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Which one is Worse? Acute Myocarditis and Co-existing Non-compaction Cardiomyopathy in the Same Patient

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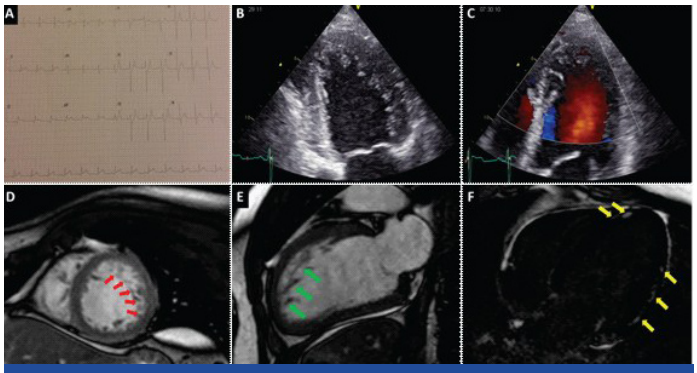
Non-compaction cardiomyopathy is a relatively rare cardiac condition known to be found in 0.12 per 100,000 cases characterized by increased trabeculations in the ventricular wall due to embryologic malformation predisposing malignant ventricular arrhythmias [1,2]. Although acute postviral myocarditis has been well-documented in the medical literature, co-existence of these two clinical entities is extremely uncommon [3-5].

A 21-year-old male without any previous cardiac history presented with retrosternal chest pain and dyspnea lasting for three days. He was having a flu-like syndrome during the last week with symptoms of sore throat and myalgia. ECG showed diffuse ST elevations without any reciprocal changes [Table/Fig-1a]. Laboratory analysis showed marked increase in white blood cells with lymphocyte dominance. Both C-reactive protein, Troponin-I and N-terminal pro BNP levels were significantly increased. Transthoracic echocardiography (TTE) showed hypokinesis of the apical and postero-lateral walls with an ejection fraction of 42%. Prominent apical trabeculations were noted along with marked spontaneous echo-contrast in the left ventricular cavity [Table/Fig-1b] [Video 1]. Colour Doppler analysis demonstrated the entrance of blood flow between these trabeculations [Table/Fig-1c] [Video 2]. Although clinical history was compatible with acute myocarditis with a fulminant course, non-compaction cardiomyopathy was also suspected as a differential diagnosis. Cardiac magnetic resonance imaging (MRI) with contrast study was performed in order to further explore the pathology. Diffuse hypokinesis of the dilated ventricle along with marked trabeculations especially on the apico-anterior wall strengthened the diagnosis of non-compaction cardiomyopathy [Table/Fig-1d,1e] [Video 3,4]. However, there was also strong evidence for acute myocarditis shown by diffuse edema in T2A sequences as well as late contrast enhancement in the subepicardial layer of the posterolateral and apical walls [Table/Fig-1f]. Myocarditis was treated conservatively. Symptoms and laboratory findings diminished in a few days with ongoing systolic dysfunction. After discharge, the patient was referred for implantable cardioverter defibrillator (ICD) implantation to prevent sudden death for non-compaction cardiomyopathy.

Despite the typical signs and symptoms supporting a diagnosis of myocarditis, clinicians should be alert for investigating additional causes of heart failure using comprehensive imaging modalities such as cardiac MRI. Present case demonstrates the laboratory, ECG and imaging features of such a patient that was initially treated conservatively for acute myocarditis and subsequently referred for ICD implantation for prevention sudden cardiac death.



[Table/Fig-1a]: ECG on presentation showing diffuse ST elevations without any reciprocal changes **[Table/Fig-1b]:** TTE in apical two-chamber view showing marked trabeculations and spontaneous echo-contrast **[Table/Fig-1c]:** TTE with colour Doppler. Note the entrance of blood flow between the trabeculations **[Table/Fig-1d]:** Cardiac MRI, short-axis view demonstrating marked trabeculations (arrows) in the left ventricular cavity **[Table/Fig-1e]:** Cardiac MRI, long axis view represents the dilated left ventricle along with prominent trabeculations (arrows). **[Table/Fig-1f]:** Late contrast enhancement of the subepicardial layer of apical and lateral walls (arrows) compatible with myocarditis

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