

Case Report

Unexpected cause of NSTEMI in a 50-year-old male: Spontaneous Coronary-Artery Dissection (SCAD)

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Abstract

Spontaneous coronary-artery dissection (SCAD) has emerged as an important cause of myocardial infarction in young people. Although SCAD is uncommon, awareness of both the disease and its angiographic appearance has improved. Accurate and rapid diagnosis is paramount because the management of acute myocardial infarction caused by SCAD differs vastly from that of atherosclerotic myocardial infarction. The use of endocoronary imaging such as IVUS and optical coherence tomography is necessary in case of diagnostic doubt (especially in SCAD type 2 and 3). The optimal management of SCAD remains unclear. A conservative approach should be the preferred strategy. Here we report a case of a 50-year-old male patient who presented with chest pain for 7 days with the diagnosis of non-ST-segment elevation myocardial infarction (NSTEMI). Coronary angiography showed a type 1 SCAD of the obtuse marginal & right coronary artery. The patient was discharged under medical treatment.

Key words: Spontaneous Coronary Artery dissection, Non-ST Elevation Myocardial Infarction.

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Introduction

Spontaneous coronary artery dissection (SCAD) is defined as an epicardial coronary artery dissection that is not associated with atherosclerosis or trauma and not iatrogenic. The predominant mechanism of myocardial injury occurring because of SCAD is coronary artery obstruction caused by formation of an intramural hematoma (IMH) or intimal disruption rather than atherosclerotic plaque rupture or intraluminal thrombus. SCAD has emerged as an important cause of acute coronary syndrome, myocardial infarction, and sudden death, particularly among young women and individuals with few conventional atherosclerotic risk factors [1]. Recent studies show SCAD to be the cause of up to 1–4% of acute coronary syndrome (ACS) [2]. We report the case of a 50-year-old male patient with no major cardiovascular risk factor who presented as a case of NSTEMI & eventually diagnosed as SCAD. The patient was treated medically with favorable results.

Case report

A 50 year old normotensive, nondiabetic, nonsmoker male patient presented the complains of chest pain for last 7 days & shortness of breath for last 3 days. He was treated as NSTEMI with 6 doses of LMWH at outside hospital. On

examination his vital signs were stable, Lungs: bi -basal crepitation. GRACE SCORE: 132. His ECG (Figure 1) revealed ST depressions in infero-lateral leads (Leads II, III, AvF, V3-V6). The results of the transthoracic echocardiography showed hypokinesia of inferior, posterior, apical lateral & apical anterior wall with an ejection fraction of 40-45%. His troponin I was raised (1.43 ng/ml, normal range: up to 0.6 ng/ml). Other biochemical parameters & x-ray chest were normal.

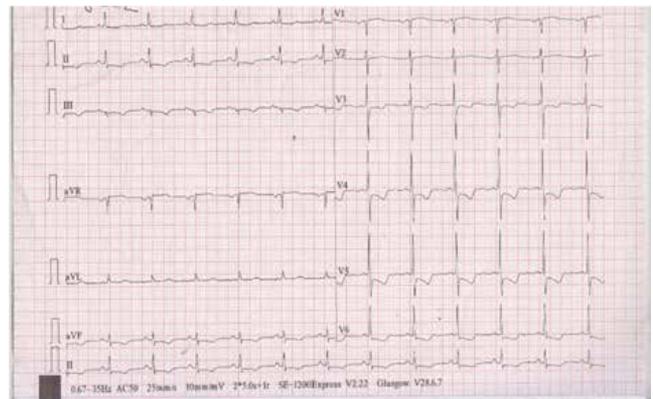


Figure 1: 12 lead surface ECG shows infero-lateral ischemia.

