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CASE REPORT

Embolic complication of left ventricular non-compaction as an unusual cause of acute myocardial infarction

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KEYWORDS
Left ventricular non-compaction;
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Abstract One of the greatest challenges in medicine consists of arriving at a correct diagnosis despite different presentations of the disease. We present a case in which, notwithstanding the initial diagnosis, the search for the etiology was essential for clinical guidance. Left ventricular non-compaction (LVNC) was first described by Chin et al. in 1990. This relatively new entity is characterized by excessive thickening of the myocardial wall, formed of a thin epicardial layer and a substantially thicker non-compacted endocardial layer. The clinical presentation is highly variable but it must always be borne in mind that heart failure, atrial and ventricular arrhythmias and embolic events are common complications of LVNC.

PALAVRAS-CHAVE
Ventrículo esquerdo não compactado;
Síndroma coronária aguda;
Arritmias;
Insuficiência cardíaca

Embolia com causa incomum de enfarte agudo de miocárdio na não compactação do ventrículo esquerdo

Resumo Um dos maiores desafios da medicina é a realização de um diagnóstico correto, apesar das diferentes possíveis apresentações de cada doença. Em seguida apresentamos um caso que, apesar de um diagnóstico inicial, a busca da etiologia foi fundamental para a orientação clínica. A não compactação do ventrículo esquerdo (LVNC) foi primeiramente descrita por Chin et al, em 1990. Esta patologia relativamente nova é caracterizada pelo espessamento da parede miocárdica, constituída por uma camada fina epicárdica e uma camada substancialmente mais espessa de miocárdio não-compactado na região endocárdica. A apresentação clínica pode ser altamente variável, mas é preciso sempre ter em mente que a insuficiência cardíaca, arritmias atriais e ventriculares e eventos embólicos são as complicações mais comuns de LVNC.

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Case report

We present the case of a 65-year-old woman with known cardiovascular risk factors of hypertension, obesity and dyslipidemia, with no other relevant medical history. She had excellent functional capacity and reported only occasional episodes of palpitations, short and self-limited.

In the course of her normal daily activities she presented an episode of severe anterior chest pain after exertion, accompanied by breathlessness, sudoresis, and nausea. She was taken to our emergency room, where the admission ECG showed sinus rhythm, intraventricular conduction disturbance – incomplete left bundle branch block – and ST-segment elevation in V1–V3 (Figure 1). In the emergency room her clinical condition started to deteriorate with marked hypotension and alteration of consciousness. The physical examination revealed no relevant alterations.

She was immediately taken to our catheterization laboratory, where coronary angiography showed occlusion of the distal left descending coronary artery, with no other lesions in the coronary tree. Thrombus aspiration was performed, with immediate establishment of TIMI 3 flow, and no stenosis was observed. The thrombus appeared highly organized (Figure 2), complementary in shape to the coronary artery. No atherosclerotic lesions were observed despite the patient's age and cardiovascular risk factors, which was highly suggestive of myocardial infarction without obstructive coronary disease. Further investigation was indicated to clarify the etiology.

She was admitted to our coronary care unit after the procedure and evolved favorably in Killip class I; peak troponin I was 38 ng/ml. Hemodynamically she gradually improved, responding well and tolerating therapy with low-dose beta-blockers. Telemetric ECG monitoring documented short periods of atrial fibrillation and persistence of left bundle branch block. There were no more episodes of chest pain.

The transthoracic echocardiogram revealed a normal-sized left ventricle with moderate systolic impairment and hypertrabeculation of the left ventricular apex and hypokinesia of the anteroseptal wall and apical region (Figure 3). Doppler color flow mapping confirmed flow between the trabeculations. Cardiac magnetic resonance imaging (MRI) confirmed left ventricular hypertrabeculation, mainly in the apical and mid segments, with moderate systolic dysfunction. Transmural apical late enhancement was also evident, compatible with myocardial infarction (Figure 4).

These findings were suggestive of left ventricular non-compaction; this case presents the three major manifestations of the disease that ultimately resulted in coronary artery occlusion, precipitating an acute coronary syndrome.

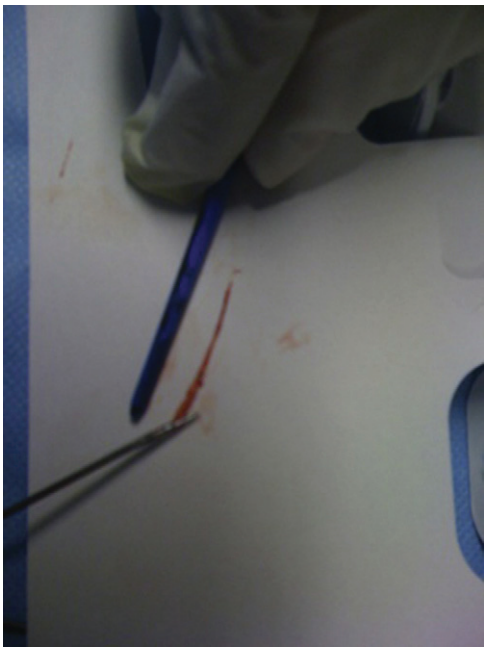

Figure 2 Intracoronary thrombus.

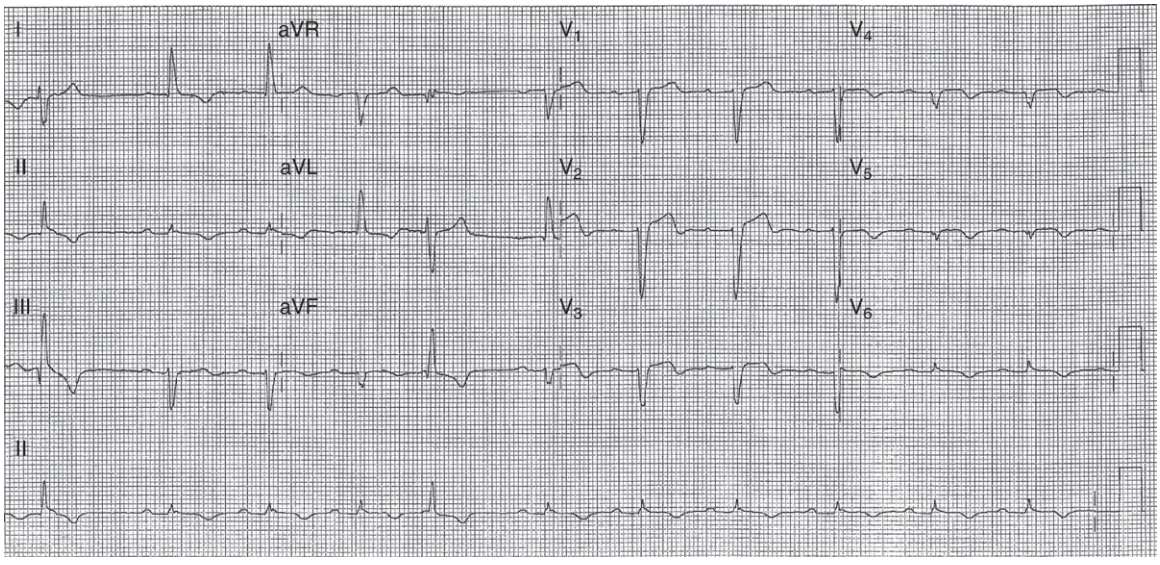

Figure 1 Admission EKG showing sinus rhythm, intraventricular conduction abnormality – incomplete left bundle branch block pattern – and ST segment elevation in V1–V3.