



Delayed Diagnosis of Transthyretin Cardiac Amyloidosis in Recurrent Heart Failure with Preserved Ejection Fraction: A Narrative Review of Multimodality Imaging Pathways

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Abstract

Transthyretin amyloid cardiomyopathy (ATTR-CM) is no longer a rare curiosity of the autopsy room. It is a treatable cause of heart failure with preserved ejection fraction (HFpEF) that is still recognised years after the first decompensation, by which time irreversible myocardial infiltration has occurred and the therapeutic window has narrowed.

Objectives: *To review the published evidence on the diagnostic performance and optimal sequencing of electrocardiography, echocardiography with deformation imaging, cardiovascular magnetic resonance and bone-avid radiotracer scintigraphy in patients presenting with recurrent HFpEF, and to identify where in that pathway diagnostic delay is generated.*

Material and Methods: *A narrative review was undertaken. MEDLINE, Embase and the Cochrane Library were searched from January 2005 to May 2026 for prevalence studies, diagnostic accuracy studies, consensus recommendations and randomised outcome trials in ATTR-CM. Prevalence estimates, sensitivity and specificity values and trial endpoints were extracted from the primary reports and tabulated without pooling.*

Results: *Reported ATTR-CM prevalence in HFpEF cohorts with ventricular wall thickening ranges from 3% to 16.8%, driven largely by age, wall-thickness threshold and whether screening was systematic. Median diagnostic delay is 3.4 years in wild-type disease and misdiagnosis is reported in 34-57% of patients. Grade 2 or 3 uptake on bone scintigraphy with absence of a monoclonal protein carries a specificity and positive predictive value of 100%, removing the need for biopsy in most patients. Relative apical sparing of longitudinal strain and elevated extracellular volume identify infiltration earlier than wall thickness alone, but neither distinguishes transthyretin from light-chain deposition.*

Conclusion: *The tools required to identify ATTR-CM in HFpEF are widely available and well validated; the delay is generated by failure to suspect the diagnosis rather than by any deficiency of the tests. A protocolised pathway triggered by clinical red flags, in which immunofixation with free light chains is requested at the same time as scintigraphy, is the single change most likely to shorten time to treatment.*

Abbreviations: ATTR-CM – Transthyretin Amyloid Cardiomyopathy, HFpEF – Heart Failure with Preserved Ejection Fraction, CMR – Cardiovascular Magnetic Resonance, ECV – Extracellular Volume, LGE – Late Gadolinium Enhancement, RALS – Relative Apical Longitudinal Strain.

Keywords: Transthyretin Amyloidosis; Heart Failure with Preserved Ejection Fraction; Multimodality Imaging; Bone Scintigraphy

Introduction

Heart failure with preserved ejection fraction accounts for roughly half of all heart failure and has, for most of its history, been managed as a single syndrome defined by what it is not. That

framing has been overtaken by the recognition that a substantial minority of these patients have a specific, identifiable and now treatable infiltrative cardiomyopathy. The first prospective screening study of its kind found wild-type transthyretin amyloid in

13.3% of consecutive patients aged 60 or over admitted with HFpEF and a wall thickness of at least 12 mm [1].

Autopsy series had already suggested the pathology was common, with left ventricular amyloid present in a meaningful fraction of hearts from patients dying with a HFpEF phenotype and overwhelmingly of transthyretin type [2]. Screening in a community cohort in southeastern Minnesota identified ATTR-CM in 6.3% of consenting patients, far above the clinically recognised rate [3]; European work reports 8% aged over 60 [4] and 16.8% aged 75 or over [5], while an all-comers Dutch cohort found only 3% [6].

The consequence of missing the diagnosis changed fundamentally in 2018. Tafamidis reduced all-cause mortality from 42.9% to 29.5% over 30 months [7], and acoramidis and the RNA-interference agent vutrisiran have since produced concordant benefits [8,9]. All three prevent further amyloid formation rather than clearing established deposits, so the benefit is a function of how much myocardium remains: diagnostic delay is a direct determinant of outcome.

