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AN UNUSUAL CAUSE OF UNSTABLE ANGINA: ENDOMYOCARDIAL FIBROSIS

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ABSTRACT

Although rare globally, endomyocardial fibrosis (EMF) is the leading cause of restrictive cardiomyopathy in some tropical countries. However, its aetiology and pathogenesis remain unclear. Epidemiology data are limited but the highest prevalence has been reported in tropical regions, specifically sub-Saharan Africa.

EMF is characterized by apical filling with fibrotic tissue in one or both ventricles, often associated with thrombus and calcification. Most often it extends to the posterior leaflet of the mitral valve, papillary muscle or chordae tendineae, causing atrioventricular valve dysfunction.

Transthoracic echocardiography is the first-line modality for assessment, but magnetic resonance imaging has emerged as a more accurate tool for the tissue characterization of this disease.

KEYWORDS

Endomyocardial fibrosis, MRI diagnosis, tropical diseases, restrictive cardiomyopathy

LEARNING POINTS

- Consider endomyocardial fibrosis in patients with restrictive cardiomyopathy and a tropical origin or eosinophilia.
- Cardiac magnetic resonance imaging is essential for non-invasive diagnosis and assessment of fibrosis, calcification, and ventricular involvement.
- Microvascular angina may be an unusual initial presentation of endomyocardial fibrosis.

INTRODUCTION

Endomyocardial fibrosis (EMF) is a form of restrictive cardiomyopathy characterized by fibrotic thickening of the endocardium and subendocardial myocardium, which leads to impaired ventricular filling and diastolic dysfunction. Although EMF is relatively rare on a global scale, it is a significant cause of restrictive cardiomyopathy in certain tropical regions, with the highest prevalence reported in parts of Africa, India, Brazil, and Central America[1].

EMF commonly affects the apical and subvalvular regions of the ventricles, leading to fibrotic changes that can be detected through imaging studies. Cardiac magnetic

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resonance imaging (MRI) has proven to be a particularly useful tool, providing detailed information on the extent of fibrosis, ventricular structure, and functional impairment[2]. However, despite improvements in imaging techniques and our understanding of the disease, early or mild cases may remain asymptomatic or present in an unusual manner, making diagnosis challenging.

Here, we report the case of a Congolese patient who presented to the emergency department with exertional angina, representing an unusual presentation of a mild case of endomyocardial fibrosis. We discuss the diagnostic evaluation, including MRI findings, and the subsequent medical management, highlighting the importance of considering EMF in the differential diagnosis of restrictive cardiomyopathies in tropical regions.

CASE DESCRIPTION

We report the case of a 46-year-old woman born in the Republic of Congo, who has lived in Spain for the past 20 years. She described a severely unbalanced diet, deficient in multiple essential nutrients, and reported having lived under extremely poor personal and community hygiene conditions in her country of origin. She did not recall any relevant illnesses during childhood or adolescence. Her medical history includes hypertension, dyslipidaemia, and diabetes. Her current treatment includes atorvastatin and amlodipine. She works as a caregiver for the elderly.

The patient came to the emergency department for episodes of non-radiating sensation of oppression in the centre of the chest during the previous 3 months, which occurred during exertion and subsided completely at rest, without associated sweating. The reason for the visit was that on the same day she experienced a 20-minute episode at rest that was more intense than the previous ones. No infectious symptoms or fever and no other cardiovascular symptoms were noted.

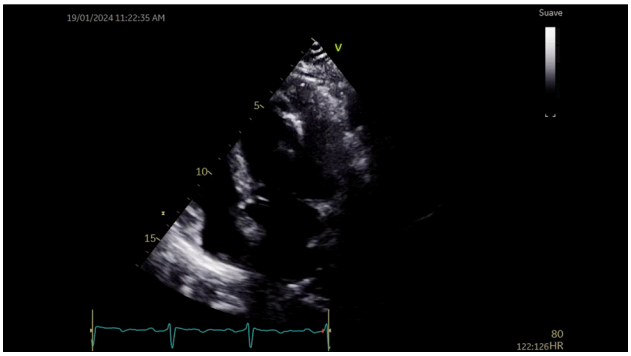
The patient had no peripheral oedema, ascites, pleural effusion or pericardial effusion. Physical examination was normal with blood pressure at 140/80 mmHg, heart rate 80 bpm, basal oxygen saturation of 98%.

An electrocardiogram was performed, showing sinus rhythm with steep QRS complexes and negative T waves from V3 to V6. No dynamic changes were observed *(Fig. S1)*.

