Spontaneous coronary dissection during *postpartum*: etiology and controversies in management

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A 31-year-old female with a history of toxic oil syndrome in childhood, presented with spontaneous left main coronary dissection 4 weeks after an uncomplicated delivery. She had an extensive myocardial infarction, severe left ventricular dysfunction and cardiogenic shock which did not resolve following urgent surgical revascularization. Temporary left ventricular support and heart transplantation were necessary. We analyze the etiology and treatment sequence in what to our knowledge is the first case with these characteristics to be reported.

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Case report

Acute myocardial infarction due to spontaneous coronary dissection is an unusual pathology. The real incidence is unknown and the diagnosis is usually made *postmortem*. The etiology[1-3] remains unclear. Hormonal contraception, vasculitis, connective tissue diseases, intense physical exercise, von Willebrand disease, polycystic kidney disease, chest trauma and cocaine abuse have been proposed as risk factors, but pregnancy and *postpartum* are those more frequently related to coronary spontaneous dissection. This pathology mainly manifests during the first weeks of pregnancy and 3 months after delivery[3]. Seventy-eight percent of pregnancy-related cases appear in the immediate *postpartum*, with a 40% mortality rate[1,3]. Other risk factors for coronary disease are not usually found[1-3].

The left anterior descending coronary artery (LAD) is involved in 75% of dissections followed by the left main coronary artery (LM), right coronary and circumflex artery (CX). In 40% of patients all three coronary arteries are dissected[4]. More common histopathologic findings are cystic medial degeneration and eosinophilic periadventitial inflammation[2,5,6]. The plane of dissection runs in the media-adventitia junction, in the majority of cases without intimal tear, causing true lumen compression by hematoma.

The present case report refers to a 31-year-old female. Her history included toxic oil syndrome with mild neuromuscular involvement at 10 years of age which clinically resolved 2 years later. One month after her second uncomplicated delivery, she suffered an acute anterior myocardial infarction.

Initially, she received tissue-type plasminogen activator. Persistent chest pain and antero-lateral ischemia recurred after 40 min. The creatine phosphokinase peak level was 5602 U/ml and that of creatine kinase-MB 716 U/ml. Emergency coronary angiography showed LM dissection with occlusion of the LAD and severe involvement of the CX (Fig. 1). The left ventricular contractility was severely impaired (ejection fraction 15%). The patient was submitted to urgent surgical revascularization and the left internal mammary artery was anastomosed to the LAD and a saphenous vein graft was anastomosed to the obtuse marginal branch of the CX. An intra-aortic balloon pump was necessary during surgery as were high doses of inotropic drugs. Postoperatively, she remained hemodynamically unstable and progressively went into cardiogenic shock. Left ventricular support with the ABIOMED BSV 5000 system (ABIOMED Inc., Danvers, MA, USA) was implanted in order to treat incipient multiorgan failure and urgent heart transplantation was requested.

Transplantation was done 54 hours later, with no surgical complications. Postoperatively respiratory insufficiency and pulmonary hypertension were treated with inhalatory nitric oxide and intravenous prosta-

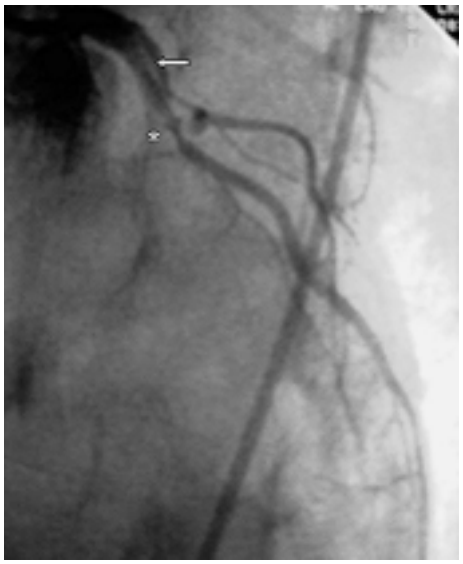

Figure 1. *Left main coronary artery dissection* (white arrow)*, complete occlusion of the left anterior descending artery and severe stenosis in the circumflex artery* (*).

cyclins. The patient was extubated at 5 days, and discharged home on the 45th day. Five months after transplantation she is in NYHA functional class I.