Delay nevertheless remains the rule: wild-type ATTR-CM was diagnosed more than four years after first cardiac presentation in 42% of a large referral cohort, whose patients had used hospital services a median of 17 times in the preceding three years [10]. This review examines what each modality contributes in recurrent HFpEF, where time is lost, and how the sequence might be rearranged.

Material and Methods

Search strategy: MEDLINE, Embase and the Cochrane Library were searched from January 2005 to May 2026. Search terms combined controlled vocabulary and free text for transthyretin amyloidosis, cardiac amyloidosis, heart failure with preserved ejection fraction, bone scintigraphy, cardiovascular magnetic resonance and speckle-tracking echocardiography. Reference lists of consensus documents and position statements were hand-searched.

Eligibility and synthesis: prevalence studies in HFpEF or unexplained left ventricular hypertrophy, diagnostic accuracy studies with a histological or composite reference standard, multisociety consensus recommendations and randomised outcome trials were eligible; case reports, unpublished abstracts and light-chain-only studies were excluded. Values were extracted by one reviewer and checked by a second. Because the studies differ in setting, age, wall-thickness threshold and reference standard, results are presented descriptively without pooling. No individual patient data were accessed and ethics approval was not required.

Results

Prevalence and the size of the unrecognised population

Reported prevalence varies roughly five-fold across published cohorts (Figure 1), and the spread is explained less by geography

than by case definition. Studies restricted to older patients with established wall thickening report the highest figures: 16.8% in a nationwide Spanish study of patients aged 75 or over [5] and 13.3% in the Madrid cohort with a mean age of 82 [1]. Cohorts admitting younger patients, more women or no wall-thickness threshold report nearer 3-8% [4,6]. Screening before transcatheter aortic valve implantation has found concomitant amyloid pathology in a comparable range, with adverse prognostic consequences [11,12].

Whichever estimate applies, the implied number of unrecognised patients is large: in the Minnesota study, systematic screening produced a prevalence roughly an order of magnitude above the contemporaneous clinical diagnosis rate [3]. A six-variable clinical score derived from that work selects which patients warrant scintigraphy and classified 11-35% of male participants in contemporary HFpEF trials as high risk [13].

Where the delay is generated

A targeted literature review of 23 studies found weighted mean and median diagnostic delays of 6.1 and 3.4 years respectively in wild-type disease, and 5.7 and 2.6 years in the hereditary form, with misdiagnosis documented in 34-57% of patients where it was reported (Figure 2) [13,15]. Evaluation by multiple specialties before diagnosis is the norm rather than the exception, and seven clinical factors predict delay, of which a prior label of hypertensive heart disease or hypertrophic cardiomyopathy is the most consequential [16].

The pattern is one of anchoring: once a plausible alternative explanation for wall thickening exists, evaluation stops, and comorbidities common at this age increase the odds of delay precisely because they supply that explanation. Extracardiac clues are correspondingly undervalued. Bilateral carpal tunnel syndrome typically precedes cardiac presentation by 5 to 10 years, and amyloid is demonstrable in tenosynovial tissue removed at release in a substantial minority of older patients [17,18]. Lumbar spinal stenosis, biceps tendon rupture and unexplained atrial arrhythmia carry similar weight (Table 1).

Electrocardiography and conventional echocardiography

The teaching that ATTR-CM produces low QRS voltages is only partly true and is a recognised source of false reassurance. Low voltage is far more characteristic of light-chain disease; in transthyretin disease the reliable finding is discordance between voltage and mass, voltages being inappropriately low for the degree of wall thickening rather than absolutely low [22,33]. Pseudo-infarct patterns and conduction disease are common and should prompt rather than close the enquiry.

