Spontaneous Coronary Dissection: Role of Intravascular Ultrasound

Disección coronaria espontánea: papel del ultrasonido intravascular

To the Editor,

A 49-year-old woman with no cardiovascular risk factors was admitted to our department with high-risk non-ST-elevation acute coronary syndrome. Coronary angiography revealed severe stenosis in a segment located in the middle portion of the anterior descending artery, with no evidence of other lesions (Fig. 1). Given the ambiguity of the angiogram and the absence of risk factors, we decided to perform intravascular ultrasound, which revealed localized dissection that had not been visible in the angiogram (Fig. 1). There was no apparent intimal rupture but there was evidence of hematoma within the intima. Given the lack of information on the course of the lesion, and since it was located in the anterior descending artery of a young woman, we decided to perform intravascular ultrasound, which revealed localized dissection that had not been visible in the angiogram and the absence of risk factors, we decided to perform intravascular ultrasound, which revealed localized dissection that had not been visible in the angiogram.

Heyde’s Syndrome is an entity to be kept in mind, even more nowadays with an aging population, when assessing patients with a history of bleeding or anemia, especially when the bleeding site is not found on initial examination. Based on these data, we propose the hypothesis that this association could be a new indication for valve replacement; however, gastrointestinal bleeding is not used as an indicator in current clinical practice guides.

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observable flap and only vessel stenosis visible. Consequently, the use of intravascular ultrasound is important in young patients with no cardiovascular risk factors in whom spontaneous coronary dissection is suspected, since this allows diagnosis and can differentiate between the false and true lumens.

Consensus is lacking on the most appropriate treatment options, which depend on factors such as the number of affected vessels, whether the left main coronary artery is affected, distal coronary flow, and clinical stability. Thus, patients who are clinically unstable or have poor distal flow should be treated by percutaneous transluminal coronary angioplasty (PTCA) in localized dissections, whereas surgical revascularization would be a more appropriate option in patients with multivessel dissections, long lesions, or lesions of the left main coronary artery. In patients who have ACS with localized dissection and who remain asymptomatic, hemodynamically stable, and have good distal coronary flow, conservative treatment can be employed with double antiplatelet therapy and β-blockers, with angiographic follow-up in a few months, since in many cases the dissection is found to completely reseal. Other authors favor early, aggressive treatment with revascularization, since the natural course of this process is poorly understood.

In the case of PTCA, intravascular ultrasound should be used to ensure that the guidewire is in the native lumen and that stent placement does not cause propagation of the dissection or protrusion of a thrombus at one end.

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Acute Heart Failure in an Adult Patient With 2:1 Atrial Flutter: Zebras or Horses?

Insuficiencia cardiaca aguda en un adulto con flutter auricular 2:1: «¿cebras o caballos?»

To the Editor,

We present here the case of a 47-year-old patient with no past medical history of interest who went to the Accident and Emergency Department suffering from palpitations, progressive dyspnoea and orthopnoea. Examination revealed a II/IV systolic murmur; bibasal cracks; preserved and symmetrical pulses, albeit weak; blood pressure of 100/70 mmHg and cardiomegaly on chest X-ray. An electrocardiogram revealed 2:1 atrial flutter with a ventricular rate of 150 bpm. The emergency room, based on an initial diagnosis of suspected “tachycardiomyopathy”, administered digoxin, beta blockers, diuretics, and supplemental oxygen, thereby controlling the ventricular rate (75–100 bpm) with rapid clinical improvement. The next day, an echocardiography was performed that revealed left ventricular dysfunction with ejection fraction of 35%, dilated aortic root, moderate aortic insufficiency, and an abnormal mitral subvalvular apparatus with very elongated chords with a myxoid appearance with moderate mitral regurgitation (Fig. 1).

Given the new data, a multislice CT was performed, which showed a 49 mm aortic root, severe aortic coarctation (Fig. 1) and left atrial thrombus with no coronary abnormalities. Reviewing the basic diagnostic tests, the chest X-ray showed small costal notches that had gone unnoticed. The final diagnosis was left ventricular dysfunction by severe aortic coarctation with associated lesions of aortic and mitral insufficiencies. The rhythm became atrial fibrillation and cardioversion was not indicated by the left atrial thrombus. Cardiac catheterization was performed in an attempt to interrupt the aortic arch gradient of 40 mmHg. A radiofrequency catheter was used to get past the coarctation, allowing subsequent dilation and stenting (Fig. 2) with a disappearance of the gradient. At six months, the patient was asymptomatic, with an ejection fraction of 55% and the patient remained in sinus rhythm and asymptomatic with slight mitral regurgitation.

“Zebras or horses?” This case clearly illustrates one of the basic axioms of medicine: “when you hear hoofbeats behind you, it is almost certainly the sound of horses and not zebras.” This can be translated as “what is most frequent is most likely.” The presence of 2:1 atrial flutter with severe ventricular dysfunction in a young man without any past medical history made us think of the most obvious: dilated cardiomyopathy secondary to tachycardia. However, the echocardiogram revealed valvular disorders (pronounced myxoid degeneration of the mitral chordae and aortic annuloectasia). A subsequent multislice CT revealed another previously undetected disorder (severe aortic coarctation). Reviewing the medical history, blood pressure was normal (which can be due to heart failure), and femoral pulses were present and symmetrical in the arms (possibly due to collateral circulation). Only after a careful review of the X-rays were the small costal notches observed. Coarctation of the aorta constitutes 6% of congenital heart disease in childhood and 15% in adulthood.1 Its clinical manifestations depend on severity: in mild cases the manifestations do not appear until adulthood, usually with the discovery of hypertension. In our patient, however, there were no disorders until the onset of

Figure 1. Left: echocardiography showing linear images within the left ventricular cavity corresponding to very elongated mitral chordae (1). Right: aortic CT with striking collateral circulation (2), an image of aortic interruption (3), and dilation of the aortic root (4).