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Reviews

Spontaneous Coronary Artery Dissection

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Summary: Spontaneous coronary artery dissection (SCAD) is an unusual cause of acute myocardial ischemia with complex pathophysiology. This paper reviews the major diagnostic and therapeutic issues of this rare but important disease. The diagnosis of SCAD should be strongly considered in any patient who presents with symptoms suggestive of acute myocardial ischemia, particularly in young subjects without traditional risk factors for coronary artery disease (especially in young women during the peripartum period or in association with oral contraceptive use). Urgent coronary angiography is indicated to establish the diagnosis and to determine the appropriate therapeutic approach. The decision to pursue medical management, percutaneous coronary intervention, or surgical revascularization is based primarily on the clinical presentation, extent of dissection, and amount of ischemic myocardium at risk.

Key words: coronary vessel, coronary artery disease, dissection, myocardial ischemia, acute coronary syndrome, spontaneous coronary artery dissection, peripartum complication

Introduction

Spontaneous coronary artery dissection (SCAD) is an unusual cause of acute myocardial ischemia with complex pathophysiology. Fewer than 200 cases of SCAD have been reported in the literature, and the majority (80%) have occurred in young women during the peripartum period or in association with oral contraceptive use. Although medical therapy and revascularization using percutaneous or surgical procedures are available treatment modalities in these patients, the optimal strategy for this disease process has not been clearly defined. This article will review the major diagnostic and therapeutic issues of this rare but important disease.

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Incidence and Clinical Presentation

The clinical presentation of this syndrome relates to the extent and rate of dissection as well as the degree of myocardial ischemia.[1] Patients may present with chronic stable angina, acute coronary syndromes, myocardial infarction, cardiogenic shock, sudden cardiac death, or pericardial tamponade. Sudden death may occur in up to 50% of cases, particularly in those with left main coronary artery (LMCA) dissection.[2–6]

Patients with SCAD have traditionally been assigned to three groups: those who present with (a) significant preexisting atherosclerosis, (b) during the peripartum period or in association with oral contraceptive use, and (c) idiopathic presentation.[1, 7–10] Women with SCAD frequently present during the peripartum period or in association with oral contraceptive use with involvement of the left anterior descending (LAD) artery and/or LMCA, usually in the absence of traditional risk factors for coronary artery disease (CAD). In their review of peripartum cases, Koul *et al.*[11] reported that 22% presented during pregnancy and 78% presented in the postpartum period. Most cases occurred within 2 weeks of delivery, but some patients presented as long as 10 to 12 weeks postpartum. Spontaneous coronary artery dissection secondary to connective tissue disorders is more commonly seen in women, and they may also present after an episode of intense physical activity.[8, 9] Men frequently present at a slightly later age, usually with involvement of the right coronary artery (RCA), with evidence of CAD or risk factors for atherosclerosis.[1, 8, 12, 13]

Unlike intimal dissection that occurs with significant preexisting atherosclerosis, the plane of dissection in SCAD usually lies within the outer third of the tunica media or between the media and adventitia.[11, 14–18] The dissection is typically observed in one vessel, but may encompass several coronary arteries concomitantly, including the LMCA.[2, 9, 19–23] Although not invariable, left coronary artery dissections are more common in women, while the RCA is usually involved

in men.[4, 8–10, 24, 25] Overall, the LAD artery is affected in 75% of cases, the RCA in 20% of cases, the left circumflex artery in about 4% of cases, and the LMCA in < 1% of cases.[9, 26]

Etiology and Pathogenesis

The precise etiology of SCAD is multifactorial, complex, and largely unknown. Several authors have speculated that dissection during pregnancy may be due to changes in the media due to increasing hormone levels, in addition to the shear stress present during labor.[2, 11, 27–35] In addition to damage to the media, the hormonally induced changes in pregnancy have included fragmentation of reticulin fibers, the loosening of ground substance, and the hypertrophy of smooth muscle, which in combination may significantly increase the risk of dissection.[36] Due to these factors, multiparous women appear to be at greater risk of developing SCAD.[37]

Vasculitis has been noted in several cases.[8, 10, 18, 38–42] The presence of eosinophilic infiltrates, periarteritis-like adventitial changes, tissue-damaging granule components, and the close interaction between eosinophils and degraded collagen have been well documented.[4, 11, 43–45]

Coronary artery dissection can occur secondary to atherosclerosis after rupture of a plaque or vasa vasorum.[16, 46, 47] Nevertheless, some authors have suggested that atherosclerosis itself may prevent SCAD via a stenting effect, whereby scarring and atrophy of the media prevents extension of the dissection and may be associated with improved collateral circulation.

