

Multiple Arterial Thrombosis and Resistance to Conventional Antiaggregation

Trombosis arterial múltiple y resistencia a la antiagregación convencional

To the Editor,

The destabilization of vulnerable atheroma plaques plays a major role in the genesis of acute coronary syndrome (ACS). Apparently, this is not a local reaction of a single atherosclerotic plaque: in the thrombogenic, proinflammatory context of ACS, vulnerability extends to lesions situated at other levels.¹

A 69-year-old woman with a past history of high blood pressure, type 2 diabetes mellitus, overweight (body mass index, 28), and tobacco use was referred for urgent percutaneous intervention, indicated for 2 h of highly intense central chest pain with electrocardiograph changes compatible with extensive infarction and subepicardial lesion current in the inferior wall and leads V3-V6 (Fig. 1). On arrival, the patient was hemodynamically stable and in complete atrioventricular block. She was administered 300 mg acetylsalicylic acid (ASA) and 600 mg clopidogrel when the intervention began. With the onset of chest pain, the patient simultaneously reported pain in the right lower extremity. Emergency echocardiography showed a left ventricle of normal size and general function, without intracavity thrombus, and right ventricular dysfunction. The patient was moved to the cardiac catheterization laboratory where the right coronary artery was catheterized and an acute thrombotic occlusion found in the proximal segment (Fig. 2). She underwent angioplasty and drug-eluting stent implantation after intracoronary administration of abciximab. When the left coronary tree was catheterized, another thrombotic occlusion was found in the left anterior descending artery mid-segment (Fig. 2), where simple angioplasty was performed due to the small diameter of the vessel. When catheterization was being completed, the patient reported right lower extremity pain. On examination, we found coldness in the extremity and an absence of distal pulses. Consequently, a contrast injection was administered via the right femoral artery introducer, revealing complete superficial right femoral branch occlusion

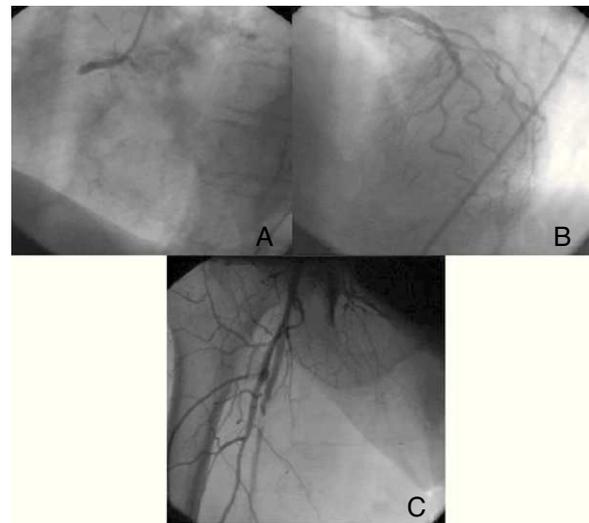


Figure 2. Acute occlusion at the level of the proximal right coronary artery (A), anterior descending artery mid-segment (B), and superficial femoral artery (C).

(Fig. 2). We therefore transferred the patient to the vascular surgery theater for urgent embolectomy with a Fogarty catheter.

No subsequent complications presented. Following clinical practice guidelines, the patient was administered a maintenance regimen of 100 mg ASA and 75 mg clopidogrel. Ten days later, platelet aggregation tests were requested. These revealed the patient's resistance to drugs, as her serum arachidonic acid and adenosine diphosphate concentrations were within the normal range (61% and 50%, respectively; normal, 50%-100%). Given the high thrombotic load of her symptoms, we decided to start treatment with 300 mg ASA and 10 mg prasugrel daily, after a 60 mg load, and repeated the aggregation study at 5 days. This showed arachidonic acid values were wholly suppressed and adenosine diphosphate had fallen to 26%. The patient progressed favorably, without complications, and at 4 months follow-up presented no new cardiovascular events.

In the literature, the series and cases described^{1,2} seem to suggest the most logical strategy when treating ACS with two

