We report a case of spontaneous dissection of the left main coronary artery in a 32-year-old healthy woman that was unrelated with childbirth or other known risk factors. The clinical presentation was an extensive acute anterior myocardial infarction. She was treated by thrombolysis, but did not meet reperfusion criteria. Coronary angiography revealed dissection of the left main coronary artery that extended into the anterior descending and circumflex coronary arteries. Rescue angioplasty with stenting was performed. The clinical, diagnostic, and therapeutic features of this rare entity are discussed.

Key words: Acute myocardial infarction. Coronary artery dissection. Stent.

INTRODUCTION

Spontaneous coronary dissection is a serious and infrequent cause of acute coronary syndrome. Its true incidence rate is probably underestimated since in the majority of cases the diagnosis is established post mortem. Approximately two-thirds of published cases occurred in women and, among these patients, the most frequent location for the dissection was the left coronary artery.

A broad range of clinical presentations has been described, including stable and unstable angina, infarct, cardiogenic shock, and, frequently, sudden death. The etiology remains uncertain, and the majority of authors agree to the classification of these patients into 3 groups: those cases associated with atherosclerotic heart disease, those cases related to pregnancy and the post-partum period, and those cases without an identifiable cause.

CLINICAL CASE

Our patient was a 32-year-old woman, without known cardiac risk factors, with a gynecological history of 1 uncomplicated pregnancy 28 months previously, who went to the emergency room for an episode of prolonged central chest pain which began while the patient was at rest. Physical examination was normal and electrocardiogram showed ST segment changes on leads DI and AVL and precordial leads V2-V6. With the diagnosis of acute anterior myocardial infarct she was admitted to the coronary care unit, where intravenous thrombolytic treatment with tecneplase was administered without electrocardiographic signs of reperfusion. Four hours after the thrombolysis, the patient suffered another episode of chest pain, with hypotension and signs of left heart failure, and treatment with intravenous inotropic amines...
was begun and she was transferred to the hemodynamic lab for cardiac catheterization.

Following right femoral artery canalization, there was an elevation in left ventricular telediastolic pressure of up to 40 mm Hg. Left coronary angiography revealed the presence of a long dissection of the left coronary trunk (LCT) with a double distal extension; on one side toward the anterior descending artery, the proximal third of which was occluded (TIMI-1 flow) (Figure 1) and, on the other side, toward the circumflex artery up to its middle third, compromising the origin of the principal marginal branch, and with slowed distal flow (TIMI-2 flow). In the LCT the dissection extended proximally to the aortic ostium, causing, due to the compression of the true lumen by a false lumen, severe angiographic stenosis (Figure 1). Right coronary angiography did not show angiographic lesions.

We decided to treat the dissection percutaneously by using a seal with a stent. After insertion of a double coronary guide catheter (0.014”), with one end toward the anterior descending artery and the other in the circumflex artery, we implanted the first S7 stent (Medtronic AVE, Santa Rosa, Calif., USA) 18 mm in length and pre-mounted on a 4-mm diameter balloon, which was expanded to 12 atmospheres in the LCT, from the aortic ostium to the origin of the proximal anterior descending artery, covering the bifurcation of the circumflex artery. Following the proximal sealing of the dissection, there was improvement in flow in the circumflex artery. Given the persistence of the occlusion of the anterior descending artery from its proximal third, we treated distally the area treated with stents by the consecutive implantation of 3 Multilink Penta (Guidant, Santa Clara, CA) shunts: 3.5 mm×18 mm, 3 mm×13 mm, and 3 mm×18 mm (diameter×length). Finally, an excellent angiographic result was achieved, with re-canalization of the anterior descending artery (TIMI-3 flow) (Figure 2).

Left ventriculography revealed severe left ventricular systolic dysfunction (ejection fraction, 22%) by anterolateral akinesia and apical dyskinesia (Figure 3).

Later, the patient’s clinical course in the coronary care unit was favorable. The enzyme curve peaked at 10 hours after the procedure, with a creatinphosphokinase MB fraction of 197 ng/mL and a troponin I of 34.6 ng/mL. Serological study ruled out the existence of connective tissue disease. On hospital discharge the post-stent antithrombotic regimen was continued (oral clopidogrel 75 mg/day, oral aspirin 150 mg/day, and dalteparin 5000 U/12 hours) for 1 month following the procedure, in addition to treatment with ACEI and beta blockers.

The patient remained symptomatic at 6-month follow up, and angiographic re-evaluation at the end of this period showed the absence of re-stenosis in the segments treated with stents and the improvement of left ventricular systolic function (Figures 2 and 3).

**DISCUSSION**

Our case is considered a spontaneous coronary dissection, as the possibility of an iatrogenic lesion from the catheter over pre-existing atherosclerotic stenosis of the LCT capable of causing the clinical picture described is unlikely in a young patient without risk factors or obstructive lesions in other areas.

Excluding the group of individuals in whom spontaneous coronary dissection exists in association with pregnancy, it has been associated with habitual intense
physical exercise, the use of oral birth control medication, Ehlers-Danlos syndrome, eosinophilic syndrome, sarcoidosis, Kawasaki disease, fibromuscular dysplasia, hypersensitivity vasculitis, systemic lupus erythematosus, and, often, there are co-existent risk factors for atherosclerotic disease.

Multi-vessel changes are rare, tend to occur when the LCT is involved, and are associated with a poor prognosis, frequently ending in sudden death or extensive acute myocardial infarction.

Angiographically, coronary dissection is identified by the presence with the use of contrast material of a double lumen (a true lumen and a false lumen) separated by an intimal flap. In addition, the false lumen keeps the vessel wall tinted with contrast medium after the medium has been washed out.

Many therapeutic options have been suggested for this rare condition. Zampieri et al published a series of 5 patients who were treated conservatively (aspirin, nitrates, and beta-blockers), and did not show signs of recurrent ischemia after a mean of 25 months follow-up. Surgical revascularization has been shown to be beneficial in terms of survival, principally when there is multi-vessel involvement and when there is data indicative of recurrent ischemia. Results with thrombolysis are controversial; although cases have been published with favorable results where thrombolysis has been used to establish the diagnosis of acute coronary dissection, in others thrombolytic treatment caused expansion of the dissected area.

Percutaneous revascularization through the implantation of an intracoronary stent and sealing of the false lumen is the ideal treatment for selected cases of spontaneous coronary dissection. In recent years, multiple studies have reported excellent results in the treatment of atherosclerotic lesions affecting the unprotected LCT via the percutaneous implantation of stents, although, due to their principal limitation of stenosis, at present there is controversy concerning the selection of patients who may benefit from percutaneous treatment versus surgical treatment.

In our patient, the decision to proceed percutaneously via the implantation of a stent was based on 2 determining factors. The first was the unstable clinical situation of the patient, and on the second the fact that emergency surgical revascularization carries with it, in our hospital, the necessity of the patient being transferred to a referral hospital. Given that this condition in many cases has an accelerated fatal course, such a delay could put the patient at very high risk.
REFERENCES