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Case Report

Infiltrative Cardiomyopathy Caused by Massive Fatty Myocardial Expansion

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Abstract:

Infiltrative cardiomyopathies are uncommon myocardial disorders characterized by abnormal interstitial deposition of biological material, leading to impaired myocardial structure and function. We report a previously undescribed form of infiltrative cardiomyopathy caused by diffuse myocardial adipose tissue expansion. A 75-year-old man was admitted with progressive heart failure, severe left ventricular hypertrophy, reduced systolic function, pleural and pericardial effusion, and systemic edema. Electrocardiography demonstrated atrial fibrillation, low QRS voltages, and bifascicular block. Cardiovascular magnetic resonance showed severe asymmetric left ventricular hypertrophy, diffuse myocardial signal abnormalities, increased native T1 and extracellular volume values, and global T2 elevation, initially suggesting cardiac amyloidosis or hypertrophic cardiomyopathy with extensive fibrosis. However, bone scintigraphy and immunoelectrophoresis excluded amyloidosis. Endomyocardial biopsy revealed massive diffuse adipocyte infiltration involving the myocardium, intramural vessels, and conduction tissue, associated with cardiomyocyte atrophy and cellular disconnection, without evidence of inflammation, fibrosis, or intracellular lipid accumulation. Ultrastructural examination confirmed preserved cardiomyocyte architecture with compression-induced atrophy. Immunohistochemistry demonstrated Cyclin D1 positivity in adipocytes, suggesting active adipocyte proliferation, while Ki-67 expression was largely absent. This previously unreported pathological entity expands the spectrum of infiltrative cardiomyopathies and suggests that diffuse myocardial fatty infiltration may represent a distinct cause of heart failure, electrical instability, and conduction abnormalities.

Key words: infiltrative cardiomyopathy; myocardial fatty infiltration; endomyocardial biopsy; cardiac magnetic resonance; conduction tissue disease; heart failure; low qrs voltage

Introduction

Infiltrative heart muscle diseases are rare entities characterized by deposition in the interstitium of the myocardium of abnormal biologic material altering heart nutrition and activity with compromise of diastolic and systolic function. Major exemplifications are represented by cardiac amyloidosis 1 and sarcoidosis 2. Following herein we describe a previously unreported case of a massive fat infiltration of the myocardium involving cardiomyocytes, conduction tissue (CT) and intramural vessels causing heart failure, Low QRS voltages with abnormal conduction and electrical instability at ECG.

Case Presentation

A 75-year-old male was admitted with heart failure, a severely hypertrophied heart, and a reduced systolic function with pleural and pericardial effusion and systemic edema. His body weight was normal (MBI 20 Kg/m2) and normal was his blood pressure (120/80 mmHg). The ECG (Figure 1) was characterized by atrial fibrillation, low QRS voltages and a bi-fascicular block (Right bundle branch block with + left anterior hemiblock). His blood chemistry screening revealed normal levels of blood sugar and hypercholesterolemia (280 mg/dl). His clinical history was uneventful. Cardiovascular Magnetic Resonance (CMR) imaging (Figure 2) was performed on a 3T scanner (Vida, Siemens Healthcare) to assess myocardial tissue characteristics and to aid in the differential diagnosis of hypertrophic phenotype, particularly to distinguish HCM

from secondary phenocopies due to storage or infiltrative diseases (e.g., Fabry disease or cardiac amyloidosis).

