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Multivessel spontaneous dissection of the left coronary tree in the postpartum period: case report and review of the literature

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Abstract. – Spontaneous coronary artery dissection is a rare cause of myocardial infarction predominantly associated with young women during the third trimester of pregnancy or during the postpartum period. Multivessel spontaneous coronary artery dissection is an even less frequent condition with limited reports in medical literature. Hormonal changes as well as hemodymanic stress are some of the factors that have been implicated in the etiology of this condition. However, the exact pathophysiological process leading to spontaneous coronary artery dissection has not yet been elucidated. The spectrum of clinical presentation ranges from mild symptoms to cardiac arrest.

Herein, we report the case of a 39-year-old woman with spontaneous two coronary vessel dissection during her postpartum period presented with ST elevation myocardial infarction on electrocardiogram complicated with pulmonary edema and cardiorespiratory arrest.

Key Words:
Puerperium, Myocardial infarction, Coronary dissection, Thrombolysis.

Introduction

Acute myocardial infarction (AMI) during pregnancy is a very uncommon clinical condition. Furthermore, spontaneous coronary artery dissection (SCAD) although is a rare cause of AMI in the general population, it is usually responsible for the occurrence of acute coronary events in women during pregnancy or the postpartum period. In most cases the dissection involves one coronary artery and usually the left descending coronary artery (LAD). Multivessel SCAD is extremely rare and is often followed by more complicated clinical course and poor prognosis.

There is a wide spectrum of clinical presentations of this condition ranging from typical angina pectoris, in most cases, to sudden cardiac death. The limited data on this condition is responsible for the difficulty in elucidating its etiology and specifying treatment guidelines. The localization of the dissection and the number of vessels involved determine the treatment, which varies from conservative medical treatment to percutaneous intervention or a surgical approach.

We present SCAD in a young woman four days after an uncomplicated full term vaginal delivery. She was initially treated with fibrinolysis. The coronary angiography that followed revealed SCAD of two coronary arteries for which she underwent coronary artery bypass graft surgery (CABG).

Case Report

A 39-year-old woman, intubated, sedated and under inotropic agents for cardiogenic shock of four hours duration was transferred to our hospital from a maternity center. The patient had been previously hospitalized for four days after an uncomplicated childbirth. It was her second normal uncomplicated delivery of a healthy infant. On the day of planned discharge from hospital, she developed chest pain, dyspnea and acute pulmonary edema.

The patient's medical history was largely unremarkable, apart from Hashimoto's thyroiditis. There was no family history of Marfan's syndrome, sarcoidosis, connective tissue disorders, vasculitis or heart disease. Her social history was unremarkable for alcohol, tobacco, or illicit drug use. At the maternity hospital she had been diagnosed with acute pulmonary edema which had been complicated with a successfully inverted cardiorespiratory arrest. The electrocardiogram

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showed sinus rhythm and ST-segment elevation in all precordial leads. A bedside echo demonstrated global myocardial wall hypokinesis with a left ventricular ejection fraction (LVEF) of 25%. Intravenous alteplase and 8,000 IU of unfractionated heparin were administered. The patient was referred to our hospital for further investigation and treatment. Due to patient's condition an intraaortic balloon pump was inserted and immediate coronary angiography was performed. Coronary arteriography revealed a spiral dissection in the left main coronary artery (LMCA) extending down the LAD and the left circumflex (LCX) coronary arteries. The LAD was occluded whereas the LCX showed 60% stenosis (Figure 1). The right coronary artery (RCA) was normal. The likelihood of a successful recanalization of the LAD by a percutaneous technique was considered low due to the spiral dissection total occlusion of the vessel. The patient was urgently transferred to the operating room for CABG surgery. She received left internal mammary artery to the proximal LAD and two saphenous vein grafts – to the obtuse marginal branch of the LCX and the distal LAD respectively. Additionally, she received an extracorporeal membrane oxygenation levitronix device (ECMO) for left ventricular support. Peri- and postoperatively the patient exhibited excessive bleeding disorders; therefore, she was transfused with multiple units of red blood cells, fresh frozen plasma and platelets. She remained in the intensive care unit (ICU) for twenty-four days in a critical condition and died of multiple organ failure.