Coronary angiogram (Figure 2) was done on next day which revealed: left anterior descending artery (LAD): type IV vessel, normal with thrombolysis in myocardial infarction

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(TIMI) 3 flow; left circumflex artery (LCX): normal with TIMI 3 flow, obtuse marginal (OM)1: dissecting plaque in osteo-proximal segment; right coronary artery (RCA): TIMI 3 flow with dissecting plaque (red arrow) at mid segment. He was diagnosed as SCAD & treated with guideline directed medical therapy (antiplatelet drugs, statins, beta blockers, angiotensin receptor-neprilysin inhibitor & sodium-glucose cotransporter-2 inhibitors). Patient was discharged in stable condition & he was in favourable condition on 6 months follow up.

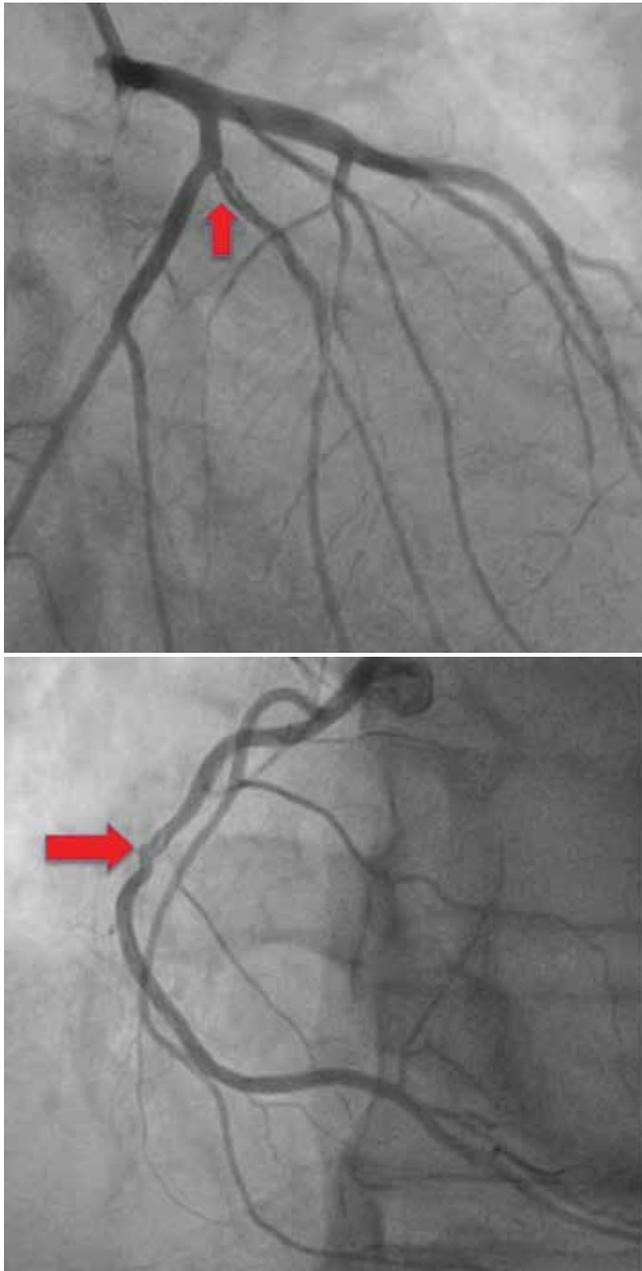


Figure 2: left panel still image of left coronary system shows non flow limiting dissecting plaque in osteo-proximal segment of first obtuse marginal branch of left circumflex artery (red arrow); right panel still image of right coronary system shows that dissecting plaque (red arrow) at mid segment.

Discussion

SCAD is defined as a separation of the layers of the epicardial coronary-artery wall by intramural hemorrhage, with or without an intimal tear. This condition is not associated with atherosclerosis, iatrogenic injury, or trauma [3]. SCAD accounts for up to 4% of all ACS, but the incidence is reported to be much higher (22-35% of ACS) in women <60 years of age, in pregnancy-related MI, and in patients with a history of fibromuscular dysplasia, anxiety, depression, or previous neuropsychiatric disorders [4,5]. The development of SCAD can be explained by two interrelated mechanisms [6,7]: ‘Inside-out’ phenomenon: blood extravasation from the intima to the media following a rupture of the intima (creating an intimal flap) accessible to an angiographic diagnosis. The ‘outside-in’ phenomenon: The vasa vasorum located at the level of the media ruptures, resulting in the occurrence of an isolated intramural hematoma without access to the arterial lumen (Figure 3). In both situations, the false channel, usually in the outer one third of the media, tends to extend and compress the true channel, leading to myocardial ischemia [1].

