# Scintigraphy showing the possible progression of transthyretin cardiac amyloidosis

Alessandro Graziani[1], Eleonora del Giudice[2], Matteo Lisi[3], Marco Domenicali[1], Ludovico Graziani[4]

1 Internal Medicine Department, Ravenna Hospital, Ravenna, Italy
2 Department of Nuclear Medicine, Faenza Hospital, Faenza, Italy
3 Department of Cardiology, Ravenna Hospital, Ravenna, Italy
4 Catholic University of the Sacred Heart Rome, Rome, Italy

In cardiac amyloidosis (CA), myocardial infiltration by amyloid fibrils causes structural and functional changes that lead to heart failure, angina, and arrhythmias (atrial fibrillation, conduction disorders).[1]

Light-chain amyloidosis and transthyretin amyloidosis (ATTR; wild type or hereditary) account for most cases of heart involvement in amyloidosis.[2]

There is an increasing interest in using noninvasive cardiac imaging in the diagnostic workup of CA. Echocardiography can demonstrate various diastolic abnormalities in the setting of left ventricular wall thickening, and the apical sparing strain pattern may differentiate CA from other causes of left ventricular hypertrophy.

In cardiac magnetic resonance, late gadolinium enhancement, T1 mapping, and myocardial extracellular volume are used in the diagnostic workup and prognosis in CA.[4]

Studies demonstrated that 99m technetium (Tc)–3,3-diphosphono-1,2-propanodicarboxylic acid (99mTc-DPD) and 99m Tc-pyrophosphate (99mTc-PYP) have high affinity for ATTR uptake and thus can differentiate this form from amyloidosis. The mechanism of uptake seems related to the high calcium content in ATTR amyloid deposits. Using these bone tracers in nuclear scintigraphy may ultimately render biopsy unnecessary to diagnose CA in patients with ATTR. [5]

Here, we describe an 84-year-old man with a history of diet-controlled type 2 diabetes mellitus, hypertension, heart failure, and percutaneous coronary intervention due to acute coronary syndrome that occurred 3 years before. At that time, high-sensitivity troponin T concentration was 90 ng/l (reference range, <15 ng/l) and transthoracic echocardiography revealed left ventricular wall thickening (13 mm), preserved left ventricular ejection fraction of 55%, and signs of abnormal diastolic relaxation pattern. Transthoracic echocardiography performed 12 years before showed a similar pattern of left ventricular hypertrophy attributed to long-standing hypertension. 99mTc-DPD scintigraphy did not demonstrate significant cardiac uptake suggestive of ATTR (FIGURE 1A).

At the admission to our institution, the patient experienced pain and swelling in the right shoulder, lumbar pain, and weakness of the legs. Blood cultures were positive for a methicillin-sensitive *Staphylococcus aureus* and magnetic resonance imaging of the spine was consistent with L4–L5 spondylodiscitis. Transthoracic echocardiography did not reveal signs of endocarditis, but showed a left ventricular wall thickening (14 mm), left ventricular ejection fraction of 48%, left atrial dilatation, and moderate mitral valve regurgitation. During the hospitalization, he developed acute cardiogenic pulmonary edema treated with high-dose diuretics and noninvasive mechanical ventilation. Transesophageal echocardiography demonstrated severe functional mitral regurgitation with no evidence of valvular endocarditis. After 2 weeks, the patient gradually improved and further transthoracic echocardiography revealed a significant reduction of mitral regurgitation. Prior to discharge, he underwent 99mTc-DPD scintigraphy that was indicative of ATTR (FIGURE 1B). Laboratory tests showed the N-terminal pro–brain natriuretic peptide level of 18 628 ng/l and high-sensitivity troponin T, 188 ng/l. Since the patient's age indicated wild-type ATTR, we did not perform genetic testing.

**Correspondence to:**
Alessandro Graziani, MD, Internal Medicine Department, Ravenna Hospital, Ravenna, Italy, phone: +39 0544285322, email: alessandro.graziani@auslromagna.it
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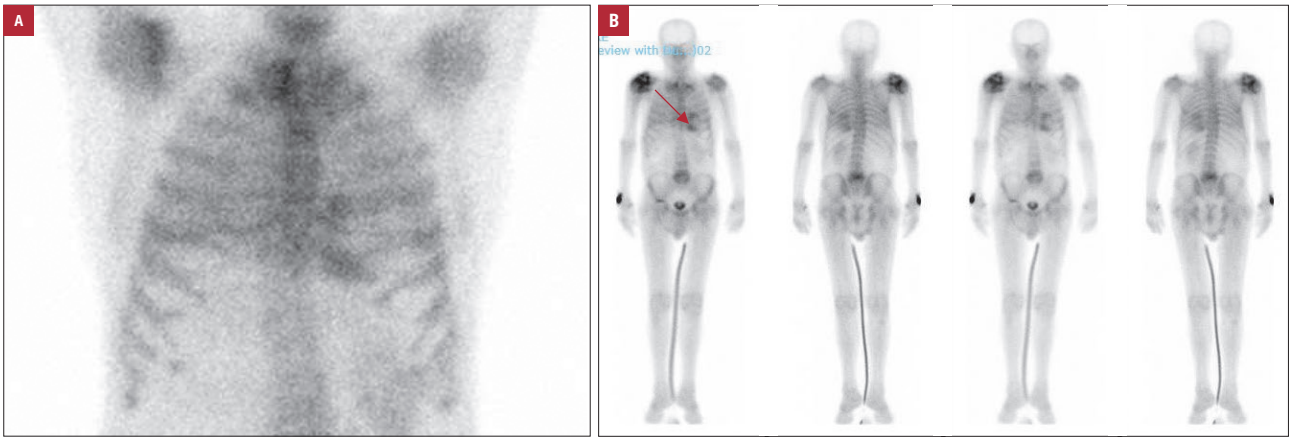


**FIGURE 1** Scintigraphy at 180 minutes after the administration of the tracer: **A** – the anterior planar view of the chest showing no significant cardiac uptake; **B** – the whole-body view indicating the myocardial uptake of the tracer, typical of transthyretin amyloidosis (arrow)

In ATTR, repeat scintigraphy could identify a greater myocardial amyloid burden that determines a clearer diagnostic imaging. Even if the specific wild-type ATTR therapy is actually only supportive, the diagnosis allows a better prognostic stratification and therapeutic strategies.

## ARTICLE INFORMATION

**CONFLICT OF INTEREST** None declared.



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