Optical Coherence Tomography in the Diagnosis and Management of Spontaneous Coronary Artery Dissection

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KEYWORDS

• Spontaneous coronary artery dissection • Optical coherence tomography • Intravascular ultrasound • Intracoronary imaging

KEY POINTS

- Spontaneous coronary artery dissection (SCAD) is an infrequent condition that has been underdiagnosed and misdiagnosed.
- Use of intracoronary imaging with intravascular ultrasound (IVUS) or optical coherence tomography (OCT) enables the accurate diagnosis of this challenging condition.
- Diagnostic and management algorithms have been proposed to improve the diagnosis and therapeutic stratification of this condition.
- OCT has better spatial resolution than does IVUS and is instrumental in the diagnosis of SCAD cases where angiographic findings are ambiguous for confirming SCAD.
- Understanding the role and appropriate use of this technology is expected to improve the diagnosis of SCAD, and also improve outcomes with percutaneous intervention, when clinically indicated.

SPONTANEOUS CORONARY ARTERY DISSECTION

Epidemiology

Spontaneous coronary artery dissection (SCAD) is a clinically challenging entity that is an important cause of acute myocardial ischemia/infarction and sudden cardiac death in women.[1] The first case of SCAD was described in 1931 on autopsy of a 41-year-old woman presenting with sudden cardiac death and without classic risk factors for atherosclerotic disease. The first angiographic report of SCAD was in 1973 by Forker and colleagues[2] describing the angiographic appearance of extraluminal dye. Since then, fewer than 1000 cases of SCAD have been noted in the medical literature. Retrospective registries have reported SCAD in 0.07% to 1.1% of all coronary angiograms.[3–6] Previous reports have alluded to the rare observation of SCAD as a causative element in acute coronary syndrome (ACS) and sudden cardiac death, accounting for 0.1% to 4% and 0.4%, respectively.[5,7] In a series

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by Vanzetto and colleagues,[3] the prevalence was higher among women age younger than 50, where it accounted for 8.7% of troponin-positive ACS. We recently reported a retrospective series in which 24% of women younger than age 50 who were undergoing coronary angiography had angiographically detectable SCAD.[1,8] Taken together, these data suggest that SCAD is much more prevalent than previously considered; however, at present, the true population-based incidence of SCAD remains unknown.

There are several reasons why the incidence of SCAD has been underestimated in the medical literature. The association of SCAD with cardiac arrest may identify a cohort of patients who die before presentation to hospital. Second, many cases of SCAD may have been mistakenly identified as atherosclerotic coronary dissection, which is a mechanically distinct variant from non-atherosclerotic SCAD. Finally, given the often subtle clinical and angiographic presentation, it remains possible that many cases of SCAD have been misdiagnosed as mild atherosclerotic disease or missed altogether.

More recently, through meticulous angiographic review, we and others have described larger cohorts of patients with SCAD and identified SCAD as a far more prevalent cause of ACS in young women.[9–11] In a retrospective single-center cohort study from the Mayo clinic,[10] 87 angiographically confirmed cases of SCAD were identified. Among those with angiographically confirmed SCAD, 82% were female and the mean age was 43 years. The initial clinical presentation was ST-elevation myocardial infarction in 49% of cases. SCAD recurred in 17% with a 10-year recurrence rate of 29%, underscoring the need for close follow-up. In the Madrid cohort,[9] a prospective series of 45 patients with SCAD treated conservatively were followed for more than 6 years. Again, most were young (<50 years old) women presenting with acute myocardial infarction. The predominant angiographic appearance of SCAD in this series was a long diffuse narrowing (type 2 angiographic SCAD, discussed later) rather than presenting as an intimal flap or vessel wall stain. In those cases with angiographic follow-up, more than 50% of SCAD lesions had resolved with conservative therapy, underscoring the natural history of this disease and providing a clear rationale for conservative management.

We recently reported the largest cohort of prospectively and retrospectively identified patients with SCAD (N = 168).[11] In this cohort, 92% of patients were female (62% of whom were postmenopausal) and the mean age was 52. The dominant angiographic appearance of SCAD was again that of a smooth diffuse narrowing (type 2 angiographic SCAD), observed in 67% of cases. We examined the prevalence of potential predisposing conditions and demonstrated evidence of fibromuscular dysplasia (FMD) in 72%. Spontaneous angiographic "healing" of SCAD was observed in all 79 cases with angiographic follow-up, supporting the use of a conservative management strategy in most patients. Taken together, these more recent data identify SCAD as a clinically important cause of ACS in women, and should be considered in the differential diagnosis.

Pathogenesis

SCAD is defined as a nontraumatic and noniatrogenic separation of the coronary arterial wall by intramural hemorrhage and the resultant creation of a false lumen. The dissection plane can occur at the intimal-medial or medial-adventitial interface and need not have an intimal dissection flap.[12] The resulting intramural hematoma (IMH) can occlude or compromise the true vessel lumen leading to myocardial ischemia and infarction.

There are two proposed mechanisms of SCAD (Fig. 1). The first includes initiation of medial dissection and hemorrhage by an intimal tear and creation of a false lumen. The second involves the spontaneous development of an IMH potentially caused by disruption of the intra-arterial vasa vasorum.[13]

The cause of SCAD is multifactorial with contribution of a predisposing arteriopathy (resulting in vulnerable vessel wall segments) and precipitating stressor events. Predisposing arteriopathies can be broadly classified as atherosclerotic SCAD and non-atherosclerotic SCAD (NA-SCAD).[11] Disruption of the atherosclerotic intima can lead to SCAD; however, these dissections tend to be limited in extent by medial atrophy and scarring.[14] Predisposing arteriopathies in NA-SCAD include peripartum (likely a culmination of hormonal exposure and hemodynamic changes during pregnancy), multiple previous pregnancies,[15] connective tissue disorders (eg, Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome type 4, cystic medial necrosis, α_1-antitrypsin deficiency, and polycystic kidney disease), systemic inflammatory conditions (eg, systemic lupus erythematosus, Crohn disease, ulcerative colitis, polyarteritis nodosa, sarcoidosis, Churg-Strauss syndrome, Wegener granulomatosis, rheumatoid arthritis, Kawasaki, giant cell arteritis, and celiac disease), coronary spasm, or idiopathic.[11]

The predominance of female sex in the SCAD population seems to support a mechanistic role