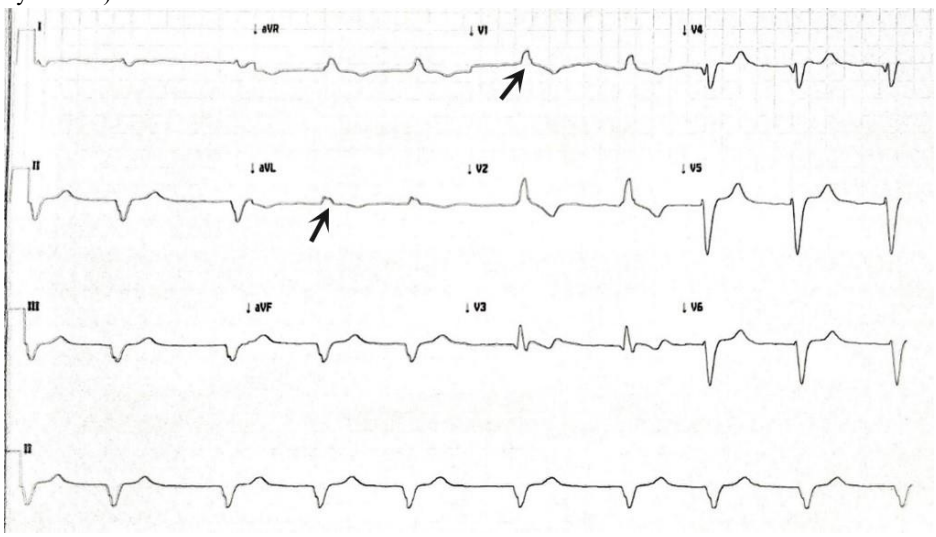

Figure 1: ECG showing atrial fibrillation, Low QRS voltages and bi-fascicular block (right bundle block (arrow in V1) with LA hemiblock (arrow in aVL).

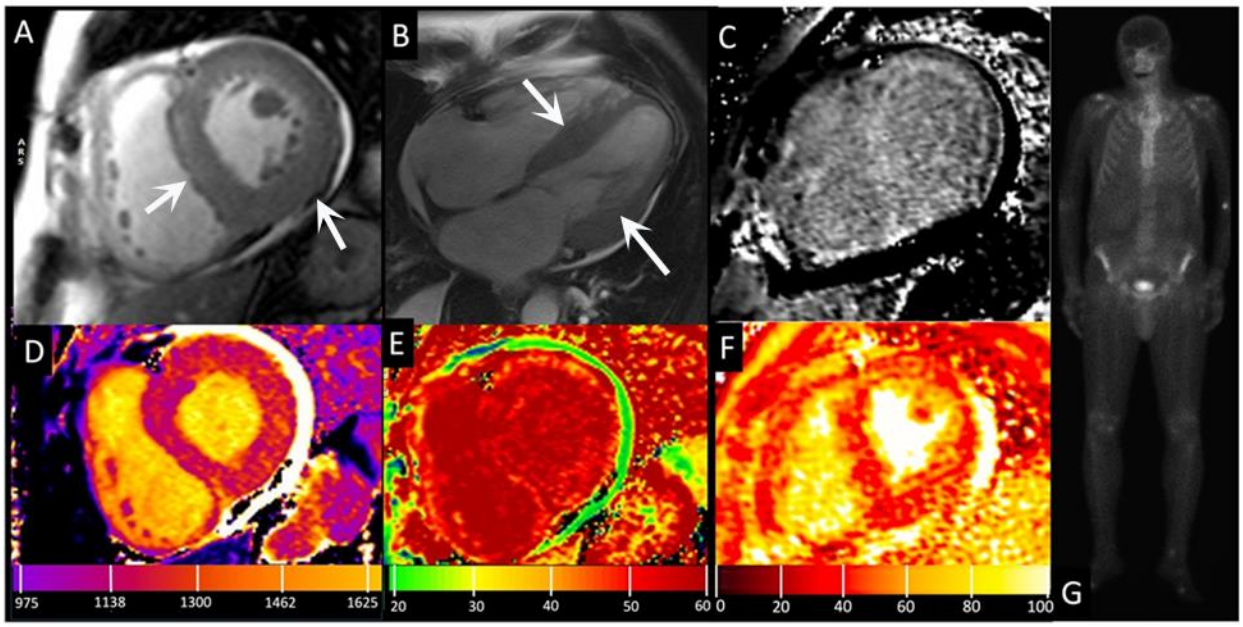

Figure 2: Cine CMR images acquired in short-axis (a) and four-chamber (b) views demonstrate severe left ventricular hypertrophy, marked biatrial enlargement, and a small pericardial effusion. (c) Late gadolinium enhanced image shows diffusely increased myocardial signal intensity, as typically observed in cases of diffuse fibrosis or cardiac amyloidosis. Native T1 (d) and extracellular volume (ECV) (e) maps show diffusely elevated myocardial values, raising suspicion for myocardial amyloidosis. (f) Myocardial T2 map demonstrates globally increased values, consistent with diffuse myocardial edema. Bone scintigraphy (g) did not reveal any myocardial tracer uptake, corresponding to a Perugini score of 0 and indicating a very low probability of transthyretin (ATTR) cardiac amyloidosis.