Discussion

SCAD comprises an uncommon cause of AMI, most commonly associated with pregnant women or those in the postpartum period. The first case of SCAD was documented in 1931 by Pretty on an autopsy performed on a 42-year-old female who presented with chest pain[1]

The presence of AMI under the age of 40 years is uncommon and especially in women, accounting for approximately 0.7% of all AMI cases[2]. Furthermore, the prevalence of AMI during pregnancy and puerperium reported by various studies ranges from 2.8-10 cases per 100,000 deliveries[3-5]. From AMI cases, during pregnancy and postpartum period, 27% are associated with SCAD compared to ≤ 1% in the general population. The remained AMI cases are being due to coronary atheromatosis or coronary thrombosis[4]. Moreover, multivessel involvement, as in our case, accounts for less than one-quarter of the cases. Independent predisposing factors for AMI

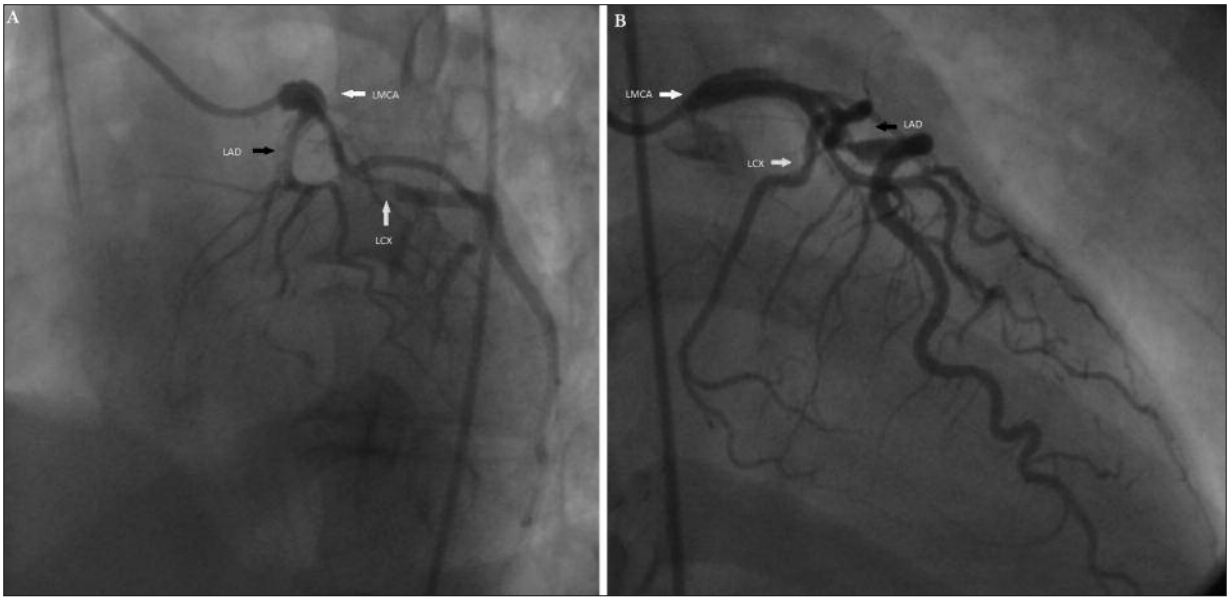

Figure 1. ***A,*** Coronary angiography in the left anterior oblique view demonstrating the LMCA dissection (*white arrow*), extending to the LAD (*black arrow*), which after the first septal and diagonal branch is totally occluded, and to the LCX (*yellow arrow*) creating significant proximal lumen loss with a good distal target. ***B,*** Coronary angiography in the right anterior oblique view revealing the LMCA dissection (*white arrow*) extending to both the LAD (*black arrow*) and the LCX (*yellow arrow*).

in pregnant women include age over 30 years, the third trimester of pregnancy, multiparity, hypertension, eclampsia and pre-eclampsia, diabetes mellitus, smoking, thrombophilia, need for blood transfusion and infection during delivery[3,4]. On the other hand, the occurrence of SCAD in the general population is between 0.28% and 1.1%, an estimation derived from studies of consecutive patients with AMI undergoing coronary angiography[6-7].

