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Amr Elkammash, *

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Review Article

Spontaneous Coronary Artery Dissection; A Literature Review

Parkha Khan [1], Amr Elkammash [2,3*]

[1] Leeds Teaching Hospitals NHS Trust, Leeds, UK.

[2] Cambridge University Hospitals NHS Foundation Trust, Cambridge, UK.

***Corresponding Author:** Amr Elkammash, MRCPUK, MSc. Cambridge University Hospitals, Cambridge, UK.

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Abstract

Spontaneous coronary artery dissection (SCAD) is the spontaneous splitting of the coronary tunica media with the formation of a false lumen in the vessel wall and compression of the true lumen. Its incidence is often underestimated, especially in cases of sudden cardiac death before coronary angiography. Predisposing factors include pregnancy, systemic arteriopathy (such as fibromuscular dysplasia), systemic inflammatory conditions (such as systemic lupus erythematosus and polyarteritis nodosa), systemic connective tissue diseases (such as Marfan and Ehlers-Danlos syndromes), hypertension and migraine. Common triggering factors involve straining, stress, and hormonal treatment. SCAD is usually diagnosed through coronary angiography, with CT coronary angiography as a non-invasive diagnostic tool. Experts advise minimal coronary instrumentation due to vessel fragility and the risk of dissection extension and vessel occlusion. SCAD patients can have percutaneous coronary intervention (PCI) if they have ongoing ischemia or hemodynamic instability. Coronary artery bypass grafting (CABG) can be an option in cases of failed PCI and high-risk anatomy.

Keywords: spontaneous coronary dissection; pregnancy; fibromuscular dysplasia; coronary; grafts; predisposing factors; connective tissue diseases

Introduction:

Spontaneous coronary artery dissection (SCAD) is a kind of coronary dissection that is not caused by atherosclerosis, trauma, or coronary intervention [1]. Its incidence rate has recently increased, probably due to advancements in intracoronary imaging techniques [2]. It is responsible for 1-4% of cases of acute coronary syndrome (ACS) [3]. It also forms 35% of the cases of myocardial infarction (MI) in women aged 50 years or older [4]. It is considered the commonest cause of MI in pregnant women [5]. The left anterior descending coronary artery is the commonest coronary artery affected by this condition [6]. The clinical data on SCAD is still relatively scarce, leading to impaired recognition of the disorder [7]. Therefore, the authors of this work put effort into gathering most of the available data on the topic, hoping to increase the medical community's awareness about it.

Histology:

The histological hallmark of SCAD is the presence of intramural hematoma (IMH) within the tunica media, compressing the true lumen [8]. Two theories have been proposed to explain the such finding. The first is the occurrence of intimal disruption, leading to the tracking of blood away from the true lumen into the false lumen. The second is the spontaneous rupture of the blood vessels in the vasavasorum, leading to intramural bleeding [9].

Etiology:

SCAD happens due to the interaction between underlying predisposing factors in the affected subject and environmental precipitants [1]. (figure 1) Pregnancy is one of the prominent risk factors for SCAD.

