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Cardiac Amyloidosis Associated with Multiple Myeloma, Revealed by Heart Failure with Preserved Ejection Fraction in A 56-Year-Old Man: Case Report

SOW Salif[1*], NAHAL Salma[1], SOUMARE Ahamadou[1], ALLAOUI Lala Ghita[1], GOITE Adama[1], LOUDIYI Nadia[1], Pr NAJAT Mouine[1], Pr AATIF Benyass[1]

[1]Clinical Cardiology Department, Service Des Examens Non Invasifs, Cardiology Center, Hôpital Militaire Mohammed V, Rabat, Morocco

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***Corresponding author:** SOW Salif
Clinical Cardiology Department, Service Des Examens Non Invasifs, Cardiology Center, Hôpital Militaire Mohammed V, Rabat, Morocco

Abstract **Case Report**

We report the case of a 56-year-old man with cardiovascular risk factors, including statin-treated dyslipidemia and long-standing smoking, who presented with acute decompensated heart failure. Clinical, biological, immunohistological and imaging examinations led to the diagnosis of cardiac amyloidosis, a frequently underdiagnosed cause of heart failure with preserved ejection fraction, and multiple myeloma requiring multidisciplinary management.

Keywords: Heart failure, cardiac amyloidosis, multiple myeloma, echocardiography, cardiac MRI, multidisciplinary.

INTRODUCTION

Although rare, cardiac amyloidosis represents a significant but often overlooked cause of heart failure, with preserved left ventricular ejection fraction. AL amyloidosis, commonly associated with multiple myeloma, can be difficult to diagnose due to its non-specific clinical presentation. Diagnosis relies on a combination of clinical, biological and imaging examinations.

CASE REPORT

A 56-year-old man with cardiovascular risk factors (statin-treated dyslipidemia and a history of smoking) and no significant past history was admitted to hospital with progressively worsening exertional dyspnea (NYHA stage III), eventually progressing to stage IV with paroxysmal nocturnal dyspnea and orthopnea, with edema of the lower limbs, dry cough and marked asthenia.

On admission, clinical examination revealed

- heart rate 112 bpm, blood pressure 128/84 mmHg.
- Oxygen saturation 84% on room air.
- Signs of right heart failure (peripheral edema).
- Bilateral auscultatory rales, no neurological signs.

The ECG (Figure 1) showed sinus tachycardia at 114 bpm, with QS in the anterior leads and negative T waves in the inferolateral leads.

As part of the diagnostic work-up

Chest X-ray (Fig. 2) showed an alveolar-interstitial syndrome with right basal infection and scissuritis, complemented by thoracic angioscan (Fig. 3), which revealed a pulmonary mosaic, a moderate bilateral pleural effusion (more prominent on the left), a small pericardial effusion and no pulmonary embolism. The patient underwent antibiotic therapy for a total of 10 days.

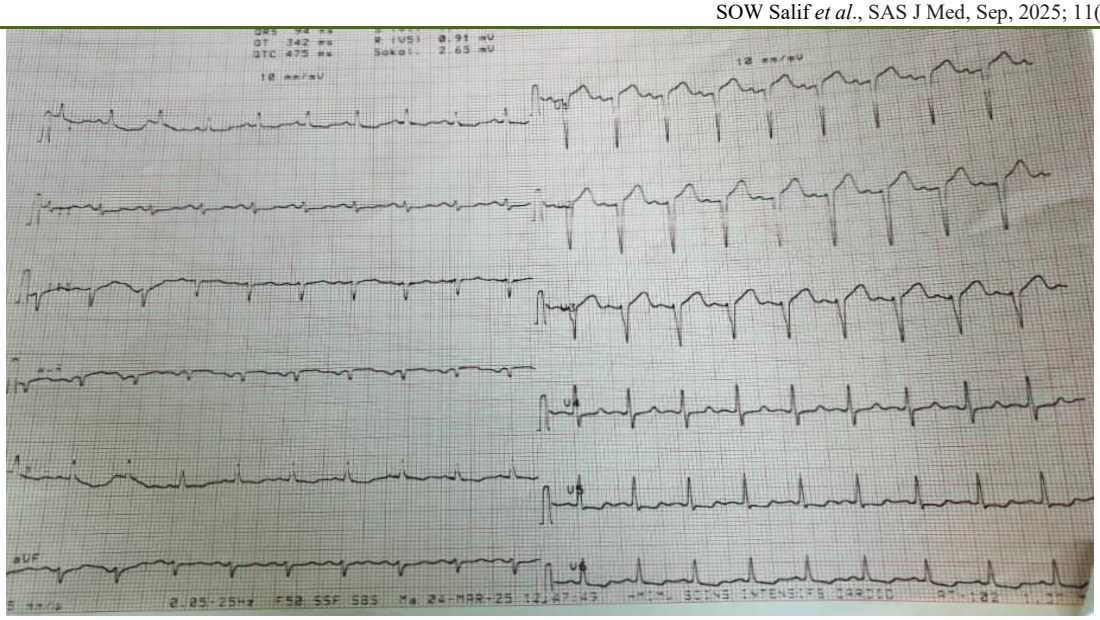

Figure 1: ECG showing microvoltage, sinus tachycardia, and electrical changes.