Additionally, the mortality rate in AMI due to SCAD in pregnant and postpartum women is relatively high, ranging from 38% to 66%[8,9]. It is estimated that the aforementioned mortality rate is constantly decreasing, presumably as an effect of earlier interventions and improvements in treatment of AMI[9].

The clinical presentation of SCAD ranges from asymptomatic pathology to AMI (> 80% presenting with ST-segment elevation on electrocardiogram) and sudden cardiac death[10].

Pregnancy and postpartum period are associated with several hormonal and hemodynamic alterations that can persist for up to six months after delivery. For the time being there is no unifying theory proposed accounting for the correlation between SCAD and the peripartum period. However, it is widely believed that a combination of morphological changes in the arterial wall and hemodynamic stress may be responsible for the condition described.

The pathophysiological changes during the peripartum period are believed to have an etiological role in SCAD. For example, the increase in cardiac output can enhance arterial shear forces and predispose to dissection. The excess progesterone can lead to smooth muscle hyperplasia, thus, weakening the arterial tunica media. The pregnancy hormone relaxin is believed to be increasing cardiac chronotropy and inotropy, and eosinophilia weakens the medial wall (although many suggest that this is reactive rather than causative). Additionally, the decreased collagen production (mechanism proposed to be similar to inborn defects such as Marfan's and Ehrler-Danlos syndromes), disruption of vasa vasorum and alterations in the coagulation-fibrinolysis system may lead to a prothrombotic state, while antiphospholipid antibodies, elevated lipoprotein (a) and decreased plasma factor lead to thromboembolic complications. Furthermore, oral estro-progestinic contraception, through increased water retention, cocaine abuse and intense physical exercise comprise rare causes of SCAD both in the general population and peripartum women. Finally, bromocriptine, which is used for lactation inhibition and may initiate coronary spasm, has been related to postpartum AMI and SCAD.

As far as the location of SCAD is concerned, a review of pregnancy-associated SCAD case reports shows that it generally involves the LAD solely or in concomitance with the LMCA and the RCA or the LCX[10]. To the best of our knowledge there are only two cases in the current literature reporting SCAD involving the LMCA, the LAD and the LCX arteries, simultaneously[11,12].

Currently, there are no guidelines that have been established regarding the optimal management strategy for patients with SCAD in the peripartum period. This may be the result of the rarity of the disease, reported sporadically with little long-term follow-up data available. However, the treatment strategy for peripartum SCAD should take into consideration the hemodynamic status of the patient, the extent of the myocardium at risk and certainly, in pregnant patients, the well-being of the fetus. Medical therapy is currently reserved for patients who have demonstrated limited dissection on angiography, percutaneous coronary intervention (PCI) being the treatment of choice in patients with ongoing signs of ischemia and single-vessel disease, or in those in whom a large, viable myocardial territory is at risk, such as proximal LAD disease. CABG surgery has been successfully used in SCAD involving the LMCA, multivessel dissections or in cases of medical or PCI failure.

Finally, pregnancy comprises a relative contraindication to thrombolysis, even though it is not believed to pose a direct risk to the fetus, since the placental transfer of thrombolytics is too low[13-15] and teratogenicity in animal models has not been observed. However, and this is valid under any circumstances of SCAD, thrombolysis of AMI caused by SCAD still remains controversial, since it is suggested that it may contribute to the extension of hemorrhage into the dissection plane[16], something that could be hypothesized for our patient.

Conclusions

SCAD is a rare cause of AMI, most commonly associated with pregnancy and puerperium. The overall mortality in those patients is in excess of 60%. Thus, the possibility of dissection should always be considered by the treating physician

when any woman during pregnancy or puerperium presents with chest pain and/or AMI. The physician's consideration of the described condition will be absolutely crucial for deciding the diagnostic and therapeutic strategy, and in the end absolutely crucial for the patient's life itself.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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