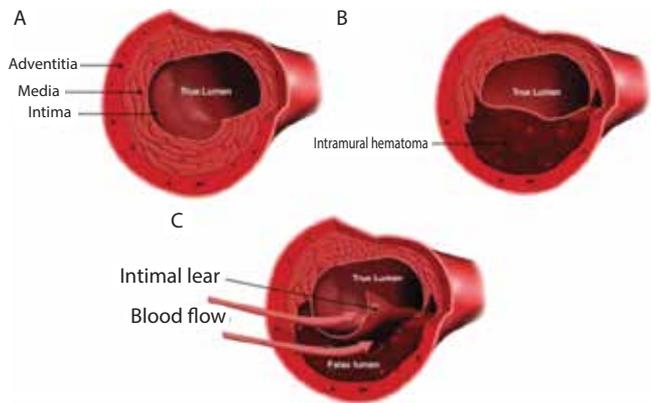


Figure 3: Cross-sectional views of the coronary artery [1]. (A) Normal coronary artery. (B) Coronary artery with intramural hematoma. (C) Coronary artery with intimal tear.

Spontaneous coronary artery dissection is characterized by the spontaneous formation of an intramural hematoma, which can lead to compression of the true lumen and myocardial infarction. An intimal tear may be present. The cause of SCAD is unknown but probably includes factors related to patient vulnerabilities and inciting triggers such as emotional stress, physical stress (e.g., from an extreme Valsalva maneuver, retching, vomiting, coughing, or isometric exercise), the use of stimulant medications or illicit drugs, and hormonal triggers (e.g., pregnancy) [8]. Meta-analyses of case series suggest that it presents as ST-elevation myocardial infarction in 48%, as non-ST-elevation myocardial infarction in 36%, and as unstable angina in 6.5%. Stable angina, congestive heart failure, and ventricular arrhythmia account for the remainder of presentations; 8–14% of cases present as life-threatening arrhythmia [9,10]. Coronary angiography remains the ‘first-line’ examination in case of suspected

ACS and is the gold standard for the diagnosis of SCAD. The classification of Saw et al. [1,3,8] describes angiographic signs which are widely adopted (Figure 4). In the Saw angiographic SCAD classification [8]:

- ❑ SCAD Type 1 (contrast dye staining of the arterial wall with multiple radiolucent lumen)
- ❑ SCAD Type 2 A (long diffuse and smooth narrowing)

with non-obstructive coronary arteries (stenosis <50%)

- ❑ SCAD Type 2 B (long diffuse and smooth narrowing) with severe coronary obstruction (>50)
- ❑ SCAD Type 3 (focal or tubular stenosis that mimics atherosclerosis)
- ❑ SCAD Type 4: as a complete occlusion of the vessel

Classification	Angiographic appearance	Anatomical appearance	Intravascular appearance on OCT
Normal coronary artery			
Type 1 SCAD			
Type 2A SCAD			
Type 2B SCAD			
Type 3 SCAD			
Type 4 SCAD			

Figure 4: Angiographic, anatomical, and intravascular appearance of SCAD [3,8]. Asterisks indicate a guidewire shadow artifact.

