

In the early diagnosis of cardiac amyloidosis by point-of-care ultrasound (POCUS) in older patients with heart failure: towards a new standard of care?

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It is estimated that approximately one in ten people over the age of 80 may suffer from cardiac amyloidosis (CA), a disease in which various aging-related factors, such as increased oxidative stress, can promote abnormal protein folding and the formation of amyloid deposits in the heart. Over the long term, this tends to impair cardiac function, increasing the risk of developing cardiac conduction disorders, atrial fibrillation, thromboembolic events, heart failure (HF), and/or ventricular dysfunction.^[1,2]

Although CA has traditionally been considered underdiagnosed, its clinical implications have driven a paradigm shift in its identification in recent years.^[3] Nevertheless, the nonspecific symptoms of the disease and the difficulty in accessing the necessary complementary tests for its detection have posed a challenge for its early diagnosis in daily clinical practice.^[4] We present a case in which point-of-care ultrasound (POCUS) performed by the geriatrician was crucial to the early identification of this disease.

This 89-year-old patient, previously independent in her basic activities of daily living and without cognitive impairment, has given her written consent for the publication of her clinical information and images presented in this case report. Her only notable medical history includes hypertensive heart disease with preserved left ventricular ejection fraction (LVEF), left bundle branch block, and carpal tunnel syndrome. She presented to the Emergency Department with progressive dyspnea on minimal exertion, paroxysmal nocturnal dyspnea, olig-

uria, and palpitations accompanied by a sensation of chest tightness. Physical examination revealed rhythmic heart sounds, bibasilar crackles and bilateral lower limb pitting edema. Laboratory findings showed troponin T of 318 ng/L and N-terminal pro-B-type natriuretic peptide of 15,185 pg/mL. She was admitted to the Acute Geriatric Unit with a diagnosis of a HF episode and non-ST-segment elevation acute coronary syndrome.

On the day after admission, the patient experienced symptomatic worsening and *de novo* atrial fibrillation. POCUS revealed significant global ventricular hypertrophy and hyperechogenicity, suggestive of CA; as well as mitral insufficiency, left atrial dilation, and a qualitative subjective visual evaluation consistent with a reduced LVEF (Figure 1A). Once the patient was stabilized, a cardiac gammagraphy was performed, which demonstrated biventricular and extracardiac radiotracer deposits (Grade 3 on the Perugini scale) (Figure 1B). Hematological studies revealed no significant abnormalities. The patient progressed favorably and was discharged home, with direct oral anticoagulation being initiated.

The patient was next seen in the cardio-geriatric outpatient clinic for a comprehensive and collaborative multidisciplinary assessment by Cardiology and Geriatrics specialists. She was clinically and functionally stable, and a transthoracic echocardiogram performed by the cardiologist showed a high correlation with the POCUS results obtained during her admission. Additionally, other echocardiographic findings suggestive of CA were observed, including thickening of the interatrial septum,

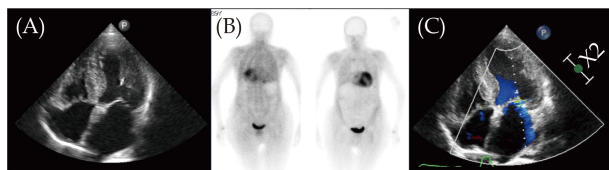


Figure 1 Early diagnosis of cardiac amyloidosis. (A): Point-of-care ultrasound; (B): cardiac gammaigraphy; and (C): transthoracic echocardiogram.

myocardial “sparkling,” mild pericardial effusion, and a restrictive pattern (Figure 1C). With the confirmation of the affected LVEF, treatment with intravenous iron and sodium-glucose cotransporter-2 inhibitor was initiated. The test for the transthyretin gene was negative.

Subsequent evaluations confirmed the patient's good clinical progress. Other cardiac-targeted drugs (for HF with reduced ejection fraction or specifically for the selective stabilization of transthyretin in CA) should not be titrated due to the persistent reduced LVEF and tendency to hypotension. After twelve months, the patient remains stable and has not required any hospital readmissions.

In this case, the information provided by POCUS, together with other red flags of CA, made its diagnosis highly probable and led to the performance of pertinent confirmatory tests without the need for a cardiac biopsy.^[3] Additionally, the early identification of amyloidosis guided by POCUS allowed for better-targeted pharmacological treatment of HF. On one hand, the treatment with the angiotensin-converting enzyme inhibitor (usually poorly tolerated in these patients due to their tendency toward arterial hypotension) was suspended. Similarly, the use of drugs with potentially deleterious binding to amyloid fibers was also avoided. The identification of CA in such patients may be useful by alerting clinicians to use beta-blockers with special caution, as they should be prescribed with particular care in patients with chronotropic incompetence.^[4]

It is crucial to have a high clinical suspicion to facilitate early and accurate diagnosis of CA. In this regard, our clinical experience has shown that POCUS can be useful not only for early diagnosis but also for therapeutic management of older patients with heart disease.^[5] It is particularly helpful in the detection of CA as it reveals, from greater to lesser complexity, symmetric left ventricular hypertrophy, biatrial dilatation, interatrial septal thickening, mild pericardial effusion, and the characteristic myocardial granular pattern or “sparkling”.^[6]

In conclusion, this case illustrates how POCUS optimizes treatment, enabling more efficient use of time and resources, and thereby improving the therapeutic approach in cardiogeriatrics. In our opinion, this case underscores the importance of incorporating POCUS into the diagnosis and treatment of older adults with HF.

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