IMAGE IN CARDIOLOGY

Multimodality imaging in discovering the etiology of negative T waves

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A 50-year old woman, with cardiovascular risk factors (CVRF) (smoking, hypertension), is referred to our cardiology department for atypical chest pain (rare episodes of persistent chest discomfort, not related to exercise) and for recently detected electrocardiographical (ECG) changes (diffuse negative T waves in V1-V6, DII, DIII) (Panel A). Of note, a resting ECG from 2015 showed a similar aspect.

After obtaining an unremarkable clinical examination and a similar ECG tracing, the 2D echocardiography revealed a normal sized left ventricle (LV) with preserved ejection fraction (LVEF) and normal segmentary systolic function. While examining the right ventricle (RV), the subcostal view suggested a hypertrophied RV free wall (measured as 10 mm, with apparent homogeneous echogenicity) (Panel B), with normal longitudinal function and no segmentary contraction abnormalities.

Cardiac magnetic resonance (CMR) was done with a referral suspicion of arrhythmogenic cardiomyopathy (AC). It found normal cardiac cavities, normal wall thickness of both ventricles (RV free wall of 3 mm), no late gadolinium enhancement, but described a large amount of fat with concentric disposition around the heart, maximum 9 mm anterior of RV and 3 mm posterior of LV (Panel C).

While this was not fully and safely explaining the ECG changes, and the patient presented with a low to intermediate pretest probability for coronary artery disease (CAD), an angio-computed tomography (CT) coronary scan was performed, with a calcium score of 7 AU, no significant coronary atherosclerosis and a hypoplastic circumflex artery (Panel D). No changes in pericardial thickness or structure were described, but a total volume of 149 ml of pericardial fat was measured (Panel E).

The evaluation of chest pain was completed by stress echocardiography: at maximal workload (145 bpm, 85% predicted - 100 W), contractile reserve was present, with no segmentary contractility changes; no chest pain was noted, and at maximal exercise there was a pseudo-normalization of T waves.

To synthesize, multimodality imaging helped us following a differential diagnosis which included: AC (negative T waves, RV free wall apparent changes on echocardiography, but no family history of sudden cardiac death), ischemic heart disease (negative T waves, atypical angina, cardiovascular risk factors), and pericarditis (but negative biological inflammatory markers, no pericardial fluid or pericardial friction rub were noted). As a result of this workup, the final hypothesis is that the ECG changes are related to the abnormal pericardial adipose tissue (PAT) deposit, which acts similar to a process of chronic pericarditis.

The present case report is, to the best of our knowledge, the first to describe an association between large pericardial fat and ECG changes, when other causes were excluded. Of course, even if the current