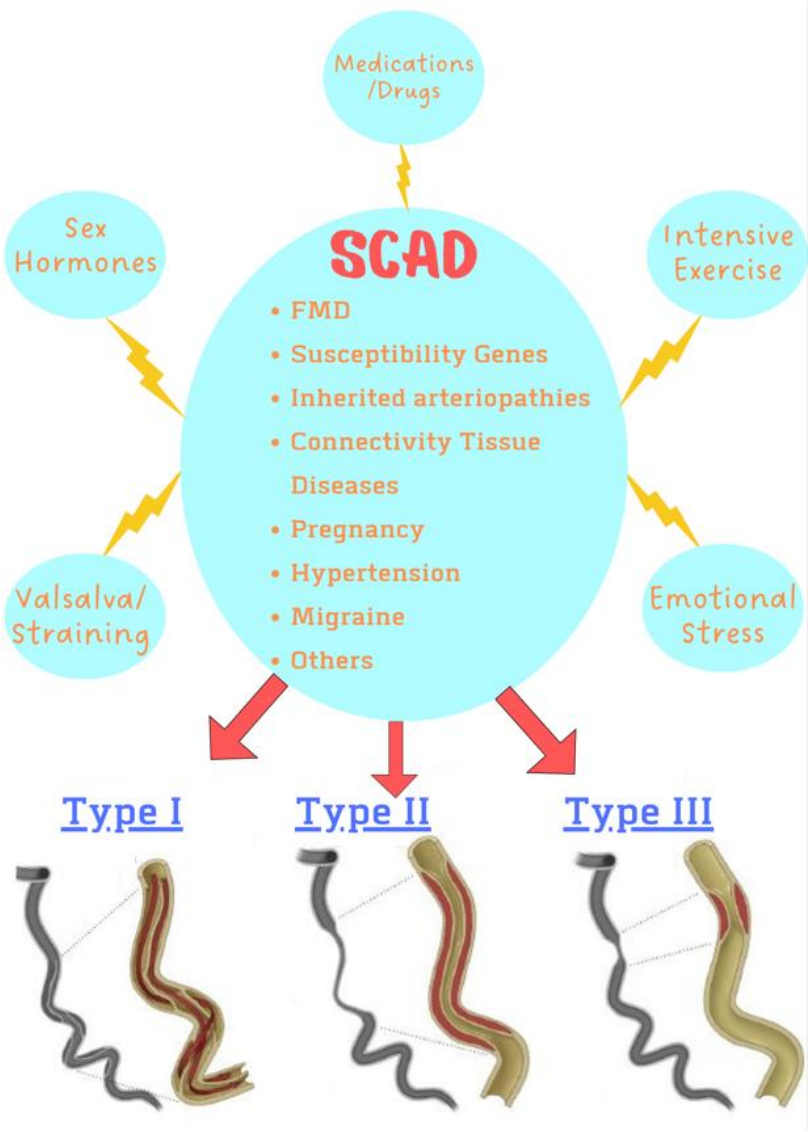

Figure 1: The interaction between the predisposing and triggering factors for producing SCAD.

It was hypothesized that pregnancy induces changes in the blood vessels, vis the estrogen and progesterone receptors, leading to blood vessels' weakness and IMH [5]. SCAD happens in the third trimester and early postpartum in most instances [10]. The incidence is higher in multiparous women [11]. The risk factors for pregnancy-associated SCAD are the black race, high blood pressure, dyslipidemia, depression, migraine, advanced maternal age, and infertility treatment [10,11]. The condition is associated with a higher complication rate, including cardiogenic shock, ventricular fibrillation, and in-hospital mortality [12]. Fibromuscular dysplasia is strongly linked with SCAD [13]. It is a non-atherosclerotic blood vessel disease with abnormal cellular growth in the vessel wall leading to vessel narrowing, dilatation, or dissection [14]. Other peripheral arteriopathies in patients with SCAD include dissections, aneurysms, and tortuosities [1]. Patients with systemic inflammatory conditions are at a higher risk of having SCAD. These include patients with systemic lupus erythematosus, inflammatory bowel disease, polyarteritis nodosa, sarcoidosis, celiac disease, and hepatitis C patients with cryoglobulinemia [1]. There are reported cases of SCAD in patients with connective tissue disorders, such as Marfan and Ehlers Danlos syndromes [15] and Loeys Dietz syndrome [16]. However, SCAD did not appear to have familial inheritance [17].

The reported triggering factors involve high physical and emotional stress, straining [18], high-dose corticosteroids [19], oral contraceptive pills [20], and infertility treatment [21].

Clinical presentation:

Most cases of SCAD present as MI (STEMI or NSTEMI) (90%). Other presentations can include ventricular dysrhythmias (up to 5%) and cardiogenic shock (2%) [22]. Sudden cardiac death can occur in up to 11% of patients [3].

Diagnosis:

1- Coronary angiography:

The first presentation as ACS makes coronary angiography the first line of investigation for SCAD. SCAD can be divided into three types based on its angiographic appearance [23]. (table 1) The LAD is the most commonly affected coronary artery (especially in the mid and distal segments);

however, multivessel SCAD was reported in 5-13% of the cases [24]. An increased coronary tortuosity puts the patient at risk of SCAD recurrence [25]. Intracoronary imaging using intravascular ultrasound (IVUS) or optical coronary tomography (OCT) may be needed to differentiate SCAD from its mimics (e.g., coronary thrombus) [26,27].

SCAD type	Description	Incidence
Type I	Visible intimal flap with the contrast running in the false lumen	29%
Type II	Long segment (>20mm) of intra-mural hematoma and luminal narrowing without an obvious intimal tear.	67%
Type III	Short segment (<20mm) of intra-mural hematoma and luminal compression without an obvious intimal tear.	4%

Table 1: Types of SCAD according to their angiographic morphology and their incidence rates:

2. Intracoronary imaging:

The coronary fragility and the tortuosity, the small vessel diameter, and the distality of the lesions make intracoronary imaging an unsafe option to diagnose SCAD [28]. Therefore, IVUS and OCT are usually used for cases of diagnostic uncertainty, preferably to scan the proximal wider segments of the vessels to avoid extension of the dissection [29]. OCT is superior to IVUS in detecting SCAD due to the increased spatial resolution [30].

3. Coronary Computed Tomography Angiography (CTCA):

CTCA is a useful non-invasive modality for the follow-up of SCAD patients with the involvement of large vessels and proximal segments [31]. It is not recommended as a first-line investigation in acute ACS patients with presumed acute SCAD due to the false negative results [32]. The challenges with using CTCA include the lower spatial and temporal resolution relative to coronary angiography and the motion artifacts. These limit the diagnostic accuracy of CTCA in diagnosing SCAD, especially in small-sized vessels [29].