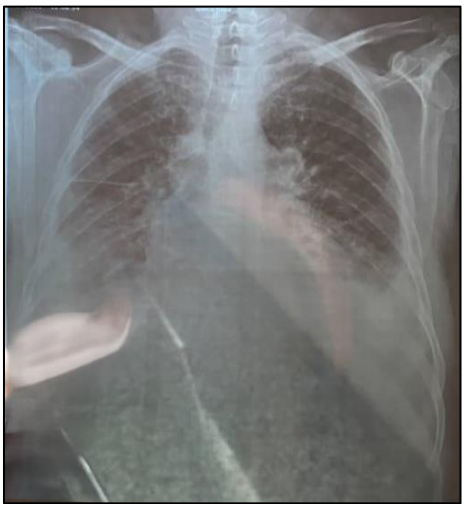

Figure 2: Chest X-ray showing alveolar-interstitial syndrome and basal infection of the right lung.

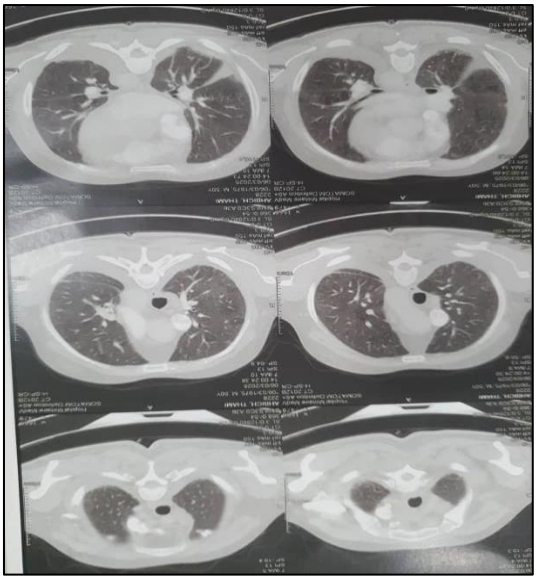

Figure 3: Chest angioscan showing pulmonary mosaic and bilateral pleural effusion.

Transthoracic echocardiography (figure 4): showed a non-dilated left ventricle with concentric hypertrophy, preserved ejection fraction, moderately altered overall longitudinal strain (-13.2%) with a pattern (apical sparing) suggestive of amyloidosis. To confirm the diagnosis, a cardiac MRI scan (figure 5) was carried out: in favour of cardiac amyloidosis, with a non-dilated LV showing diffuse myocardial hypertrophy, preserved systolic function at 55%, and diffuse late sub-endocardial enhancement of the LV, atria and SIA. Coronary angiography revealed no coronary artery disease. The Holter ECG performed as part of the search for electrical complications was unremarkable.

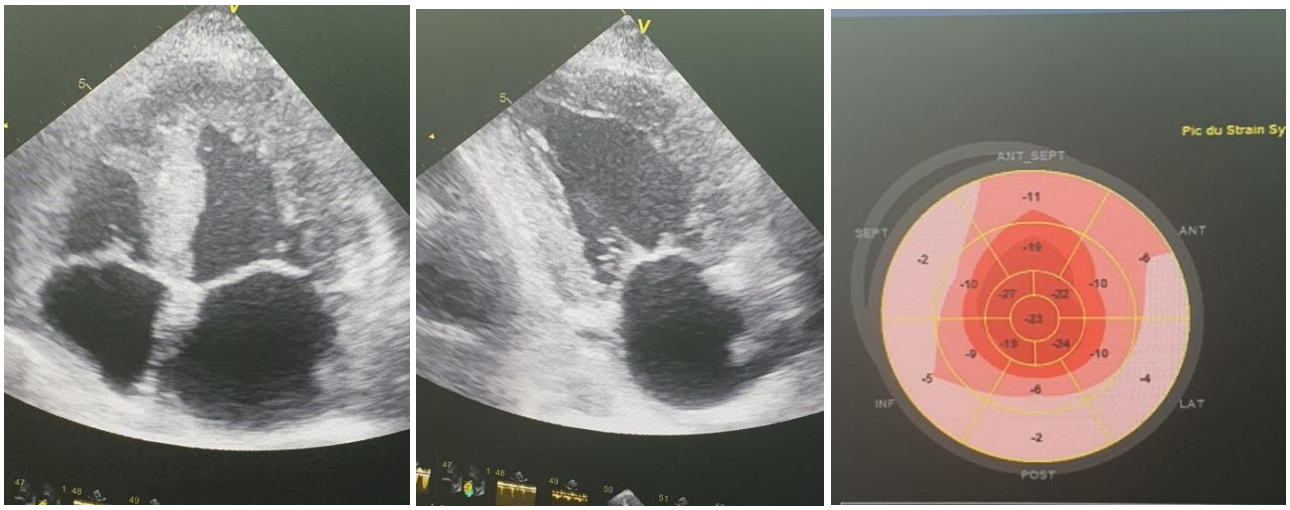

Figure 4: Transthoracic echocardiography: showing left ventricular hypertrophy and moderately altered global longitudinal strain (-13.2%) with an apical sparing pattern

