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(acoramidis)

BAYER

Transthyretin Amyloid Cardiomyopathy (ATTR-CM) Signs, Symptoms and Diagnostic Pathways

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BEYONTTRA is indicated for the treatment of wild-type or variant transthyretin amyloidosis in adult patients with cardiomyopathy (ATTR-CM).

What is transthyretin (TTR)?

TTR is a highly conserved tetrameric protein, essential for transporting vitamin A and is involved in various physiological functions.[1–3]

What is ATTR-CM?

Transthyretin amyloid cardiomyopathy (ATTR-CM) is a progressive cardiac condition that begins with the destabilisation of the TTR tetramer.[2,4–6]

1

TTR is produced in the liver, brain and eyes[2,4]

2

Unstable TTR tetramer dissociates into monomers[4,5]

3

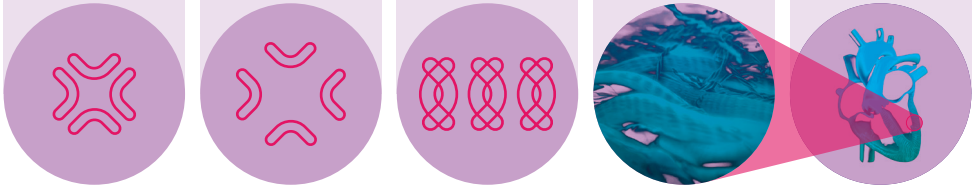
Monomers misfold and aggregate into insoluble amyloid fibrils[4,6]

4

Amyloid fibrils get deposited in the heart extracellularly[4,6]

5

Heart walls thicken and stiffen. Progressive decline in cardiac function, leading to heart failure symptoms[2,4,6]



There are two forms of ATTR-CM: **wild-type ATTR-CM (ATTRwt-CM)** and **variant ATTR-CM (ATTRv-CM)**.[4]
The average age of onset is 75 years (usually >60 years) for ATTRwt-CM and 30–80 years for ATTRv-CM, depending on the mutation.[4]

ATTR-CM is an underdiagnosed cause of heart failure which requires early diagnosis and treatment[7–10]

Without timely intervention, ATTR-CM could lead to impaired quality of life, recurrent hospitalisations and early death.[8–10]

The **median survival** from diagnosis in untreated patients is **~2.6 years** in ATTRv-CM V122I and **3.6 years** in ATTRwt-CM.[10,11]

ATTR-CM affects
11 in 100
HFpEF patients[7]

When to suspect ATTR-CM

ATTR-CM can be suspected if the left ventricular (LV) wall thickness is ≥12 mm and one or more red flags are present.[12]

*LV wall thickness **≥12 mm** + ≥1 **Red Flag** = **Suspect** cardiac amyloidosis

*Women have naturally thinner septal walls; the ≥12mm ATTR-CM cutoff may delay diagnosis if applied without sex-specific consideration.[13]

Red flags[12]

Musculoskeletal

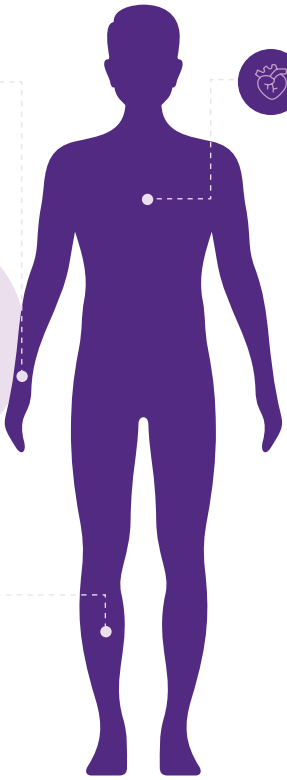
TTR amyloid deposition in ligaments starts several years before the first cardiac symptoms[14]

- Bilateral carpal tunnel syndrome **(38–49%)**[15,16]
- Ruptured biceps tendon **(33%)**[17]
- Lumbar spinal stenosis **(20–48%)**[16,18]

Bilateral carpal tunnel syndrome is the most common and early non-cardiac symptom (8-10 years before first cardiac symptoms)[20]

Nervous system

- Peripheral polyneuropathy
- Sensory involvement
- Autonomic dysfunction



Cardiac

- Heart failure or aortic stenosis (≥65 years)
- Unresponsive/ intolerant to standard heart failure therapies[19]
- Hypotensive or normotensive if previously hypertensive
- Possible family history of ATTR-CM

Adapted from Garcia-Pavia, P, et al. 2021.[12]

Prescribing Information and Adverse Event Reporting for Beyonttra® can be found on the front page.