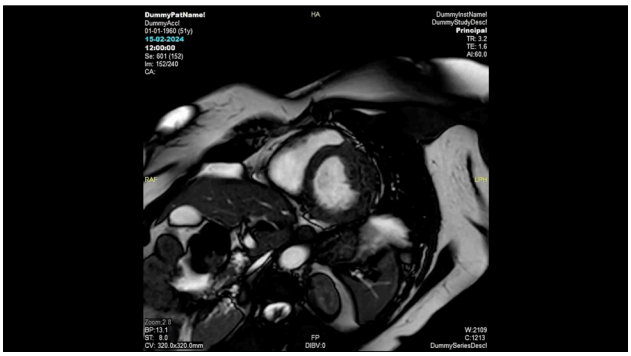
Laboratory parameters showed eosinophilia (11%, 0.8 $\times 10^9$/l; normal range: 1–5%) with eosinophils exhibiting bilobed nuclei and large cytoplasmic granules. Serology was positive for IgG against *Strongyloides stercoralis*. The rest of the laboratory parameters were normal.

The complete blood count revealed a hemoglobin level of 14.2 g/dl, white blood cell count of 6.8 $\times 10^9$/l with 43% neutrophils (2.9 $\times 10^9$/l) and 39% lymphocytes (2.7 $\times 10^9$/l), and a platelet count of 222 $\times 10^9$/l.

Serum biochemistry showed a glucose level of 90 mg/dl and glycated hemoglobin of 5.9%. Renal function parameters were within normal limits, with creatinine 0.67 mg/dl, estimated glomerular filtration rate 101.29 ml/min/1.73 m^2, and urea 30 mg/dl. Serum electrolytes were normal: sodium 141 mmol/l, potassium 4.5 mmol/l, and chloride 106 mmol/l. Angiotensin-converting enzyme levels were within the normal range (26 U/l).



Video 1. Apical 4 chamber echocardiography view.
https://youtu.be/veEuT_rSaGE



Video 2. Short-axis view of cardiac magnetic resonance imaging.
https://youtu.be/8QjwXCooLDU



Video 3. Cardiac magnetic resonance imaging showing apical 4-chamber view.
https://youtu.be/CGhIVWGxt80

Inflammatory markers were low, with C-reactive protein 0.14 mg/dl and erythrocyte sedimentation rate 12 mm/h. Cardiac biomarkers showed a high-sensitivity troponin level of 8.4 ng/l, within the reference range. The coagulation profile was normal, with an international normalized ratio of 1.06.

The lipid profile was as follows: triglycerides 50 mg/dl, low-density lipoprotein cholesterol 74 mg/dl, and high-density lipoprotein cholesterol 57 mg/dl. Liver function tests were within normal limits: aspartate aminotransferase 21 U/l, alanine aminotransferase 28 U/l, total bilirubin 0.54 mg/dl (direct bilirubin 0.24 mg/dl), alkaline phosphatase 58 U/l, and lactate dehydrogenase 198 U/l. Total serum protein was 7.0

g/dl, with albumin 3.8 g/dl.

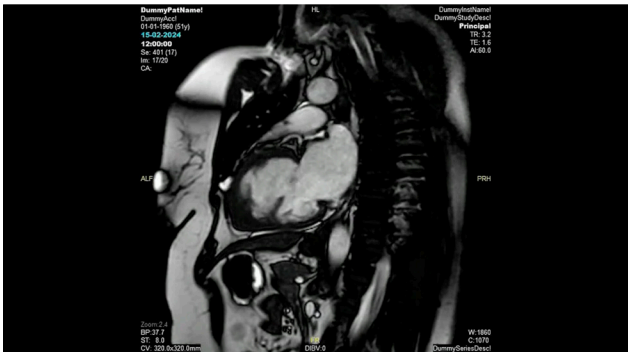
Calcium and phosphate metabolism were normal, with calcium 9.6 mg/dl and phosphorus 3.7 mg/dl. Iron studies showed serum iron 84 µg/dl, transferrin 226 mg/dl, transferrin saturation 30%, and ferritin 133 ng/ml. Thyroid function tests were also normal, with thyroid-stimulating hormone 1.21 µIU/ml and free thyroxine (T4) 1.04 ng/dl.

Chest X-ray was normal. Echocardiography indicated that the left ventricle was not dilated (end diastole volume 110 ml) with a normal global systolic function (56% by Simpson). There were no alterations in segmental contractility, and the parietal thickness of the basal septum was normal (9-10 mm). However, there was marked thickening in the middle and apical segment of the lateral and apical segments (13 mm) with calcified endocardial areas suggestive of fibrosis *(Video 1 and S1)*. The apical region showed marked obliteration with no apical expansion. There was a diastolic pattern of impaired relaxation, with an E/e' ratio of 8.13. The left atrium was slightly dilated (34 ml/m^2). The mitral valve exhibited severe apical displacement of the papillary muscles, causing tension on the chordae and the posterior mitral leaflet, leading to mild insufficiency (vena contracta: 3 mm). The right ventricle was normal in size and function, with a peak systolic velocity by Doppler tissue imaging of 10 cm/s, a shortening fraction of 40%, and a basal diameter of 33 mm. Right ventricular wall thickness was measured at 5 mm. No other abnormalities were observed in the study. Cardiac catheterization revealed no lesions in the coronary arteries *(Video S2)*.