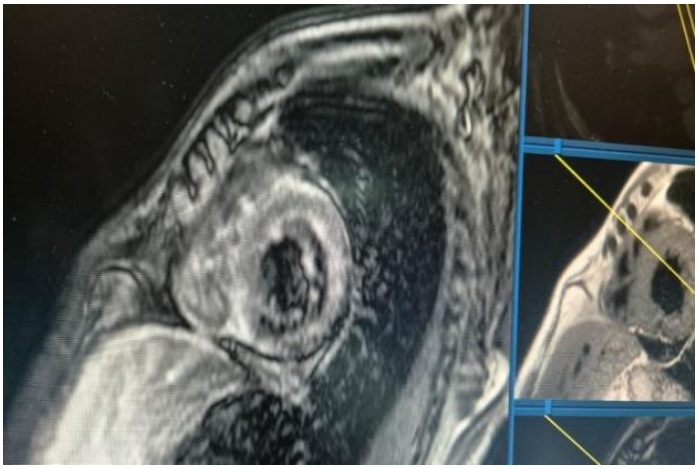

Figure 5: Cardiac MRI: non-dilated LV with diffuse myocardial hypertrophy, preserved systolic function at 55%, diffuse late sub-endocardial enhancement of the LV, atria and SIA

Biological work-up revealed CRP 36.3 mg/l, troponin 84 ng/ml, NT-proBNP 868 pg/ml, negative infectious cultures, an unremarkable ionogram and renal work-up, a disturbed hepatic work-up, the presence of a lambda monoclonal IgG peak at 11.4 g/L, a very low kappa/lambda ratio of 0.01 and a bone marrow plasmacytosis of 20%. The diagnosis of multiple myeloma associated with cardiac amyloidosis was made after multidisciplinary consultation between cardiology and haematology.

The patient was put on chemotherapy for multiple myeloma. Despite treatment, he remained symptomatic, with persistent dyspnea and oxygen dependence, and was eventually transferred to the hematology department for specialized care.

DISCUSSION

This case highlights an atypical presentation of cardiac amyloidosis associated with multiple myeloma, initially masked by pneumonitis. The combination of preserved LVEF, altered strain and myocardial hypertrophy on MRI should suggest amyloidosis. Diagnosis of both entities relies on complementary immunological and histological examinations. Early diagnosis is essential, as it determines the prompt initiation of specific hematological treatment. Multiple myeloma and AL amyloidosis both have a poor prognosis when associated. AL amyloidosis is known as

a classic complication of multiple myeloma, affecting 10-15% of people with multiple myeloma [1]. These are similar diseases, but with different outcomes, and may benefit from different therapeutic approaches once remission has been achieved [3]. Amyloid light chain amyloidosis is a rare disease caused by the extracellular deposition of misfolded immunoglobulin light chains [4-7]. Renal or cardiac involvement is the most common, although any organ outside the central nervous system may develop an amyloid deposit, and symptomatic manifestations may therefore vary. [5.6.8]. Cardiac AL amyloidosis is a potentially life-threatening condition due to its rhythmic complications. Prognosis is linked to NT pro BNP and troponin levels, enabling cardiac involvement to be classified according to the Mayo-clinic classification [9]. Cardiac involvement is considered a major prognostic factor. Echocardiography should be performed routinely in patients with confirmed amyloidosis [6]. At the time of diagnosis of AL amyloidosis, most patients present with isolated monoclonal gammopathy or multiple myeloma [10]. Symptomatic or active multiple myeloma is characterized by clonal expansion of plasma cells in the bone marrow, the detection in most cases of monoclonal immunoglobulin in serum and/or urine, and the presence of target organ lesions [11]. It accounts for 1-2% of all cancers and 10-12% of hematological malignancies. Its annual incidence is estimated at around 2-4 new cases per 100,000 inhabitants. The median age at diagnosis is 65; it rarely occurs before the age of 40 (2%). Around 10% of patients with multiple myeloma have associated AL amyloidosis. The survival of patients with AL amyloidosis and multiple myeloma is significantly less than that of patients with AL amyloidosis alone [12]. The principle of treatment is to reduce the protein load by acting on the underlying monoclonal proliferation. Prognosis depends on the reduction in serum free light chain concentration [13].

CONCLUSION

This case highlights the need for a multidisciplinary approach to the diagnosis and treatment of complex pathologies. The management of heart failure associated with multiple myeloma and cardiac amyloidosis represents a real challenge. Rigorous monitoring and adaptation of treatments according to the patient's clinical course are essential to improve the patient's quality of life.

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