Early detection of cardiac manifestations is key in ATTR-CM diagnosis[12]

- Heart failure is the most common symptom of ATTR-CM[21,22]
- Presence of HFpEF (particularly in men) and intolerance to ACE inhibitors or beta blockers are also diagnostic clues for ATTR-CM[23]

ECG observations[12]

- AV block in presence of increased LV wall thickness
- Pseudo Q waves
- Reduced QRS voltage to mass ratio
- Atrial fibrillation/ rhythm disorders

Changes in CMR[12]

- Increased extracellular volume
- Diffuse late subendocardial/ transmural LGE
- Abnormal nulling time for the myocardium

Echocardiogram abnormalities[12]

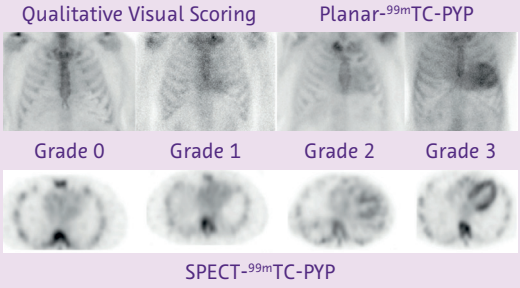
- Reduction in longitudinal strain with apical sparing
- Hypertrophic phenotype with associated infiltrative features, e.g. increased thickness of AV valves, interatrial septum and RV free wall

Scintigraphy grading: Confirming diagnosis

Evaluation of ^{99m}Tc-PYP/DPD/HMDP images[24]

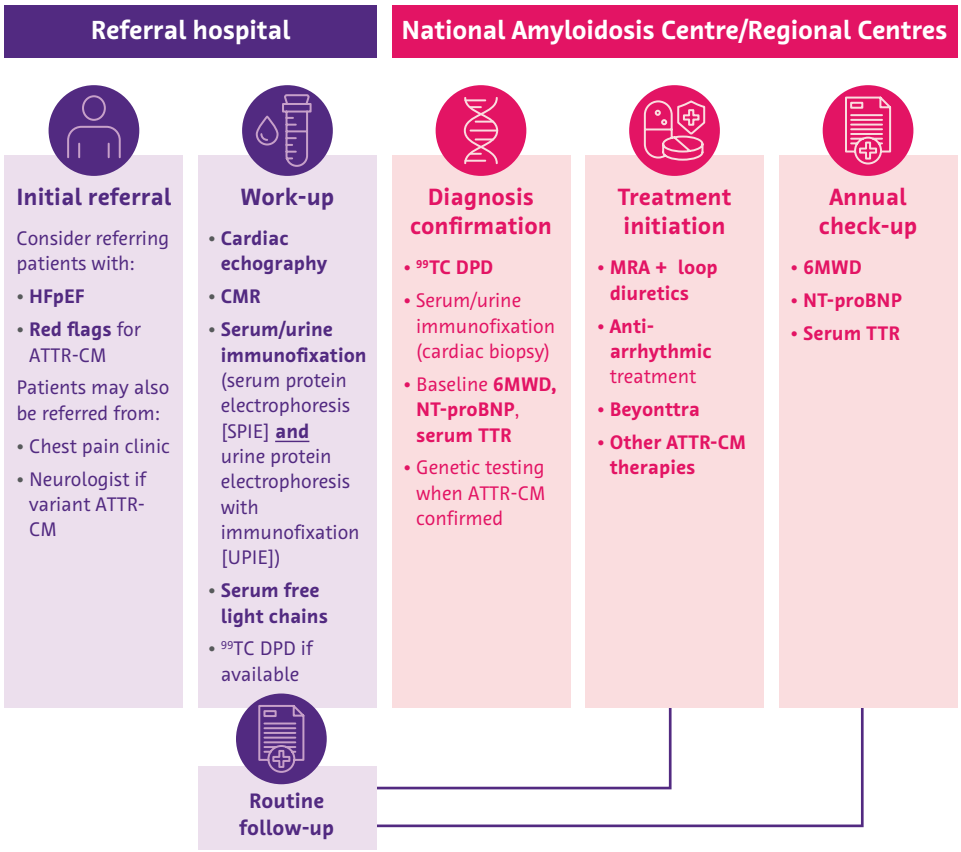
Visual grading of scintigraphy findings in ATTR-CM

Grade 0 Absent cardiac uptake	
Grade 1 Cardiac uptake < rib†	
Grade 2 Cardiac uptake = rib	**Diagnostic**
Grade 3 Cardiac uptake > rib	**Diagnostic**