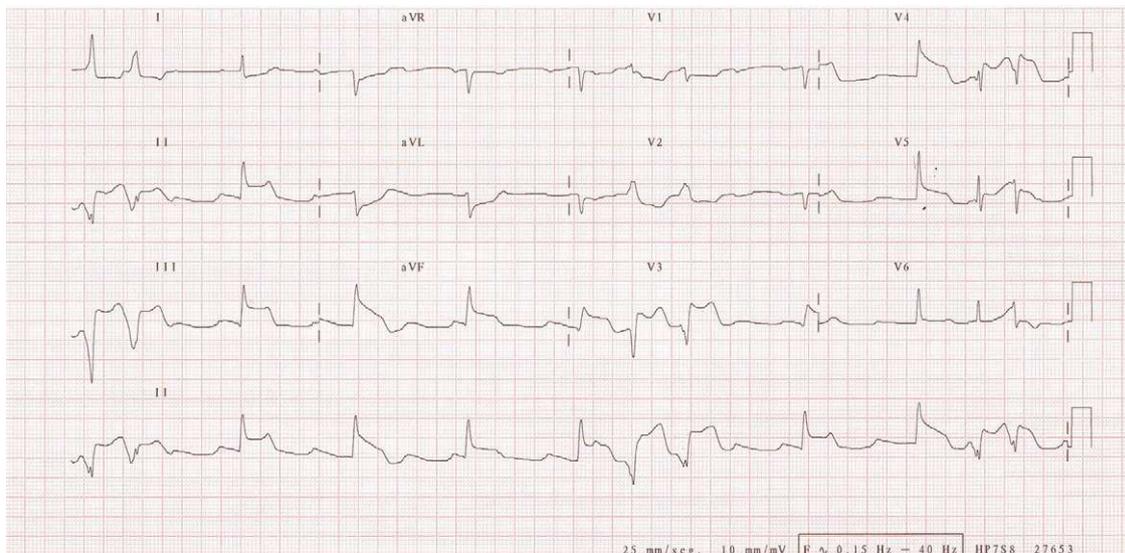


Figure 1. Electrocardiogram with subepicardial lesion in inferior and anterior leads, and complete atrioventricular block.

culprit arteries is complete revascularization in the acute phase. The most frequently described multiple coronary artery occlusions are found in the left anterior descending artery and right coronary artery ($\leq 50\%$ of cases),³ as in the patient described here. The peculiarity of our patient lies in the fact that the acute occlusion extended beyond coronary territory to the superficial femoral artery. Furthermore, we have shown that the patient presented drug resistance to conventional antiaggregation therapy. This resistance may share pathologic mechanisms with greater vulnerability to atherosclerotic plaques. New antiplatelet agents, like prasugrel, may well be more appropriate in these patients, in whom the choice of treatment benefits from a platelet aggregation study.⁴

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Sudden Death Due to Histiocytoid Cardiomyopathy

Muerte súbita debida a miocardiopatía histiocitoide

To the Editor,

Cardiac disease must be systematically evoked in the event of sudden death in the child. Histiocytoid cardiomyopathy (HC) is a rare disease responsible for severe ventricular arrhythmia very early in life. We report 2 cases of HC diagnosed after sudden death in young girls.

CASE 1

M. was the first child of healthy, non-blood relative, Caucasian parents. Hypertrophic cardiomyopathy was diagnosed at third trimester by antenatal ultrasound. At birth, clinical examination revealed a systolic cardiac murmur, an axial hypotonia, linear cutaneous erythematous lesions of the face and the neck typical of the microphthalmia with linear skin defects (MLS) syndrome (Fig. 1). The EKG registered Wolff-Parkinson-White syndrome. Transthoracic echocardiography demonstrated a biventricular hypertrophic cardiomyopathy, a small perimembranous ventricular septal defect. At 7 days of life, she presented an orthodromic tachycardia, successfully treated by amiodarone. Etiologic study of hypertrophic cardiomyopathy was noncontributive. At the age of 3 months, she presented

ventricular fibrillation with no resumption of electric activity despite defibrillations. The autopsy confirmed cardiac hypertrophy with micronodules on the mitral and tricuspid valves. Histological analysis revealed areas formed from bundles of myocardial fibers of normal appearance, contrasting with cells of histiocytoid appearance located in the myocardium, the pericardium, and the valves, leading to the diagnosis of HC.

CASE 2

M. was the first healthy child of non-blood relative, Caucasian parents. At the age of 20 months, she presented a cardiorespiratory arrest secondary to a polymorphic ventricular tachycardia refractory to cardiac defibrillations (Fig. 2A). At the autopsy, small yellow nodules were observed on the leaflets of the tricuspid valve (Fig. 2B). These nodules were composed of large, foamy, granular cells in the subendocardium. Immunostaining showed perimembranous reactivity for muscle-specific actin, but not for the histiocytic markers (PS100, CD68). Histological analysis revealed the presence of cellular clusters of histiocytoid cells in the subendocardial region, from the apex to the atrium, in the thickness of atrial septum and atrioventricular valve leaflets, resulting in the diagnosis of HC (Fig. 2C).

The diagnosis of HC should be systematically evoked in case of sudden death or severe ventricular arrhythmia in children, mainly in girls less than 2 years old.¹ Of the reported cases of HC, 19%

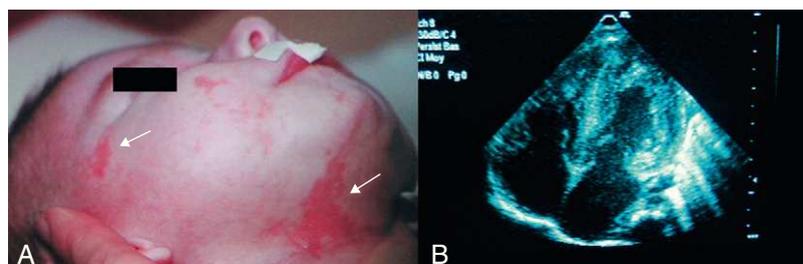


Figure 1. A: linear cutaneous erythematous lesions of the face and the neck (white arrows) typical of the microphthalmia with linear skin defects syndrome. B: trans-thoracic echocardiography showing hypertrophic cardiomyopathy.