MRI was performed using T1, T2 fat suppression and white blood sequences (2C, 3C 4C, short-axis) which showed the findings described in the echocardiogram *(Video 2 and 3)*. T2 sequences showed an absence of inflammation with hypointense areas compatible with calcification at that level *(Video S3)*. A perfusion study was conducted, revealing areas of subendocardial hypoperfusion in multiple LV segments. Intracavitary thrombus was not observed *(Video 4)*. A late gadolinium enhancement study showed subendocardial enhancement in the thickened apex and lateral segments *(Fig. 1)*.

The patient declined to undergo an endomyocardial biopsy; therefore, the differential diagnosis was established based on clinical, laboratory, and imaging findings, without histopathological confirmation. Differential diagnosis of EMF includes other more prevalent entities that may present similar echocardiographic images[2]. In particular, these includes mitral valve disease, dilated cardiomyopathy, Ebstein's anomaly, apical hypertrophic cardiomyopathy (HCM) and cardiac amyloidosis.

Apical HCM may be the most difficult differential diagnosis. In apical HCM, there is almost exclusive hypertrophy of the apical segments and a gradual obliteration of the left ventricle (LV) cavity towards the true apex resulting in an ace-of-spades diastolic configuration. Additionally, there are not early extensive intramyocardial calcifications. In our case, there was a sudden obliteration of the apical cavity of



Video 4. Apical 2 chamber echocardiography view. https://youtu.be/Ovgv5b4bv0Q

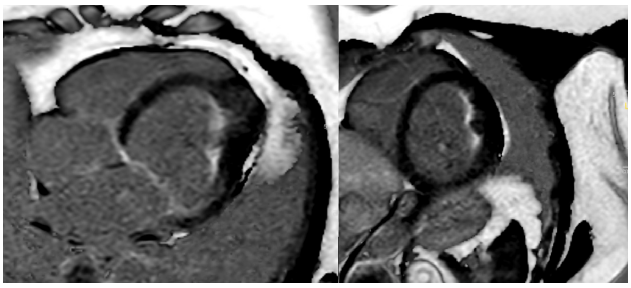


Figure 1. Four chamber, short axis cardiac magnetic resonance imaging with late gadolinium enhancement.

the LV both during diastole and systole, along with several localized calcifications. Late gadolinium enhancement pattern in our case is subendocardial, which is uncommon in HCM. LV apical thrombosis is another potential differential diagnosis[3]. While the endocardium adjacent to the thrombus in ischemic cardiomyopathy is usually hypo or akinetic in EMF, the systolic inward motion of the cardiac apex is typically preserved[3].

Cardiac amyloidosis may manifest as diffuse hypertrophy but no apical obliteration with fibrothrombotic mass deposition is observed, which is hallmark feature of EMF[4]. Our patient did not exhibit right ventricular involvement, however marked atrial enlargement with severe cavity obliteration of the right ventricle may mimic Ebstein anomaly[5]. Additionally, the same mechanism of fibrosis may cause confusion with hammock mitral valve. Apical tumours can also manifest as obliteration of the ventricular apex[6].

Finally, hypereosinophilic syndrome represents a wide spectrum of disorders that ultimately result in eosinophil-mediated organ damage and is most often associated with hematologic neoplasms, as well as secondary to specific syndromes such as parasitic infections and certain vasculitis, which might have been the case in our patient[7]. Although angina is not a typical presentation of EMF, the findings on MRI led us to primarily consider a diagnosis of microvascular angina secondary to endomyocardial fibrosis. We highlight a limitation in clarifying the aetiology of angina in this patient, as we were unable to obtain a microvascular haemodynamic study from our laboratory. However, as we argue based on the MRI findings, the diagnosis is highly suggestive. The extensive fibrosis with ventricular calcification and its hypoperfusion supports the diagnosis.

Recent studies are beginning to propose individualizing the management of angina in this patient according to the pathophysiological substrate. Consequently, the therapeutic approach was tailored to manage angina as endothelial dysfunction phenotype due to similarities in terms of anatomical substrate with the presence of fibrosis, as well as the predominant role of inflammation in this disease. Based on this, we initiated carvedilol 6.25 mg twice daily and enalapril 10 mg once daily[8].

Management of cardiovascular disease risk factors and conventional cardiac medications are used for coronary microvascular dysfunction (CMD), although they do not yield satisfactory results in all case[8]. Classic anti-anginal drugs are effective in 50% of CMD patients. At a follow-up consultation after 10 months, the patient did not report further episodes of angina.