Other conditions associated with SCAD include the following: systemic lupus erythematosis,[48] hypersensitivity angiitis,[37, 49] blunt chest trauma,[50–53] intense physical exercise,[54–56] sarcoidosis,[57] mitral stenosis secondary to rheumatic coronary arteritis,[58] fibromuscular dysplasia,[23, 59] cocaine use,[60, 61] old age with and without CAD,[19, 62] cardiopulmonary resuscitation,[3] copper deficiency causes,[36] apical hypertrophic cardiomyopathy and cryoglobulinemia associated with hepatitis C virus,[63] and hypertension.[4, 64]

Diagnosis

The majority of early cases of SCAD were diagnosed at autopsy.[2, 8, 9, 47] Antemortem diagnosis is confirmed with coronary angiography,[2, 65] and the presence of extraluminal radiolucent contrast after washout of dye from the remainder of the vessel as well as a radiolucent intimal flap are indicators of dissection (Fig. 1).[8, 9, 19, 66–68] An intimal tear may or may not be present, and the dissection may be obscured by significant narrowing of the true lumen.[69, 70] It is important to rule out catheter-induced trauma to the vessel as the possible etiology of the dissection. When angiography is ambiguous, intravascular ultrasound (IVUS) is employed to distinguish dissection from atherosclerosis and to determine the morphology of the dissection.[10]

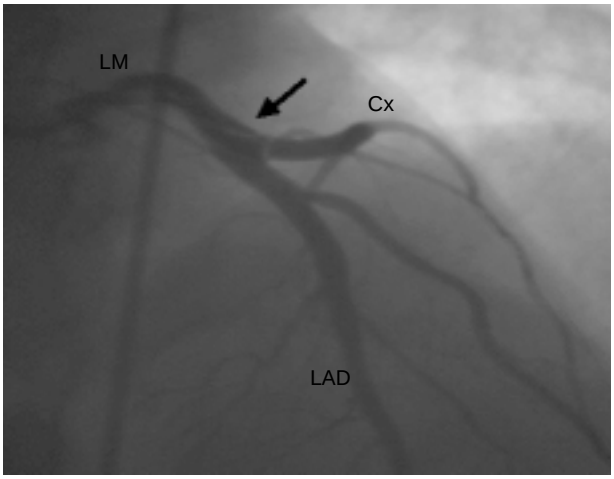

FIG. 1 Angiographic image showing spontaneous coronary dissection of the left main (LM) artery, the left anterior descending (LAD) artery, and the left circumflex (Cx) artery.

Treatment

The treatment options for SCAD include medical therapy and revascularization procedures using percutaneous coronary intervention or coronary artery bypass graft surgery. The decision of which treatment modality to utilize is dependent on the clinical presentation, the extent of coronary dissection, and amount of jeopardized myocardium present. Conservative treatment with aspirin and other antiplatelet medications, antithrombin agents, nitrates, and beta blockers may be a reasonable approach in asymptomatic, stable patients with limited dissections. Favorable long-term clinical outcomes using a conservative approach have been reported in this population.[1, 9, 11, 34, 71] Based on the findings of periadventitial inflammation in SCAD, favorable results with immunosupppresive therapy with prednisone and cyclophosphamide have been reported.[44] Thrombolytic therapy is relatively contraindicated in SCAD due to the potential risk of worsening the dissection.[54, 72, 73] There are limited data on the utilization of newer antiplatelet agents such as glycoprotein IIb/IIIa inhibitors in SCAD. Cheung *et al.*[51] reported successful use of glycoprotein IIb/IIIa inhibitor therapy with resolution of dissection, observed by angiography, within 20 h. Treatment with percutaneous coronary balloon angioplasty alone has been reported;[74–78] however, intracoronary stenting may be the preferred percutaneous treatment modality for patients with single-vessel SCAD, not involving the LMCA, and in patients who present with acute coronary syndromes or recurrent ischemia, due to the potential for the stent to obliterate the false lumen.[34, 48, 25, 72, 74, 75, 78–80] Surgical revascularization is indicated with SCAD with multivessel involvement, LMCA involvement, evolution of the dissection and/or narrowing of the lumen,[11] or refractory recurrent ischemia.[8, 25, 34, 81, 82] Orthotopic heart transplantation has been employed in cases of severe heart failure.[83, 84]

Complications of SCAD include extension of dissection, new dissections, and recurrent infarctions.[44] Patients with limited dissection, incomplete obstruction, and infarction without complications have a better prognosis. Prognosis is poorer with LMCA, LAD, and multivessel involvement, which typically result in extensive myocardial infarction or sudden death.[69] The impact of advanced adjunctive medical and pharmacologic agents, percutaneous coronary interventional devices, and surgical techniques on improving the short- and long-term outcomes in SCAD deserve further study.

Conclusions

Spontaneous coronary artery dissection is an unusual cause of acute myocardial ischemia with a wide range of clinical presentations. It usually occurs in young women during the peripartum period, or in those taking oral contraceptives, although men may also be affected. The diagnosis of SCAD should be strongly considered in any patient who presents with symptoms suggestive of acute myocardial ischemia, particularly in young subjects without traditional risk factors for CAD. Urgent coronary angiography is indicated to establish the diagnosis and to determine the appropriate therapeutic approach. The decision to pursue medical management, percutaneous coronary intervention, or surgical revascularization is based primarily on the clinical presentation, extent of dissection, and amount of ischemic myocardium at risk. The expeditious diagnosis and proper selection of the optimal treatment strategy for this rare but important syndrome will remain a challenge for clinicians.

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