Conventional echocardiography establishes the phenotype but not the aetiology. Increased wall thickness, biatrial dilatation, valvular thickening, a small pericardial effusion, restrictive filling and reduced tricuspid annular plane systolic excursion with a

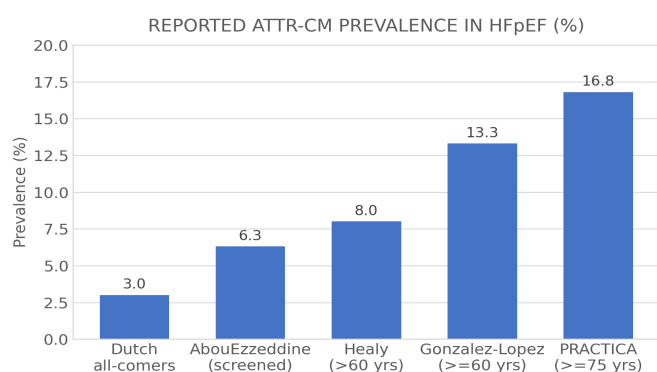


Figure 1: Reported prevalence of transthyretin amyloid cardiomyopathy across published cohorts of patients with heart failure with preserved ejection fraction.

preserved ejection fraction constitute a characteristic picture, and multiparametric echocardiographic scores built on these variables perform respectably as screening instruments [19,20]. None distinguishes transthyretin from light-chain deposition, and none should be used to exclude the diagnosis in a patient with red flags.

Deformation imaging and the apical sparing pattern

Speckle-tracking adds discriminatory power. The characteristic finding is a base-to-apex gradient of impairment with relative preservation of apical deformation, and the relative apical longitudinal strain ratio derived from it was reported as both sensitive and specific for cardiac amyloidosis against other causes of hypertrophy [21]. In practice it is a strong prompt rather than a diagnostic test: it occurs in both transthyretin and light-chain disease and is neither perfectly sensitive early nor perfectly specific late. Global longitudinal strain is independently prognostic once the diagnosis is made [20].

Cardiovascular magnetic resonance

CMR contributes tissue characterisation. Global subendocardial or transmural late gadolinium enhancement with abnormal gadolinium kinetics is characteristic and carries independent prognostic weight [23]. Native T1 rises before wall thickening is established, and quantification of the myocardial extracellular volume gives a continuous measure of amyloid burden that tracks severity and responds to treatment [24,25,26]. In a large transthyretin cohort, ECV and the enhancement pattern both predicted mortality independently of conventional markers [27].

Two limitations matter here. CMR cannot separate transthyretin from light-chain amyloid, so a positive study still requires haematological and nuclear assessment; and renal impairment, implanted devices and claustrophobia exclude or degrade a meaningful proportion of exactly the elderly population in question. CMR is therefore best placed where the phenotype is uncertain, an alternative infiltrative disease is under consideration, or quantification of burden will inform prognosis.

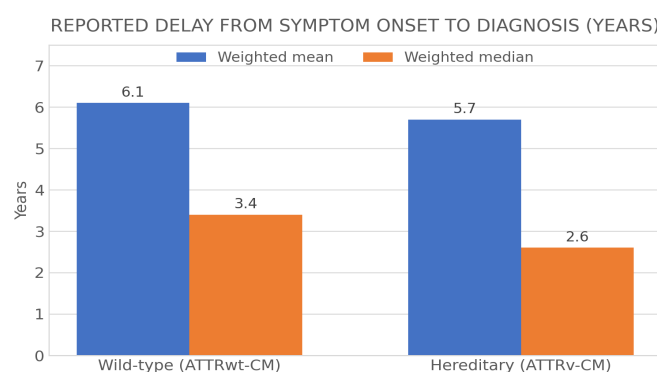


Figure 2: Reported delay from symptom onset to diagnosis in transthyretin amyloid cardiomyopathy, by disease subtype, from a targeted review of 23 studies [14].

Domain	Red flag
History	Bilateral carpal tunnel syndrome; lumbar spinal stenosis; spontaneous biceps tendon rupture
Demographics	Age over 65 years; male sex; heart failure disproportionate to comorbid burden
Electrocardiography	QRS voltage low relative to wall thickness; pseudo-infarct pattern; conduction disease
Echocardiography	Wall thickness ≥ 12 mm without adequate hypertensive explanation; biatrial dilatation; relative apical sparing of longitudinal strain
Clinical course	Intolerance of beta blockade or renin-angiotensin blockade; discordantly elevated troponin; recurrent decompensation

Table 1: Clinical and imaging red flags that should trigger evaluation for ATTR-CM in a patient with heart failure with preserved ejection fraction.