Methods

Cine CMR images (Figure 2) revealed severe asymmetric left ventricular hypertrophy, predominantly involving the mid-to-basal segments (MWT 18 mm at the interventricular septum) with apical sparing. A restrictive filling pattern was suggested by marked bi-atrial enlargement, along with mild mitral and tricuspid regurgitation, and a small pericardial effusion. Left ventricular systolic function was reduced, with an ejection fraction of 35%. Tissue characterization sequences showed diffusely elevated native myocardial T1 values and a markedly increased extracellular volume (ECV), consistent with extensive interstitial expansion. Global myocardial T2 values were also increased (T2 global: 48 ms; normal <43 ms), suggesting diffuse myocardial edema. Late gadolinium enhancement (LGE) imaging demonstrated a globally increased signal intensity throughout the myocardium, without clear regional variation. The first diagnostic indication was for cardiac amyloidosis or a sarcomeric HCM with extensive myocardial fibrosis 3,4,5 Total body scintigraphy with TC99 was negative for ATTR deposition and no monoclonal

gammopathy was shown at immune-electrophoresis ruling out AL amyloid. To clarify the myocardial substrate of this entity, the patient underwent cardiac catheterization with coronary and left ventricular angiography and LV endomyocardial biopsy with withdraw of 5 samples from different sites of IV septum and LV apex. At catheterization, LV end-diastolic pressure was elevated (25 Hg mm), LV walls were remarkably thickened and LVEF was reduced to 35%. Coronary arteries presented no obstructions.

Results

At histology cardiomyocytes (Figure 3, panel A-B-D-F) appeared in all samples normally or reduced in size (diameter in the transverse section at nuclear level around ≤15 μ) and regularly arranged with no inflammation or fibrosis and devoid of lipid drops or accumulation of storage material at ultrastructural examination (Figure 3, panels A-B-D-F). Myocytes were often thinned and disconnected because of adipocytes grow, infiltration and compression (see Figure 3 panel B) losing their electrical and mechanical integration. Intramural vessels were infiltrated as well causing cell disconnection of adventitial and muscular layers (Figure 3, panel C). In the biopsy fragments were included some sections of Purkinje fibers that appeared diffusely and remarkably infiltrated by adipocytes (Figure 3 panel D).