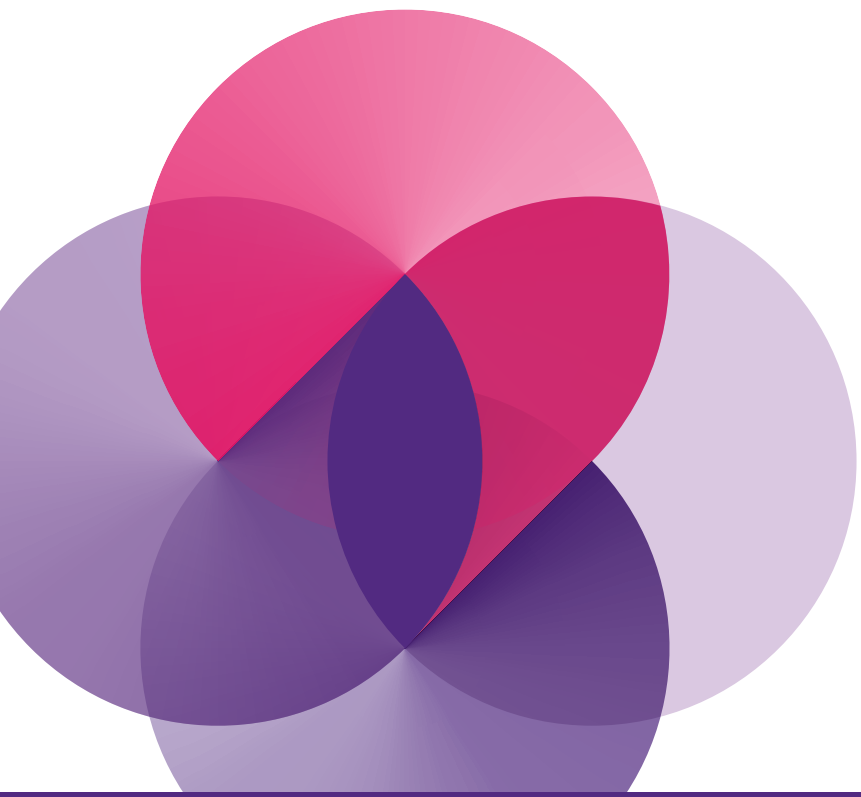


Must confirm myocardial uptake using SPECT imaging[25]

The ATTR-CM patient pathway



This treatment pathway has been derived from the opinions of ATTR-CM Health care experts included in the United Kingdom ATTR-CM steering committee organised and funded by Bayer.



* Reprinted from Journal of the American College of Cardiology, volume 73 (issue 22), Ruberg FL, et al, Transthyretin Amyloid Cardiomyopathy: JACC State-of-the-Art Review, pages 2872–2891, copyright 2019, with permission from the American College of Cardiology.
† Histological confirmation (cardiac/extracardiac) to diagnose.

6MWD, 6-minute walk distance; **^{99m}Tc,** Technetium-99m; **ACE,** angiotensin-converting enzyme; **AL,** light-chain amyloidosis; **ATTR-CM,** transthyretin amyloid cardiomyopathy; **ATTRv-CM,** variant transthyretin amyloid cardiomyopathy; **ATTRwt-CM,** wild-type transthyretin amyloid cardiomyopathy; **AV,** atrio-ventricular; **CMR,** cardiac magnetic resonance; **CoE,** centre of excellence; **DPD,** 3,3-diphosphono-1,2-propane-dicarboxylic acid; **EC,** extracellular; **ECG,** electrocardiogram; **HFpEF,** heart failure with preserved ejection fraction; **HMDP,** hydroxymethylene diphosphonate; **LGE,** late gadolinium enhancement; **LV,** left ventricular; **MRA,** mineralocorticoid receptor antagonist; **MRI,** magnetic resonance imaging; **NT-proBNP,** N-terminal pro-B-type natriuretic peptide; **PYP,** pyrophosphate; **RV,** right ventricular; **TTR,** transthyretin.

1. Liz MA, et al. Neurol Ther. 2020;9(2):395–402; **2.** Vieira M, Saraiva MJ. Biomol Concepts. 2014;5(1):45–54; **3.** Gertz MA, et al. Ann Med.2025;57(1):2536755; **4.** Ruberg FL, et al. J Am Coll Cardiol. 2019;73(22):2872–2891; **5.** Hammarstroem P, et al. Proc Natl Acad Sci USA. 2002;99(Suppl 4):16427–16432; **6.** Tschoepe C, et al. J Clin Med. 2025;14(13):4785; **7.** Magdi M, et al. Am J Cardiovasc Dis. 2022;12(3):102–111; **8.** Rintell D, et al. Orphanet J Rare Dis. 2021;16(1):70; **9.** Nativi-Nicolau J, et al. ESC Heart Fail. 2021;8(5):3875–3884; **10.** Lane T, et al. Circulation. 2019;140(1):16–26; **11.** Grogan M, et al. J Am Coll Cardiol. 2016;68(10):1014–1020; **12.** Garcia-Pavia P, et al. Eur J Heart Fail. 2021;23(4):512–526; **13.** Vogel J, et al. Eur Heart J. 2025;46(Suppl 1):1006; **14.** Westin O et al. J Am Coll Cardiol. 2022;80:967–977; **15.** Elghouneimy MA, et al. Cureus. 2024;16(12):e75582; **16.** Achten A, et al. Heart Vessels. 2024;39(10):857–866; **17.** Geller HI, et al. JAMA. 2017;318(10):962–963; **18.** Tamasauskas D, et al. Front Neurol. 2024;15:1425862; **19.** Ioannou A, et al. Eur Heart J. 2023;44(31):2893–2907; **20.** Gertz MA et al. J Am Coll Cardiol 2015;66:2451–2466; **21.** Brito D et al. Global Heart. 2023;18(1):59; **22.** Witteles RM et al. JACC Heart Failure. 2019;7(8):709–716; **23.** Maurer MS, et al. Circ Heart Fail. 2019;12:e006075. **24.** Dorbala S, et al. JACC Cardiovasc Imaging. 2020;13(6):1368–1383; **25.** Benz DC et al. J Nucl Cardiol. 2025;51:102448.

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