Bone-avid radiotracer scintigraphy

The observation that technetium-labelled bone tracers localise to myocardial amyloid, and that uptake can be graded against rib uptake, transformed the pathway [28]. In a multicentre study of 1,217 patients with suspected cardiac amyloidosis, myocardial uptake was more than 99% sensitive and 86% specific, with false positives arising almost entirely from light-chain disease. Grade 2 or 3 uptake combined with absence of a monoclonal protein gave a specificity and positive predictive value of 100%, lower confidence bound 98.0% [29]; grade 2 or 3 uptake alone gave 91% and 87% (Figure 3, Table 2).

That result removed the requirement for endomyocardial biopsy in most patients and is now embedded in multisociety recommendations and the European position statement [30,31]. Two caveats are routinely underemphasised: the reported accuracy derives from specialist referrals with high pre-test probability, so extrapolation to unselected wall thickening is not automatic; and the criteria are conditional on the haematological arm, since scintigraphy read without concurrent immunofixation and free light chains is not the validated test.

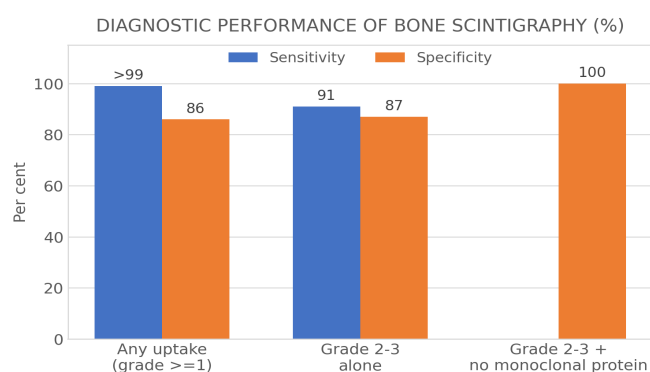


Figure 3: Sensitivity and specificity of bone-avid radiotracer scintigraphy for cardiac ATTR amyloidosis under three diagnostic criteria [29]. The third criterion adds absence of a monoclonal protein in serum and urine.

Criterion	Sens.	Spec.	PPV
Any myocardial uptake (grade ≥ 1)	> 99%	86%	–
Grade 2 or 3 uptake alone	91%	87%	–
Grade 2 or 3 uptake with no monoclonal protein	–	100%	100%

Table 2: Reported diagnostic performance of bone-avid radiotracer scintigraphy for cardiac ATTR amyloidosis in a multicentre cohort of 1,217 patients with suspected cardiac amyloidosis [29]. Grade refers to myocardial uptake scored against rib uptake.

Exclusion of a plasma cell dyscrasia and genotyping

Serum and urine immunofixation with a free light chain assay must be requested at the same time as, not after, the scintigram; protein electrophoresis alone is insufficiently sensitive. A monoclonal protein does not exclude ATTR-CM, since monoclonal gammopathy of undetermined significance is common at this age, but its presence returns the patient to a pathway requiring histology and typing [30,32].

Once ATTR-CM is confirmed, TTR genotyping is mandatory. It separates wild-type from variant disease, which differ in natural history, neurological burden and family implications, and determines whether cascade screening is required [31,33].

Staging and the value of an early diagnosis

Two validated staging systems, one using NT-proBNP with estimated glomerular filtration rate and one using troponin T with NT-proBNP, stratify survival at diagnosis [34,35]. Their relevance here is that stage at diagnosis is largely a function of how long the diagnosis took: analysis of 20 years of referrals to a national centre showed a progressive shift towards earlier stage at presentation, with improved survival that persisted after adjustment [36].

Discussion

The evidence assembled here supports one conclusion: the tests required to diagnose ATTR-CM in recurrent HFrEF are accurate, widely available and inexpensive relative to the cost of the

Trial	Agent	n	Principal result
ATTR-ACT [7]	Tafamidis	441	All-cause mortality 29.5% vs 42.9% at 30 months; HR 0.70
ATTRibute-CM [8]	Acoramidis	632	Win ratio 1.8 on hierarchical endpoint at 30 months
HELIOS-B [9]	Vutrisiran	654	Death or recurrent CV events HR 0.72; mortality HR 0.65 at 42 months
APOLLO-B [37]	Patisiran	360	Functional capacity and quality of life preserved at 12 months

Table 3: Randomised outcome trials of disease-modifying therapy in transthyretin amyloid cardiomyopathy. Endpoints are as reported in the primary publications and are not directly comparable across trials.

disease, and the delay is generated almost entirely upstream of them. No modality reviewed lacks the performance to do its job; what is missing is the decision to order the test.

