ejection fraction of 58% and a right ventricular ejection fraction of 51%. **Despite this, left ventricular function shows reduced longitudinal shortening, indicating impaired longitudinal systolic function.**



2 **Late gadolinium enhancement (LGE) images.** **(2A)** Basal short-axis; **(2B)** mid-ventricular short-axis; **(2C)** apical short-axis; **(2D)** 4-chamber view; **(2E)** 3-chamber view; **(2F)** 2-chamber view. Diffuse transmural LGE is observed in the basal third of the left ventricle. Patchy transmural enhancement is present in the mid-ventricular segments, along with subendocardial enhancement of the apical inferior wall and transmural enhancement of the apical anterior wall. In cardiac amyloidosis, myocardial infiltration of the left ventricle typically progresses from the base toward the apex. Early in the disease course, LGE is predominantly subendocardial, subsequently evolving into a patchy pattern and ultimately becoming transmural in advanced stages. Although the apical segments are often spared initially, advanced disease — as in this case — demonstrates apical involvement. Additional LGE is noted in the atrial walls, interatrial septum, and right ventricular free wall. LGE also revealed a characteristic abnormal myocardial and blood pool signal pattern, with a relatively hypointense (dark) blood pool and absence of normal myocardial nulling.



3 **Native T1 mapping acquired on a 3T MAGNETOM Skyra system using a MOLLI sequence.** A marked elevation in native T1 mapping value was observed (1501 ms), corresponding to a Z-score of +6.5 standard deviations above the normal reference range at our institution (1126–1302 ms). These findings are highly suggestive of amyloidosis and are consistent with the results reported by Baggiano et al. [27], who described that native T1 mapping values with a Z-score greater than 3.5 standard deviations are compatible with amyloidosis, with high sensitivity and specificity, as discussed in the text.

Contemporary MAGNETOM scanners facilitate high-quality CMR in often frail CA patients by enabling fast, robust acquisitions. Free-breathing LGE techniques minimize motion artifacts in patients with low ejection fraction or arrhythmias. Automated planning tools (myExam Cardiac Assist, formerly known as Cardiac Dot Engine) and accelerated mapping sequences improve workflow consistency and patient comfort while delivering excellent tissue characterization. These advances help bring the full diagnostic power of multiparametric CMR to a broader range of patients.

Case presentation

A 69-year-old Afro-Caribbean man from the Netherlands Antilles presented with a four-year history of progressive bilateral lower extremity edema. Laboratory testing revealed preserved renal function (creatinine 0.69 mg/dL) and elevated NT-proBNP (894 pg/mL), consistent with heart failure of unclear etiology.

The ECG demonstrated low-voltage QRS complexes and a first-degree AV block. Additionally, there was poor R-wave progression across the precordial leads, a pattern that may suggest underlying myocardial infiltration or conduction system involvement in the appropriate clinical context.

Transthoracic echocardiography demonstrated concentric left ventricular hypertrophy (interventricular septum: 16 mm; posterior wall: 17 mm), preserved left ventricular ejection fraction (58%), diastolic dysfunction, bi-atrial enlargement, and impaired longitudinal strain predominantly affecting the basal and mid segments.

Clinical indication for CMR: In the presence of multiple red flags suggestive of cardiac amyloidosis, as outlined below, CMR was requested to evaluate for potential infiltrative myocardial disease and to exclude alternative causes of myocardial hypertrophy.

Red flags for cardiac amyloidosis

- Age: 69 years with clinical signs of heart failure (≥ 65 years)
- ECG: Low-voltage QRS complexes, first-degree AV block
- Echo: Left ventricular myocardial thickness of 17 mm in the inferolateral wall (≥ 12 mm)
- Echo: Reduced basal and mid-left ventricular longitudinal strain with relative apical sparing

CMR was performed on a MAGNETOM Skyra 3T scanner.

Findings

1. Cine images: Morphological and functional assessment
CMR demonstrated diffuse myocardial thickening involving both ventricles, as well as the interatrial septum and atrioventricular valve leaflets. There was marked bi-atrial enlargement. Associated findings included mild pericardial and pleural effusions. Left ventricular function showed reduced longitudinal shortening, consistent with impaired longitudinal systolic function.

2. Tissue characterization: LGE imaging
LGE imaging revealed a characteristic abnormal myocardial and blood pool signal pattern, with a relatively hypointense (dark) blood pool and absence of normal myocardial nulling. There was extensive LGE with a diffuse distribution, including subendocardial and transmural patterns, as well as patchy areas of enhancement. Involvement extended beyond the left ventricle to include the right ventricle, both atria, and the interatrial septum.