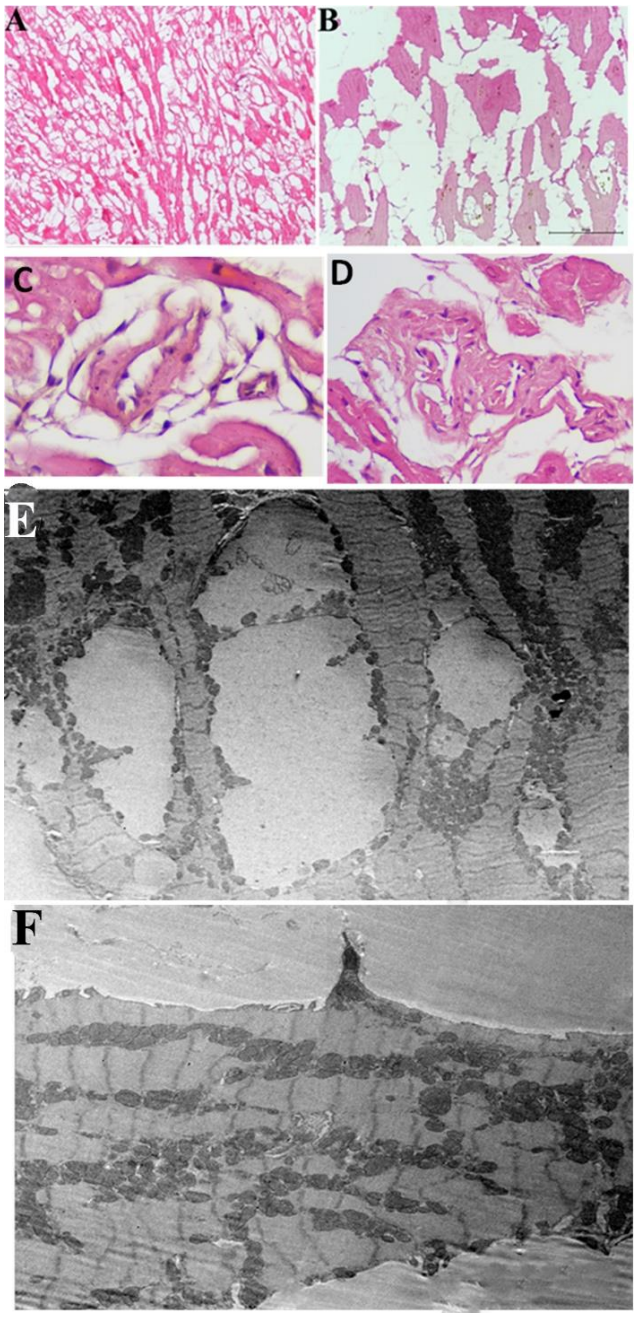

Figure 3:

Panel A: massive adipocytes infiltration of the myocardium with adjacent myocytes compression and hypotrophy. H&E, 200x.

Panel B: Cardiomyocytes disconnection by fat infiltration. H&E 400x.

Panel C: Fat infiltration of an intramural arteriole with disconnection of smooth muscle cells.

H&E 400x.

Panel D: Fat infiltration of a section of conduction tissue (i.e Purkinje fibres). H&E 400x.

Panel E: Electron-microscopy showing adipocytes compression of hypotrophic myocytes.

Panel F: Electron-microscopy of a single cardiomyocyte showing normal cell structure ruling out a primary cardiomyocyte disorder.

Cardiomyocyte atrophy and disconnection appear as the key of interpretation of both heart failure and atrial fibrillation associated to bi-fascicular block. Low QRS voltages at ECG define the severity of fatty infiltration of the myocardium similarly to that observed for cardiac amyloidosis.