Rather than a sequential cascade in which each negative step licenses the next, the evidence favours a trigger-based pathway in which defined red flags in a patient with HFrEF and wall thickness of 12 mm or more prompt simultaneous request of scintigraphy, immunofixation and free light chains. Ordering the haematological arm concurrently rather than reactively converts a positive scan into a diagnosis at the same clinic visit rather than weeks later [29,30].

Therapeutic consequences of earlier diagnosis

Three randomised trials now anchor treatment (Table 3). Tafamidis reduced all-cause mortality over 30 months, the curves separating only after about 18 months, and cut cardiovascular hospitalisation from 0.70 to 0.48 per year [7]. Acoramidis was superior on a hierarchical endpoint of death, hospitalisation, NT-proBNP and six-minute walk distance, win ratio 1.8 [8]. Vutrisiran reduced the composite of mortality and recurrent cardiovascular events (hazard ratio 0.72) and mortality through 42 months (0.65, Figure 4) [9], while patisiran preserved function and quality of life at 12 months without a demonstrated mortality benefit over that shorter horizon [37].

The delayed separation of the survival curves across all three trials is the point of greatest relevance here: a therapy whose benefit appears at 18 months confers little on a patient diagnosed with a median survival of similar length. Post-hoc analysis of tafamidis by stage found the largest absolute benefit at stage I and a diminishing, eventually non-significant effect at stage III [38]. Earlier diagnosis is therefore a precondition for these treatments working as they did in trial populations.

Study limitations

This is a narrative rather than a systematic review: studies were selected against stated criteria but without duplicate screening of

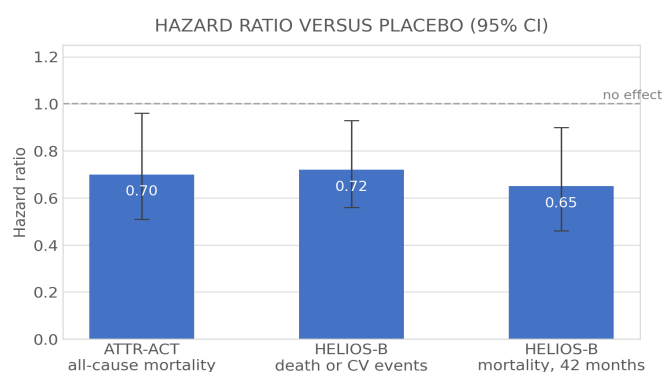


Figure 4: Hazard ratios with 95% confidence intervals for mortality and composite endpoints in randomised outcome trials of ATTR-CM. Values below 1.0 favour active treatment.

the full search yield, and no risk-of-bias instrument was applied. Prevalence figures come from cohorts differing in age, sex, wall-thickness threshold and screening strategy, so Figure 1 describes that heterogeneity rather than providing a defensible pooled estimate. Accuracy data derive predominantly from specialist centres and probably overstate performance elsewhere. The review addresses diagnosis rather than management, and does not cover the arrhythmic, anticoagulation and transplantation questions that follow.

Conclusion

Transthyretin amyloid cardiomyopathy is present in a clinically significant minority of patients with recurrent HFpEF and ventricular wall thickening, and is now treatable with agents that alter survival. The modalities available to identify it perform well: bone scintigraphy with exclusion of a plasma cell dyscrasia is definitive in most patients without histology, while deformation imaging and extracellular volume quantification detect infiltration before wall thickness declares it. Persistent delay reflects failure of suspicion, not failure of the tests. A protocolised pathway triggered by red flags, with the haematological arm requested concurrently and genotyping following confirmation, is achievable within existing resources.

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