1100

When multimodality imaging is needed to understand the etiology of diffuse negative t waves

Giuca A.[1]; Bica R.[2]; Rosca M.[1]; Stanciu S.[3]; Popescu BA.[1]; Jurcut R.[1]

[1]Institute of Cardiovascular Diseases Prof. C.C. Iliescu, Bucharest, Romania
[2]Metropolitan Hospital, Bucharest, Romania
[3]Carol Davila Emergency Clinical Military Hospital, Bucharest, Romania

Introduction: We present the case of a 50 year old woman, active smoker, hypercholesterolemic, nondiabetic, with normal body mass index, referred to our cardiology department for atypical chest pain and ECG changes (diffuse negative T waves in V1-V6, DII, DIII) (Panel A), also present on a resting ECG recorded 4 years previously.

Purpose: To highlight the fact that even though the most frequent cause of negative T waves remains coronary artery disease (CAD), the cause could be related to other more rare medical conditions.

Methods: After clinical examination and ECG, 2D echocardiography was performed. It revealed a normal sized left ventricle (LV) with perserved global and regional function, normal global longitudinal strain, nondilated atria and no significant valvular disease. The subcostal view suggested a hypertrophied right ventricle (RV) free wall (10 mm thickness, with apparent homogeneous echogenicity) (Panel B, arrow), with normal longitudinal function and no regional wall motion abnormalities.

Cardiac magnetic resonance (CMR) was performed for a better tissue characterization. The CMR examination 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, maximum thickness of 9 mm anterior of RV, 3 mm posterior of LV (Panel C).

Since this was not fully explaining the ECG changes and the patient presented with a low-intermediate pretest probability for CAD, an angio-CT coronary scan was performed and showed a calcium score of 7 AU, no significant coronary atherosclerosis, and a hypoplastic circumflex artery (Panel D, arrow). A total volume of 149 ml. of pericardial fat was measured (Panel E).

Stress echocardiography was performed with maximal workload achieved (145 bpm, 85% predicted, 100 W); no chest pain was reported, no regional wall motion abnormalities were seen and there was normal contractile reserve.

Results: Using multimodality imaging a differential diagnosis was followed, which included: arrhythmogenic cardiomyopathy (negative T waves, apparent changes of RV free wall on echo, but no family history of sudden cardiac death), CAD (atypical angina, cardiovascular risk factors, negative T waves), and pericarditis (but negative inflammatory markers, no pericardial fluid). The final hypothesis is that the ECG changes are related to the abnormal pericardial adipose tissue deposit, which acts similar to pericarditis.

Conclusions: The present case report is, to our knowledge, the first to describe an association between large pericardial fat deposit and ECG changes, when other causes were excluded. Even if the current findings are benign, the patient should be followed closely, and risk factors should be thoroughly controlled, as several studies have shown that pericardial fat is associated with poorer cardiovascular prognosis

Abstract 1100 Figure. Multimodality imaging in our patient