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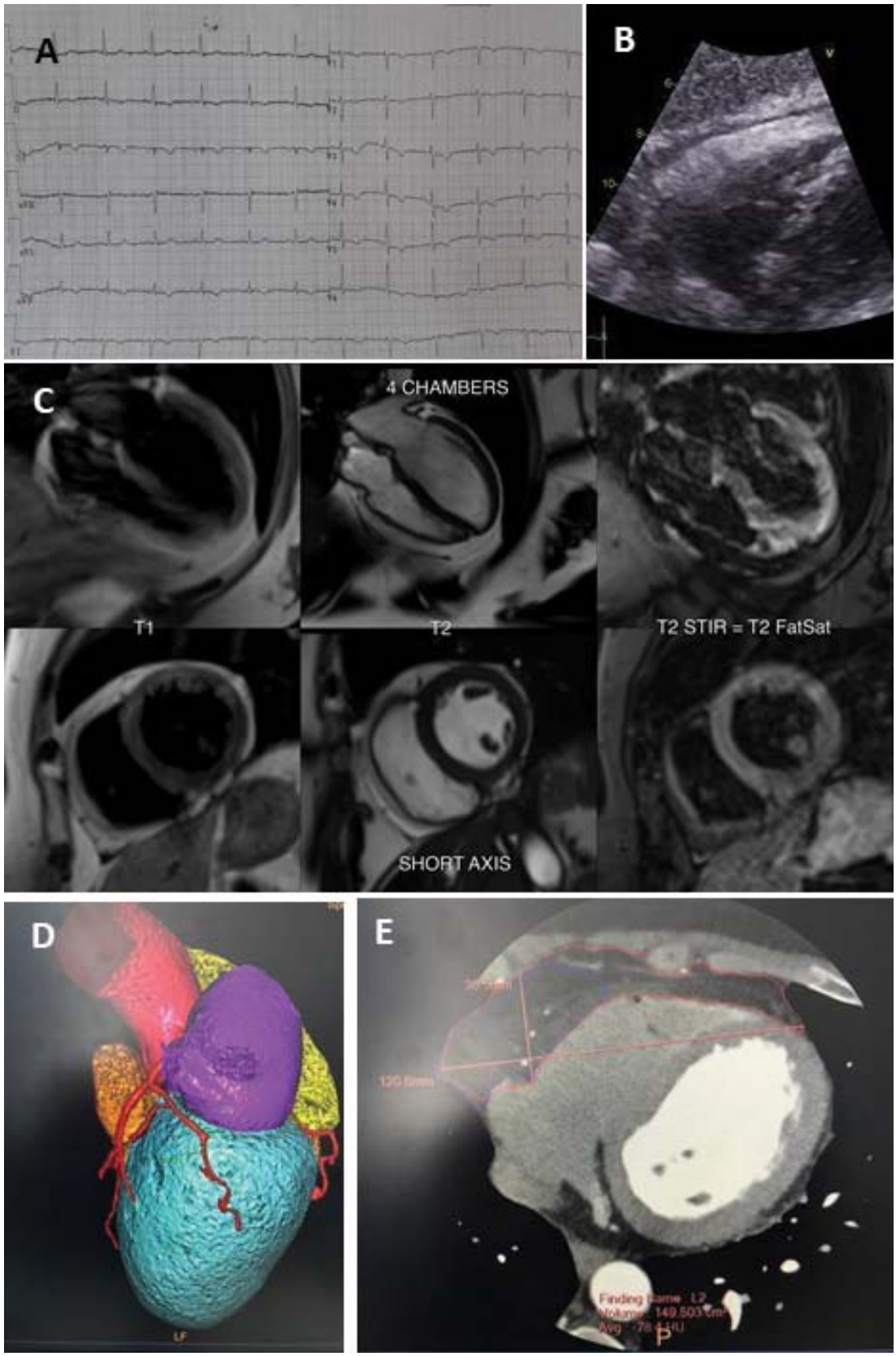

Figure 1. A. Electrocardiogram with inverted T waves in DII, DIII, V1-V6. **B.** Transthoracic echocardiography – subcostal view with impression of right ventricular hypertrophy. **C.** CMR image - four chamber view and short axis view - presence of pericardial fat tissue with high signal intensity on T1, T2-weight cine images and low signal intensity on T2 FatSat sequences. **D.** 3D reconstruction of coronary angio-CT with evidence of a hypoplastic circumflex artery. **E.** CT image with evaluation of pericardial fat volume.

findings are benign, the patient should be followed up closely and risk factors should be thoroughly controlled, as several studies have shown that pericardial fat is associated with poorer cardiovascular prognosis.

Increasingly becoming an important CVRF, PAT is represented by the quantity of fat localized between the epicardium and the outer layer of the pericardial sack 1. It exerts local pathophysiologic effects on both the myocardium and epicardic coronary arteries by: diastolic dysfunction, endothelial dysfunction, fibrosis and inflammation and is associated with coronary calcifications, diabetes, older age, dyslipidemia and arterial hypertension[1-3]. PAT increases total cardiovascular risk by 22% and predisposes to incidental heart failure, stroke and all-cause mortality[4,1]. It also predicts CAD, especially calcified and non-calcified atheroma with high-risk morphology for future adverse events[5].

Excess fat in the pericardial sack is strongly related to atrial fibrillation (Afib), most notably with its chronicity, appearance after coronary artery by-pass graft surgery and recurrence following ablation procedures[2]. Moreover, regarding the RV, excess adipose tissue around it will lead to a lowering in ventricular mass and end-systolic, end-diastolic, stroke volumes[4].

Treatment options range from weight management to pericardiotomy[2]. BELLES trial demonstrated that a high intensity statin regimen reduces PAT by 3% and the highest benefit is seen in post-cardiac surgery Afib[2].

In conclusion, following a multimodality imaging approach in a patient with clinical and electrical signs suggestive of ischemia, we established a diagnosis of excess PAT as responsible for the ECG and imaging findings. Such patients should be closely followed, as they appear to be at high risk of future adverse cardiac events.

Keywords: Multimodality imaging; pericardial adipose tissue.

Conflict of interest: none declared.

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