The use of endocoronary imaging such as optical coherence tomography and intravascular ultrasound is necessary in case of diagnostic doubt (especially in SCAD type 2 and 3) [11]. In addition to the management of acute myocardial

infarction in patients with SCAD, the treatment objectives are to manage chronic chest pain, prevent recurrence of SCAD, assess for and manage extra coronary vascular abnormalities, and improve quality of life. The optimal

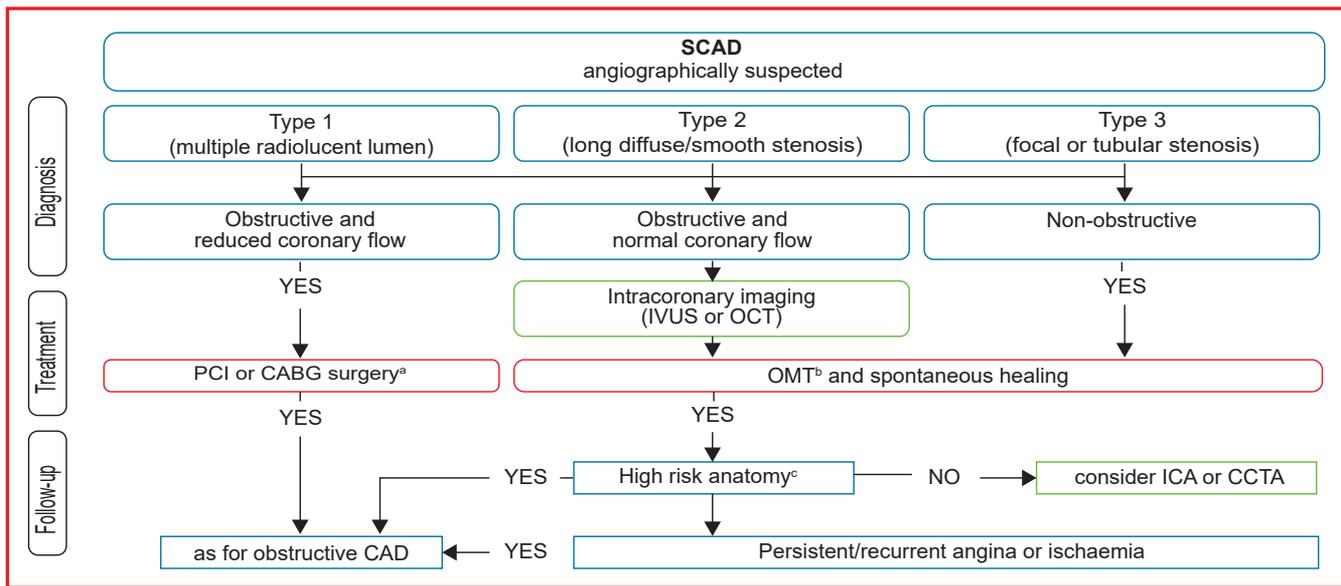


Figure 5: Diagnosis and treatment of patients with non-ST-segment elevation acute coronary syndrome related to SCAD [12]. Selection of revascularization strategy for high-risk anatomy according to local expertise, b Beta-blocker recommended while benefit of DAPT is questionable. c Left main or proximal left anterior descending or circumflex or right coronary artery, multivessel SCAD. CABG, coronary artery bypass grafting; CAD, coronary artery disease; CCTA, cardiac computed tomographic angiography; ICA, invasive coronary angiography; IVUS, intravascular ultrasound; OCT, optical coherence tomography; OMT, optimal medical therapy; PCI, percutaneous coronary intervention; SCAD, spontaneous coronary artery dissection.

management of SCAD is still unclear, since no RCTs have compared medical therapy to revascularization strategies. According to available data, with the exception of very high risk profile patients, a conservative approach should be the preferred strategy for SCAD management [12]. SCAD management has been taken from the recommendations of the 2020 European Society of Cardiology [12] (Figure 5).

Conclusion

Spontaneous coronary dissection, a particular form of ACS, is classified as myocardial infarction with non-obstructive coronary arteries (MINOCA) according to the latest recommendations and remains a challenge in terms of diagnosis and management. The diagnosis is initially angiographic and may require endocoronary imaging for greater accuracy. Conservative treatment (i.e. medical) remains the best option since in the majority of cases the evolution is favorable. Though SCAD is predominant with female, here we present a case of 50 years old male with NSTEMI who was eventually diagnosed as SCAD Type I (angiographically) & treated medically.

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