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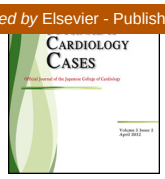

Editorial

Editorial: Spontaneous coronary artery dissection in pregnancy

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Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome or sudden cardiac death. The clinical presentation of SCAD depends on the extent and the flow-limiting severity of the coronary dissection, and ranges from asymptomatic to unstable angina, acute myocardial infarction, and ventricular arrhythmias to sudden cardiac death [1].

The incidence of SCAD in angiographic series varies widely from 0.07% up to 1.1% for patients who are referred for coronary angiography. The mean age at presentation is 30–45 years (range 30–70 years) and more than 70% of SCAD cases are women [2,3]. One third of all SCAD cases in women occur in the peripartum period, of which one third occur in late pregnancy and two thirds in the early postpartum period. The peak incidence is within the first 2 weeks after delivery [1].

In a population-based study of more than 12 million deliveries in the USA, the incidence of acute myocardial infarction was 6.2 per 100 000 deliveries [4]. The prevalence of pregnancy-related acute myocardial infarction in Japan seems lower than in Western countries [5]. Evaluation of coronary artery morphology (angiographically or at autopsy) revealed a dissection only in 27% of the patients, whereas a coronary stenosis due to atherosclerosis was observed in 40%. Coronary dissection was the primary cause of infarction in the peripartum period (50%) and was found more commonly in postpartum compared with antepartum cases (34% vs. 11%) [6].

The pathogenesis of SCAD in the peripartum period is still unclear. Hemodynamic factors together with arterial wall changes related to pregnancy, a lytic action of proteases released from eosinophils, and intimal tears are the main hypotheses presented to explain the pathophysiology involved. First, changes in the concentrations of sex hormones are thought to alter the normal arterial wall architecture, resulting in an increased susceptibility to spontaneous dissection. The changes in the vascular wall include smooth muscle cell proliferation, impaired collagen synthesis, and alterations in the protein and acid mucopolysaccharide content of the media. Also, total blood volume and cardiac output are increased during pregnancy. This may lead to augmented shear forces on the luminal surface and an increased wall stress in pregnancy and particularly during labor. These changes predispose the coronary arteries to the development of intramural dissections [1]. Second, a periadventitial infiltrate composed of eosinophilic granulocytes involving the vasa vasorum has been observed in patients with SCAD. The infiltrate of eosinophils that can be observed in dissected coronary arteries could be a systemic manifestation of this process. Eosinophil granules contain lytic enzymes including collagenase, major basic protein, and other substances that have a cytotoxic effect. It was postulated that spontaneous dissection could result from the breakdown of media-adventitial wall layers by the substances released by activated eosinophils [1,7]. Third, since an intimal tear is found only infrequently at autopsy, disruption of the vasa vasorum leading to intramedial hemorrhage and subsequent dissection without an intimal tear also has been proposed as a possible mechanism in this subset of patients with SCAD [1,8].

Whenever a young patient without major coronary risk factors or a woman in the postpartum period presents with acute coronary syndrome or sudden death, the possibility of a SCAD should be suspected and an urgent coronary angiography considered [1]. There is no specific guideline on how to manage patients with SCAD. Treatment options for SCAD include medical therapy, percutaneous coronary intervention (PCI), or coronary artery bypass graft surgery (CABG). When there is no evidence of ongoing ischemia or hemodynamic instability, SCAD can be managed successfully with medical treatment alone. If a pronounced dissection persists in a major vessel after prolonged medical treatment, or in SCAD causing marked epicardial coronary flow impairment and/or ongoing ischemia, PCI or CABG should be considered [1]. Initial conservative management and CABG were associated with an uncomplicated in-hospital course, whereas PCI was complicated by technical failure in 35%. In this regard, PCI was associated with elevated rates of technical failure relating to passage of coronary wire into the false lumen of the dissected vessel or loss of coronary flow through propagation of dissection and displacement of intramural hematoma by stent placement [9].

In this issue of the *Journal of Cardiology Cases*, Ryshten et al. present a thought-provoking case report of a postpartum woman with SCAD-related acute myocardial infarction who survived cardiac shock by excellent PCI [10].

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