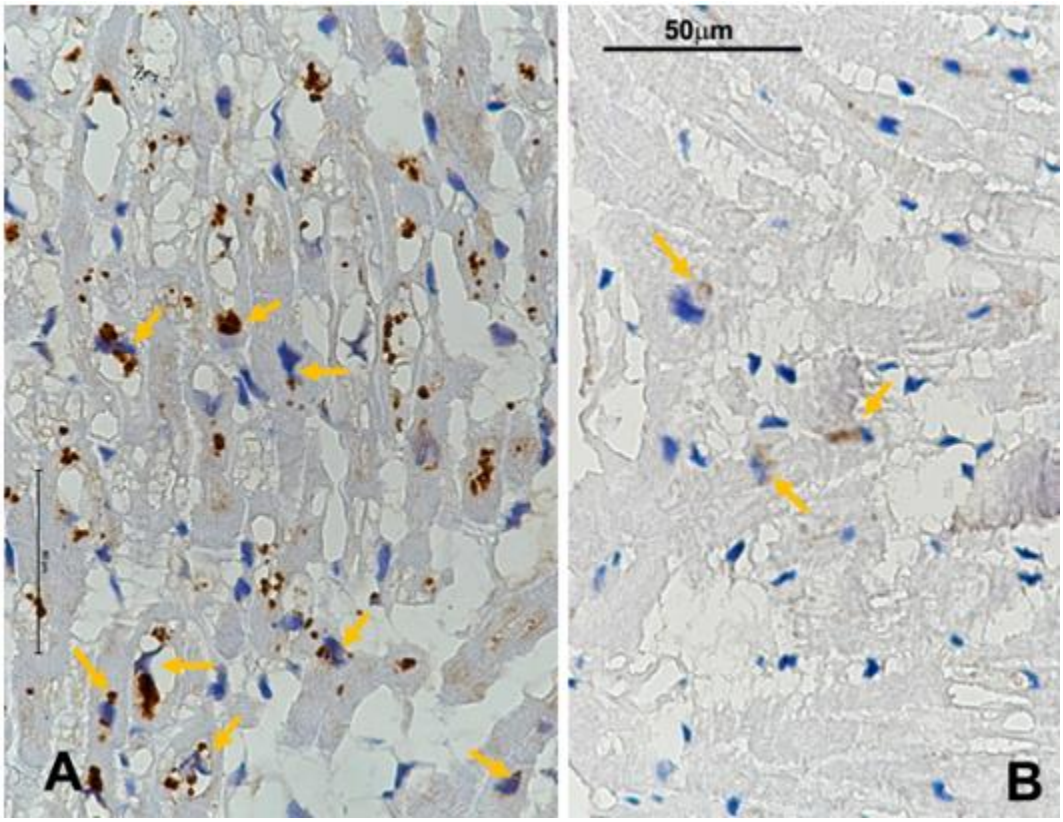

Figure 4: Immumohisto chemistry (IHC) for Cyclin D1 (A) and Ki-67 (B).

Cyclin D1 appears abundantly positive in EMB sample from patient, prevalently localized in adipocytes. Most of times the peroxidase product (brawn) is strictly associated with blue nuclei (arrows). On the contrary Ki-67 is negative in almost the cells. Very few cells (arrows) are positive. The bars represent 50 microns.

Discussion

The study reports a severe LV wall thickening sustained at histology and electron microscopy by diffuse proliferation and massive myocardial infiltration by adipocytes. Normal cardiomyocytes appeared to undergo a process of atrophy and disconnection as a consequence of adjacent adipocytes' s grow and compression. Even the conduction tissue (CT) was found similarly involved suggesting heart failure and electrical abnormalities to be a shared consequence. Indeed, cell disconnection cause a loss of integration of mechanical activity and can well be a source of LV dysfunction. Low QRS voltages at ECG define the severity of fatty infiltration of the myocardium similarly to that observed for cardiac amyloidosis. Regarding the reasons of fat infiltration, adipocyte proliferation doesn't seem to have a reparative meaning as myocytes do not show structural abnormalities and cell damage, myocardial inflammation and fibrosis were not a finding.

Interestingly, native T1 values were not reduced, as might be expected in lipid storage disorders such as Fabry disease. This paradoxical finding is likely attributable to concomitant myocardial edema, which tends to prolong T1 relaxation times and may obscure the typical T1 shortening associated with intracellular lipid accumulation or fatty infiltration. Similarly, the diffuse high signal observed on LGE images does not appear to be related to contrast agent retention alone, as observed in case of diffuse fibrosis or amyloid deposition, but is more likely due to extensive fatty infiltration of the myocardium.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the locally appointed ethics committee (opinion number 6/2019 and 2016-003014-28 (FARM12JCXN) and informed consent was obtained from all subjects.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The datasets used and analyzed during the current study are avail-able from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare that they have no competing interests.

Conclusion

The pathway of infiltrative cardiomyopathy should include a previously unreported entity represented my massive fat expansion of the myocardium.

References

1. Falk RH, Alexander KM, Liao R, Dorbala S. (2016). AL (Light-Chain) cardiac amyloidosis: a review of diagnosis and therapy. *J Am Coll Cardiol*;68(12):1323–1341.
2. Birnie DH, Nery PB, Ha AC, Beanlands RSB. (2016). Cardiac sarcoidosis. *J Am Coll Cardiol*.;68(4):411–421.
3. Frustaci A, Loperfido F, Pennestrì F. (1985). Hypertrophic cardiomyopathy simulating an infiltrative myocardial disease. *Br Heart J*;54(3):329–332.
4. Frustaci A, Russo MA, Chimenti C. (2013). Diagnostic contribution of left ventricular endomyocardial biopsy in patients with clinical phenotype of hypertrophic cardiomyopathy. *Hum Pathol*.;44(1):133–141.
5. Frustaci A, Verardo R, Caldarulo M, Acconcia MC, Russo MA, Chimenti C. (2007). Myocarditis in hypertrophic cardiomyopathy patients presenting acute clinical deterioration. *Eur Heart J*;28(6):733–740.

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