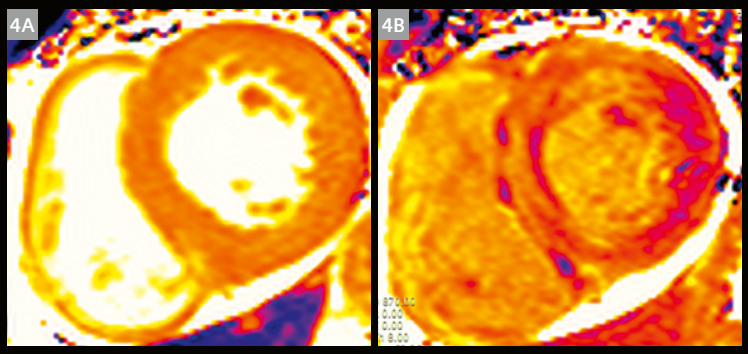
3. Native T1 mapping (non-contrast)
Native T1 values were markedly elevated at 1501 ms, corresponding to a Z-score of +6.5 standard deviations above the institutional normal reference range (1126–1302 ms), indicating significant diffuse myocardial tissue abnormality.

4. Extracellular volume fraction
The calculated ECV fraction was 59%, which is markedly increased compared to normal values for a 3T scanner (< 30%), consistent with extensive expansion of the myocardial extracellular space.

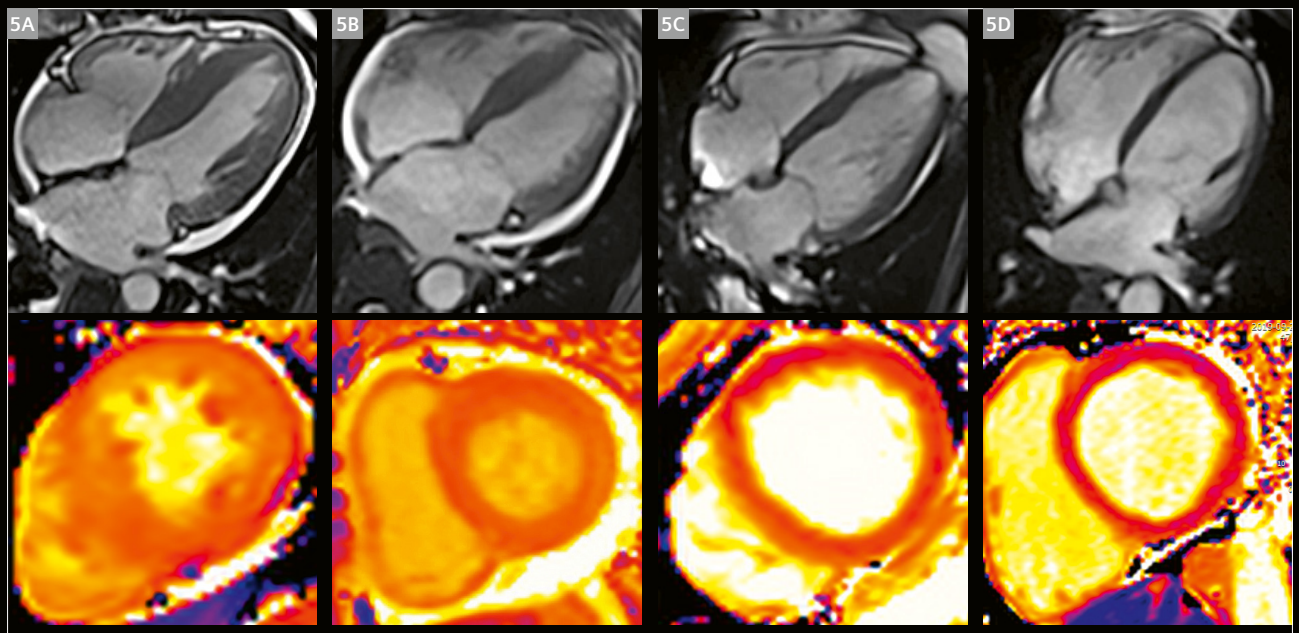
The CMR findings are highly suggestive of cardiac amyloidosis. They demonstrate the characteristic combination of diffuse myocardial thickening, abnormal myocardial nulling on LGE imaging, extensive subendocardial to transmural enhancement, and markedly elevated native T1 and ECV values, reflecting significant expansion of the myocardial interstitium.

Diagnostic value of CMR in cardiac amyloidosis:
CMR plays a pivotal role in the non-invasive diagnosis of both AL and ATTR cardiac amyloidosis, offering high diagnostic accuracy across multiple parameters [27–29]:

- LGE: Sensitivity 85% | Specificity 92%
- Native T1 mapping: Sensitivity 89% | Specificity 80%
- ECV: Sensitivity 93% | Specificity 87%



4 T1 mapping, (4A) native (without contrast); (4B) (after contrast). The calculated extracellular volume fraction (ECV) was 59%. This is markedly elevated compared with normal values for a 3T scanner (normal: < 30%), indicating extensive expansion of the myocardial extracellular space. Such markedly elevated ECV values are highly suggestive of cardiac amyloidosis and are rarely encountered in other conditions. In our experience, similarly high values have only occasionally been observed in patients with decompensated renal failure.