DISCUSSION

The natural history of EMF is not completely understood because patients often seek medical attention as they remain asymptomatic for long periods. Once the acute phase of the disease has passed, the most common presentation is heart failure. The patient did not recall any relevant illnesses during childhood or adolescence, suggesting that the acute phase may have been asymptomatic or paucisymptomatic. Her previous medical history, including hypertension, diabetes, and dyslipidaemia, was not associated with EMF.

In our patient, the diagnosis was made in the context of an investigation of chest pain. Chest pain has not been documented in the literature as one of the frequent symptoms associated with this condition.

The differential diagnosis with other entities, primarily HCM, can be challenging. In our case the size of the LV was normal, the atria were only slightly dilated, and the filling physiology was not restrictive. Therefore, tissue characterization by MRI and the absence of findings typical of other conditions relevant for differential diagnosis were crucial in reaching the diagnosis. The patient's country of origin and the laboratory finding of eosinophilia also supported the diagnosis.

In these cases, CMD is probably the main causative factor of chest pain. The pathophysiological background of this condition includes impaired microvascular vasodilatation, which leads to an inability to meet myocardial oxygen demands[8]. Inflammation reduces nitric oxide production resulting in abnormalities of CMD and myocardial ischemia by increasing collagen deposition, loss of micro vessels and development of fibrosis/hypertrophy[8]. To date, there are no publications that consider the initial presentation as microvascular angina in these patients.

Medical treatment for microvascular angina was initiated, without starting corticosteroids due to the absence of acute inflammation on MRI.

CONCLUSION

EMF is a rare condition in European countries, and its clinical presentation as exertional angina is not well defined in the literature. This may be due to microvascular ischaemia; therefore, the level of evidence regarding the symptomatic management of these patients is very low.

We present the diagnosis of microvascular angina secondary of EMF. Management with antianginal agents and strict control of cardiovascular risk factors is a feasible option for patients in the fibrotic phase of the disease without inflammation on MRI.

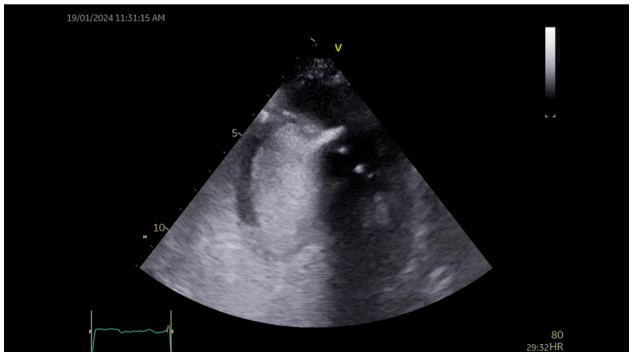
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APPENDIX



Video S1. Twelve-lead electrocardiogram.
https://youtu.be/eXy17gil9Gc



Video S2. Coronary angiography negative for coronary lesions.
https://youtu.be/8wiBG3wKJ3I



Video S3. Cardiac magnetic resonance imaging T2-weighted sequence.
https://youtu.be/iO6i5dN837I

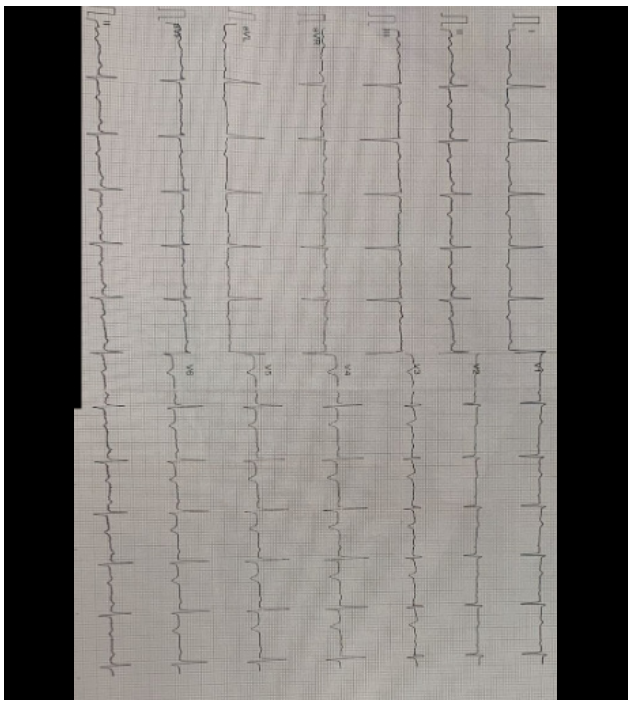


Figure S1. Twelve-lead electrocardiogram.