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CASE REPORT

Noncompaction and embolic myocardial infarction: The importance of oral anticoagulation

Giovanni Pulignano[a,*], Maria Denitza Tinti[a], Stefano Tolone[a], Carmine Musto[b], Lucia De Lio[a], Paolo Giuseppe Pino[a], Giovanni Minardi[a], Roberto Violini[b], Massimo Uguccioni[a]

[a] *Cardiology 1/CCU, S. Camillo Hospital, Rome, Italy*
[b] *Interventional Cardiology, S. Camillo Hospital, Rome, Italy*

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Abstract Left ventricular noncompaction (LVNC) is characterized by left ventricular (LV) hypertrabeculations and is associated with heart failure, arrhythmias and embolism. We report the case of a 67-year-old LVNC patient, under oral anticoagulation (OAC) therapy for apical thrombosis. After she discontinued OAC, the thrombus involved almost the whole of the left ventricle; in a few months her condition worsened, requiring hospitalization, and despite heparin infusion she experienced myocardial infarction (MI), caused by embolic occlusion of the left anterior descending artery. Although infrequent as a complication of LVNC, and usually attributable to microvascular dysfunction, in this case MI seems due to coronary thromboembolism from dislodged thrombotic material in the left ventricle.

PALAVRAS-CHAVE
Não compactação ventricular esquerda;
Embolia;
Insuficiência cardíaca;
Enfarte do miocárdio

Não-compactação e enfarte do miocárdio embólico: a importância da anticoagulação oral

Resumo A não compactação ventricular esquerda (NCVE) é caracterizada por hipertrabeculações ventriculares esquerdas (VE) e está associada à insuficiência cardíaca, arritmias e embolias. Divulgamos o caso de uma doente de 67 anos com NCVE e em terapêutica de anticoagulação oral (ACO) por trombose apical. Como descontinuou a anticoagulação oral o trombo envolveu quase todo o VE; em poucos meses a sua situação piorou necessitando

* Corresponding author.
E-mail address: gipulig@yahoo.it (G. Pulignano).

internamento e – apesar da infusão com heparina – sofreu um enfarte do miocárdio (EM), causado por oclusão embólica da DAE. Embora seja pouco frequente tal como a complicação por NCVE e seja geralmente atribuível à disfunção microvascular, o EM parece ser, neste caso, devido ao tromboembolismo coronário a partir do trombo do VE.

Case report

Isolated left ventricular noncompaction (LVNC) is a form of cardiomyopathy resulting from persistence of fetal trabeculations and intertrabecular recesses within ventricular myocardium. The clinical features associated with LVNC consist of left ventricular (LV) systolic dysfunction, arrhythmias, and thromboembolic events. We report the case of a 67-year-old woman admitted to the emergency department with acute aphasia, dyspnea, and peripheral edema associated with recent-onset paroxysmal atrial fibrillation (AF) with high ventricular rate. After anticoagulation with unfractionated heparin (UFH), she was converted to stable sinus rhythm with intravenous (IV) amiodarone. She was then admitted to the intensive coronary care unit (ICCU) and treated with IV inotropes and diuretics, resulting in prompt recovery from aphasia and improvement in congestion. The echocardiogram showed a markedly dilated left ventricle with hypertrabeculation of the apex and of the inferior-inferolateral segments (noncompacted/compacted ratio 2:1), severely reduced ejection fraction (EF) (22%), and an apical thrombus. No significant carotid artery disease was found on Doppler echocardiography. She underwent implantation of an implantable cardioverter-defibrillator and was discharged in NYHA class II, under standard heart failure therapy including oral anticoagulation (OAC), with no evidence of thrombosis (Figure 1). Twelve months later, due to a major depressive episode, the patient failed to attend the scheduled heart failure clinic (HFC) follow-up and discontinued OAC. Due to recurrent dyspnea and fatigue she presented to the HFC, where an echocardiogram showed a massive LV thrombosis (Figure 2), so she was admitted to the ICCU and IV UFH was started. After two days the patient complained of chest pain; as the ECG showed marked ST segment elevation in V3-V6 she was referred to the catheterization lab. Coronary angiography revealed a thrombotic occlusion of the mid segment of the left anterior descending artery (Figure 3A); the clot was aspirated and no significant coronary artery disease (CAD) was found (Figure 3B and C). A marked increase in plasma troponin I was observed, confirming the diagnosis of acute embolic myocardial infarction (MI). Her EF fell to 15% and after two days she became hypotensive despite intra-aortic balloon pump and inotropic support, with cardiogenic shock and acute kidney failure. She was considered for a left ventricular assist device, but sepsis and multiorgan failure occurred, and death followed 25 days later.

Discussion

LVNC is associated with HF, arrhythmias and embolism. Cardioembolic events are not uncommon, as trabecular recesses and depressed systolic function predispose to thrombosis, but presentation as an acute coronary syndrome (ACS) is rather unexpected.[1] Previous observations suggested that in patients with reported myocardial

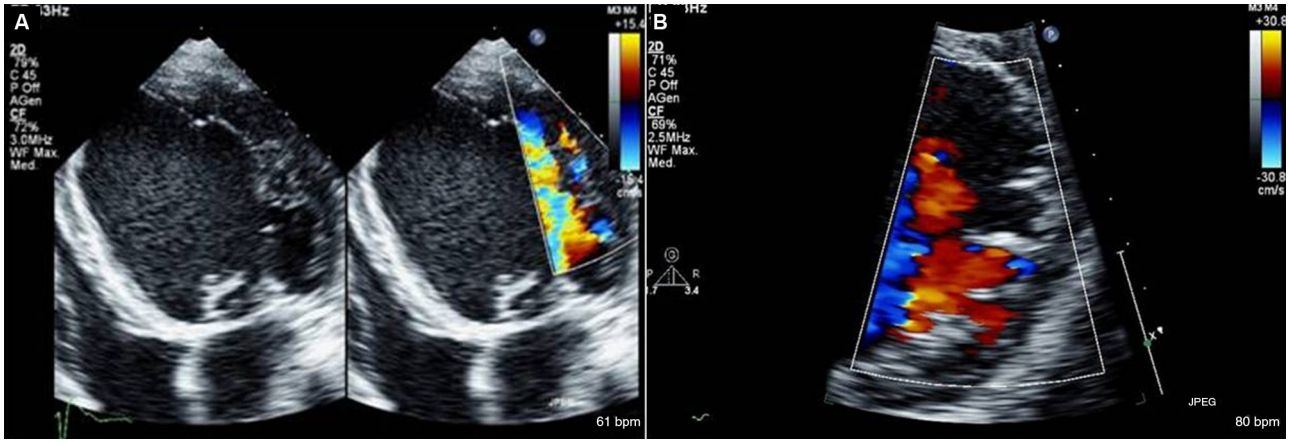

Figure 1 (A) Apical 4-chamber view and (B) parasternal short-axis view of mid segments showing intertrabecular recesses filled with blood in the anterolateral segments.