5 Panels 5A–5C show patients with amyloidosis. Panel 5D shows a healthy volunteer. The top-row images correspond to cine MR in the 4-chamber view, and the bottom-row images to native T1 mapping in the short-axis view (acquired on a 3T MAGNETOM Skyra using the MOLLI sequence). These are examples from our native T1 mapping study in patients with amyloidosis and healthy individuals [32]. Note how the myocardial signal color in native (noncontrast) T1 mapping images appears burgundy (maroon) in healthy individuals **(5D)**. As native T1 values increase and Z-scores rise **(5C, 5B, and 5A)** in patients with amyloidosis, the myocardial signal progressively shifts toward yellow. On the 4-chamber cine MR images, note how increasing native T1 values are associated with more evident morphological features of amyloidosis, including increased pericardial effusion, progressive myocardial thickening, interatrial septal thickening, and increasing atrial dilatation.

Clinical relevance and differential diagnosis [30]: Beyond its role in supporting the diagnosis of cardiac amyloidosis, CMR is equally critical in excluding other causes of increased left ventricular wall thickness and hypertrophic phenotypes. These include hypertensive heart disease, sarcomeric hypertrophic cardiomyopathy, Anderson–Fabry disease, and myocardial changes related to chronic pressure overload (increased afterload). Through its unique ability to provide comprehensive tissue characterization, CMR enables accurate differentiation between these entities, guiding appropriate clinical management and prognosis.

At our institution, prior to implementing T1 mapping techniques, we conducted a study (to be published in Revista Colombiana de Radiología) to establish local reference values for native T1 mapping [31]. The study included 60 healthy individuals aged 20 to 70 years (12 subjects per decade, with 6 men and 6 women in each decade). We found that normal native T1 values (non-contrast) using a MAGNETOM Skyra 3T system ranged from 1126 ms to 1302 ms, with two standard deviations corresponding to 88 ms. These reference values allow both the identification of patients with elevated or reduced native T1 values, and the quantification of the magnitude of abnormality using Z-scores. This approach is particularly relevant in patients with cardiac amyloidosis. According to the study by Baggiano et al. [27], patients with native T1 Z-scores greater than 3.5 standard deviations have a positive predictive value of 98% for the diagnosis of amyloidosis (both AL and ATTR). In a small pilot study conducted at our institution prior to the implementation of T1 mapping (also to be published in Revista Colombiana de Radiología) [32], we evaluated 8 patients with amyloidosis and 18 healthy controls. We found that a native T1 Z-score greater than 3.25 yielded a specificity of 100% and a sensitivity of 87.5% for the diagnosis of amyloidosis in this small cohort. These results are consistent with those reported by Baggiano et al. [27]. Additionally, as described by Kraenzusch et al. [33], T1 analysis based on Z-score mapping provides consistent results without significant variability related to magnetic field strength, CMR systems, or modified Look-Locker inversion recovery (MOLLI) sequence variants. Importantly, Z-score mapping detects cardiac amyloidosis with diagnostic accuracy comparable to conventional T1 analysis, while enabling standardized comparison of native T1 values acquired with MOLLI across different CMR platforms.

Laboratory and hematologic workup

Evaluation for monoclonal gammopathy showed elevated serum-free kappa light chains (34.10 mg/L) with a borderline abnormal kappa/lambda ratio (1.66). However, serum and urine immunofixation were negative, and no monoclonal spike was identified on protein electrophoresis. Systemic evaluation for amyloid deposition, including minor salivary gland and abdominal fat biopsy, was negative. Bone marrow biopsy revealed 0.7% polytypic plasma cells with negative Congo red staining.

Bone scintigraphy (99mTc-MDP)

Bone scintigraphy demonstrated myocardial tracer uptake, with a Perugini score of 3, consistent with a scintigraphic study suggestive of ATTR amyloidosis.

Given that monoclonal protein studies suggest AL amyloidosis, while bone scintigraphy findings are indicative of ATTR amyloidosis, the patient requires an endomyocardial biopsy for definitive diagnosis.

Definitive diagnosis

Endomyocardial biopsy demonstrated amorphous eosinophilic material with positive Congo red staining, confirming amyloid deposition. Mass spectrometry identified TTR-derived amyloid, and protein sequencing revealed a pathogenic variant (Val122Ile / p.Val142Ile). Genetic testing confirmed hereditary ATTR amyloidosis.

Discussion

CMR played a central role in our case by raising suspicion for cardiac amyloidosis through characteristic findings such as diffuse subendocardial or transmural LGE, abnormal myocardial nulling, and increased native T1 mapping values and extracellular volume. Additionally, CMR allowed exclusion of other potential causes of myocardial thickening in our patient.

This case highlights the diagnostic challenge of cardiac amyloidosis, particularly in the presence of monoclonal gammopathy of undetermined significance (MGUS), which may confound differentiation from AL amyloidosis, as initially occurred in our patient based on laboratory findings (elevated serum-free kappa light chains = 34.10 mg/L with a borderline abnormal kappa/lambda ratio = 1.66). A patient with positive monoclonal protein studies suggestive of AL amyloidosis requires biopsy, regardless of the results of bone scintigraphy.

The identification of the Val122Ile (Val142Ile) mutation is particularly relevant, as it represents a common pathogenic variant among individuals of African ancestry and is strongly associated with late-onset cardiac involvement.

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