Treatment of SCAD:

A flow chart summarizes the management in figure 2.

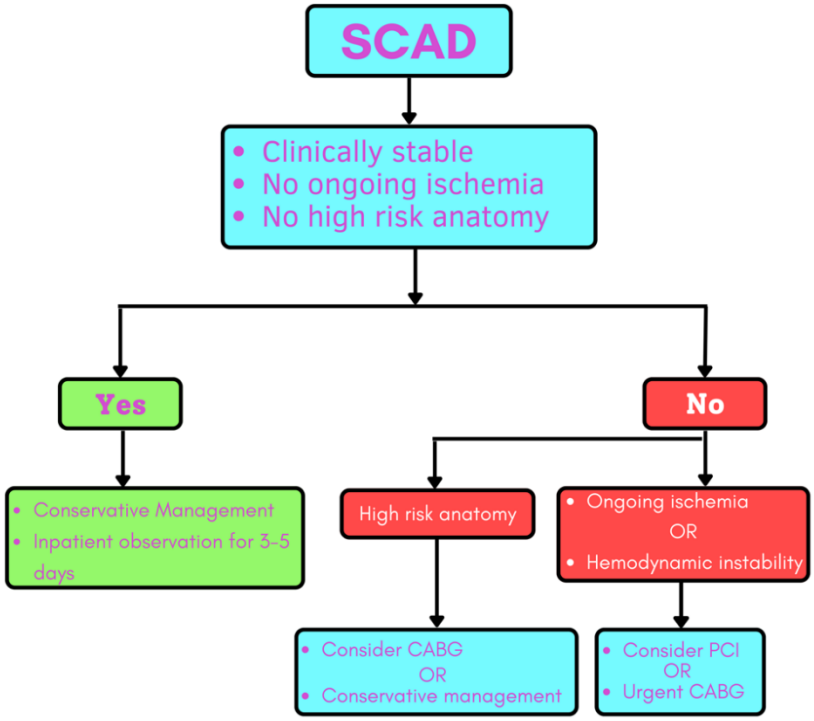

Figure 2: A flow chart summarizing the management of SCAD.

Medical treatment:

Treatment of ACS secondary to SCAD usually starts as per guidelines' recommendations [33,34]. After the angiographic diagnosis of SCAD, the anticoagulation should be stopped, and thrombolytics become contraindicated [24]. There are different approaches to deciding the duration of dual antiplatelet (DAPT) after diagnosing SCAD. The first is to use DAPT for one year after the diagnosis, followed by life-long Aspirin if there are no contraindications [35]. The second is to give DAPT for a shorter duration (1-3 months) followed by life-long Aspirin if there is no contraindication [22]. Beta-blockers are mandatory in SCAD patients with heart failure and arrhythmias [1]. There is evidence that beta-blockers can reduce the recurrence rate of SCAD in at-risk individuals [36]. Anti-anginal

medications (Nitrates, Calcium channel blockers, and Ranolazine) can be used in SCAD patients with ongoing angina and evidence of coronary spasm or microvascular dysfunction. Other guideline-recommended ACS medications (Statins and Renin-Angiotensin-Aldosterone system blockers) have no added benefit in SCAD patients and can only be used for other compelling indications [1]. The affected subjects should be advised to avoid the common precipitating factors such as strenuous physical activities and those involving straining [24].

Percutaneous coronary intervention (PCI):

PCI in SCAD is challenging. It has a higher incidence of complications, such as vessel dissection and obstruction [37]. The outcomes are usually worse than cases of atherosclerosis [22]. Stent mal-apposition is not uncommon due to the healing of the dissection over time [38]. Therefore, this option should only be reserved for subjects with ongoing ischemic symptoms, ST-elevation myocardial infarction (STEMI), and hemodynamic instability [39].

Coronary artery bypass grafting (CABG):

Similar to PCI, CABG can be technically difficult in SCAD due to vessel fragility. The graft longevity is often limited due to the competitive flow after dissection healing [1]. Hence, it is only considered for patients with failed PCI and ongoing ischemia or SCAD cases with high-risk anatomy (e.g., left main coronary artery SCAD with infarction) [40]. Complex SCAD cases, such as those with cardiogenic shock, may need mechanical circulatory support (i.e., extracorporeal membrane oxygenation and intraaortic balloons) as a bridge to cardiac transplantation [41].

Conclusion:

SCAD is an underestimated cause of CAD. It usually presents as an ACS, especially in pregnant women and patients with systemic arteriopathy. Cardiologists should take special care in managing SCAD due to its diagnosis and treatment difficulties, as outlined in the article. Conservative medical treatment is a cornerstone, with PCI and CABG as options in cases of ongoing ischemia, hemodynamic instability, and high-risk anatomy.

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