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Review article

Coronary disease in the puerperium

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Abstract

The manifestation of an acute coronary syndrome in women in the postpartum period is a rare but important clinical scenario, which, because of its multiple possible aetiologies, requires a different approach to management on the part of the clinician. Spontaneous coronary artery dissection (SCAD) is the most likely cause of an acute coronary syndrome in women in the puerperium. Unfortunately there is no medical consensus on the correct management pathway of such patients, but there is evidence that these patients have an improved prognosis when referred for bypass surgery or percutaneous intervention.

Keywords: Puerperium; Spontaneous coronary artery dissection; Eosinophils

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1. Spontaneous coronary artery dissection

Primary spontaneous coronary artery dissection (SCAD) is a rare, and often fatal, condition that must be considered when women in the puerperium present with an acute coronary syndrome. The puerperium is that period from delivery of the placenta until the reproductive organs return to their pre-pregnancy state 6 weeks post-partum. There have been approximately 250 cases of SCAD reported in the world literature and one third of these cases have occurred in women in the peripartum period [1].

The clinical spectrum of its presentation includes angina, myocardial infarction, cardiogenic shock and death. No specific cardiac risk factors have been associated with its occurrence. SCAD is the commonest cause of myocardial infarction post-partum [2], with an overall mortality of between 48 %and 66% [3,4]. In the largest review of such cases (56 cases), sudden cardiac death was the initial presentation in 27.9% (12/43) of cases. In women who did survive more than 24 h post-infarction, recurrent MI, usually due to a second coronary dissection, occurred in 20.8% (5/24). The left coronary artery was involved in 78.6% (44/56) of dissections and histopathological studies performed on 20 of these cases showed periadventitial inflammation in 80% (16/20). Of these 16 cases, 11 (68.8%) demonstrated periadventitial inflammation in which eosinophils predominated [4].

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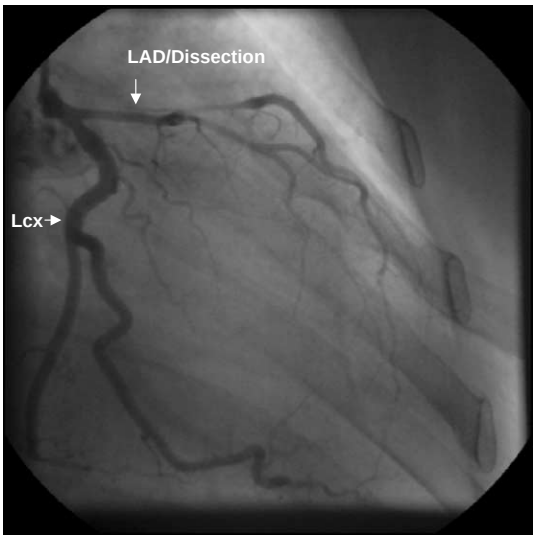

Fig. 1. Coronary angiogram of the left main coronary with left anterior descending (LAD) and left circumflex (Lcx) branches. There is a smooth diameter reduction of the lumen of the LAD of 80–90% at the origin of a diagonal branch. A separate line of contrast is seen tracking backward toward the left main coronary artery, indicated by arrow.

The classical histological finding is that of a large haematoma occupying the outer third of the media, resulting in complete compression of the true lumen. The cause of spontaneous dissection remains unclear but theories of aetiology include medial eosinophilic angiitis, pregnancy-induced degeneration of collagen in conjunction with the stresses of parturition, and rupture of the vasa vasorum [5].

2. Management

Optimal treatment remains unclear; the sporadic nature of SCAD has precluded the development of any consensus for a medical approach and treatment is usually tailored to individual patient needs. There would appear to be an obvious reason not to use fibrinolytics despite the fact that there are anecdotal case reports of thrombolysis being used successfully and safely in such cases [6,7]. The benefit of thrombolysis is controversial: hypothetically, a favourable effect could derive from the lysis of the thrombus forming inside the false lumen, allowing the adhesion of the intimal flap and, therefore, reconstitution of the true lumen and reperfusion. However, thrombolysis could also cause haemorrhage of the vasa vasorum with bloody effusions inside the vessel wall, causing compression of the true lumen, further calibre reduction, and extension of the dissected area through the increase of intraparietal pressure [8,9]. Studies have suggested that overall mortality is lower in patients treated surgically than in those managed medically [3]; however, cases where a percutaneous approach with stent deployment was possible have been reported with favourable outcomes [10,11]. Percutaneous intervention has been increasingly performed for SCAD. The deployment of a stent has the advantage that the stent tacks back the dissection flap and prevents further dissection at that site. However, the risk of stent deployment into the false lumen, completely obstructing the true lumen and coronary blood flow, exists. Critical to preventing this complication is to ensure that the guide-wire is not in the false lumen. Another possible complication of stent deployment is the extrusion of intramural thrombus either upstream or downstream of the stent, causing propagation of the dissection. Occasionally, in cases of SCAD, there are multiple noncontiguous coronary dissections, making bypass surgery or percutaneous intervention technically challenging. Noncontiguous dissections can manifest as very extensive myocardial infarction, and there are reports

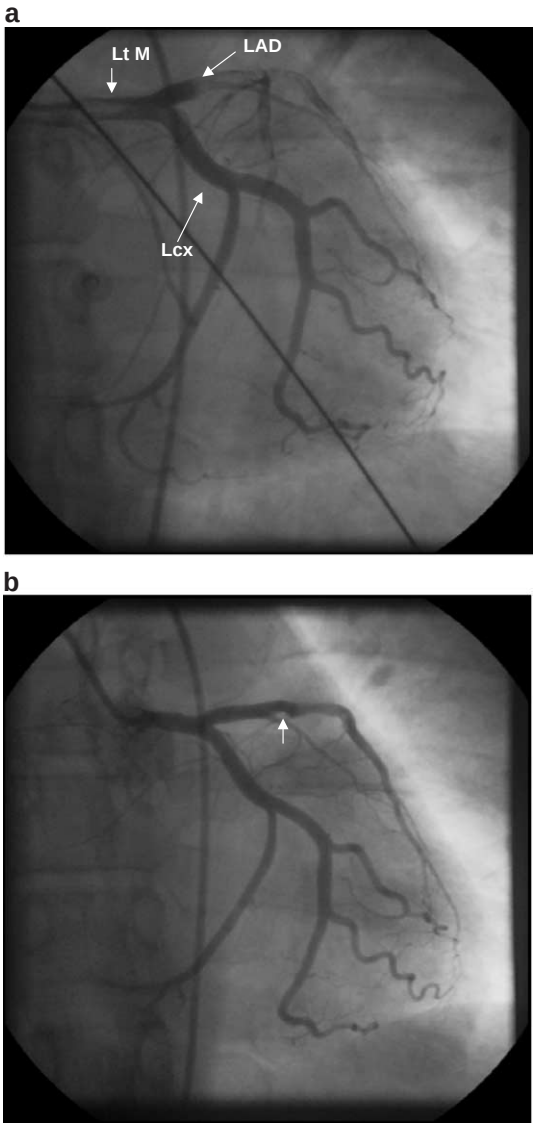

Fig. 2. (a) Coronary angiogram of the left main coronary (Lt M) with the left anterior descending (LAD) and left circumflex (Lcx) branches. The LAD is occluded proximally and thrombus is visible in the mid and distal LAD and diagonal branch. (b) Coronary angiogram of the left main coronary with the left anterior descending and left circumflex branches, performed on the same patient as in panel a 1 week later. The arrow indicates an ulcerated plaque in the proximal left anterior descending artery.