Discussion

Numerous factors are involved in spontaneous coronary dissection during pregnancy. These include hormonal changes in the arterial wall, vascular collagen degeneration and hemodynamic changes. All these situations would explain the development of hemorrhage in the coronary vasa vasorum. In our patient there was also another risk factor: a history of toxic oil syndrome during childhood. In 1981, Spain was afflicted with an epidemic of toxic oil syndrome. A new multisystem illness caused by ingestion of edible oil adulterated with denaturalized rapeseed oil, affected approximately 20 000 persons. The acute phase included dyspnea and symptoms suggesting atypical pneumonia. In the intermediate phase the respiratory syndrome remitted with progressive development of scleroderma, neuromyopathy and systemic dysfunction (myalgia, weight loss, liver disorders, pulmonary arterial hypertension, eosinophilia and thrombotic phenomena). The chronic phase continued over the subsequent years. The mortality, approximately 8%, was due to respiratory failure related to non-cardiogenic edema, severe neuromuscular deficit, large vessel thrombosis, and primary pulmonary hypertension. The heart histological findings in the necropsies of these patients included coronary arterial abnormalities such as focal fibromuscular dysplasia, myointimal cystic proliferation with degeneration or sloughing of the inner portion of the arterial wall (intima-media), abnormalities in the cardiac conduction system, and changes in the pulmonary arteries (hypertrophy and intimal proliferation) translating into pulmonary hypertension[7]. In our case, the histological findings were: cystic mean degeneration (mucinous cysts), a dissection plane between the media and adventitia, intramural hemorrhage with epicardial extension and arterial lumen compression (Fig. 2) and arterial intima enlargement and muscle cell hyperplasia alternating with intimal cysts. The LAD and CX arteries also presented moderate cystic changes without signs of dissection. Although a certain association cannot be identified, histological findings and postoperative pulmonary hypertension could relate coronary dissection to toxic oil syndrome.

In previous reports, no standardized treatment has been proposed for spontaneous coronary dissection[1-8]: survival is determined by the clinical evolution in the first hours after myocardial infarction. Urgent diagnostic coronary angiography is essential. Results with invasive techniques seem better than medical treatment. The use of fibrinolytics is controversial[2,3,5].

In our patient, the low cardiac output and cardiac failure conditioned survival. When an extensive area of myocardium is jeopardized, it is necessary to quickly attempt myocardial reperfusion. Ideally, stent insertion would contain and avoid progression in case of short dissections, affecting only one coronary vessel without total occlusion[3]. In our case, the positioning of a coronary stent was limited by severe dissection and occlusion; furthermore, the CX was severely stenosed. Surgical revascularization with bypass was ineffective. The later development of cardiogenic shock with multiorgan failure led to the use of the left ventricular assist device and subsequent cardiac transplantation. Only in three published reports did patients with spontaneous *postpartum* coronary dissection require cardiac transplantation[4-6]. In 2 cases, cardiac transplantation was performed late, due to a deferred cardiac failure. Only in 1 case was the patient transplanted during the acute phase and with the support of high doses of inotropic drugs and of the intra-aortic balloon pump.

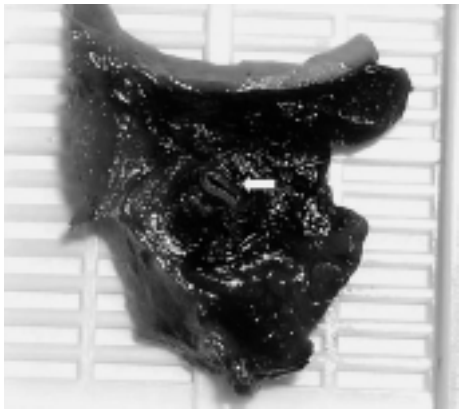

Figure 2. *Macroscopic cross-section of the left main coronary artery: an intramural hematoma extending to the epicardium compresses and totally occludes the true vessel lumen* (white arrow).

In our patient, initial improvement after fibrinolysis evolved to extensive uncontrollable myocardial ischemia. Clinical and histological findings lead us to think that, after fibrinolysis, the initial dissection progressed and that a more extensive area of myocardium was jeopardized.

Spontaneous coronary dissections should be considered in women who present with a myocardial infarction during pregnancy or during the first 3 *postpartum* months, without other coronary risk factors. It is advisable to avoid fibrinolytic treatment and to perform urgent coronary angiography. Immediate revascularization may be decisive for survival. In this case, toxic oil syndrome could have been an additional risk factor.

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