Case Report

A Case Report on Spontaneous Coronary Artery Dissection Managed by Percutaeneous Coronary Intervention (PCI)

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Abstract:

Background: Spontaneous coronary artery dissection (SCAD) is frequently underdiagnosed and often considered as a rare cause of acute coronary syndrome (ACS). Although it predominantly affects young women in the peripartum period, it can also occur in men. Most of the reported dissections have occurred in the left anterior descending coronary artery. The optimal treatment modalities are yet to be defined. Case Summary: A 50-year-old man who presented to us at National Institute of Cardio-Vascular Diseases (NICVD), Dhaka with an acute Non-ST-elevation myocardial infarction secondary to a spontaneous dissection of the Left anterior descending coronary artery. Due to ongoing chest pain, percutaneous coronary intervention (PCI) was done with drug eluting stent (DES) successfully, and the patient was discharged from the hospital on medical therapy. Conclusion: All clinicians should remain vigilant and aware of this condition, as patient outcomes and treatment guidelines differ substantially from conventional atherosclerotic ACS. Although initial conservative strategy is preferred strategy in SCAD management but timely intervention is warranted in selected cases.

Keywords: *Spontaneous coronary artery dissection, acute coronary syndrome, percutaneous coronary intervention.*

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Introduction:

SCAD is defined as a non-iatrogenic, non-atherosclerotic coronary artery dissection, resulting in formation of a false lumen or intramural haematoma in the coronary artery wall that compresses the true lumen, often compromising myocardial blood flow.[1] In early literature, the incidence of SCAD in acute coronary syndrome (ACS) was underestimated. Recent advances in awareness and widespread early angiographic investigation in ACS has led to important shifts in our understanding of the prevalence, predisposing causes, natural history, aetiology, clinical and angiographic features, management, and prognosis of SCAD.

Case Report: A 50-year-old hypertensive and smoker gentleman without any prior cardiac event presented to the emergency department with severe crushing retrosternal chest pain for 6 hours with accompanying sweating. He had no family history of heart disease. Physical examination revealed pulse 104 beats/min, blood pressure 150/90 mm Hg with no additional sound on auscultation of

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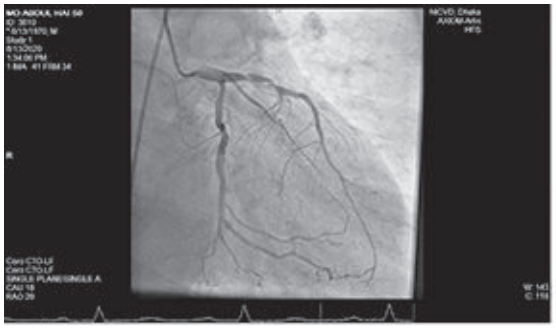
heart and lung bases. All the peripheral pulses were clearly palpable. His initial ECG showed ST depression in leads V1 to V4 and troponinI level was raised.

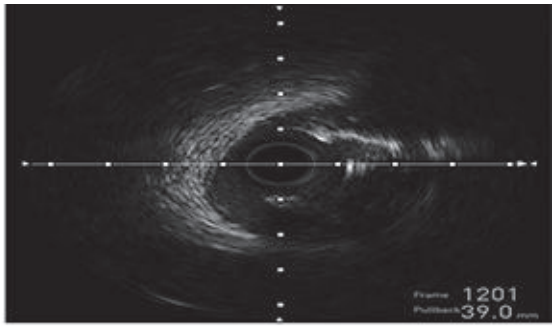
He received 300 mg aspirin and clopidogrel, intravenous morphine, nitrate, subcutaneous heparin, beta blocker, angiotensin converting enzyme inhibitor and statins

Despite of getting optimum medical management, patient was having ongoing chest pain. So, coronary angiogram was planned immediately which revealed a radiolucent linear defect in spiral fashion involving the proximal part of left anterior descending artery (LAD) suggestive of coronary artery dissection with thrombolysis in myocardial infarction II (TIMI II) flow (Figure: 1-A).

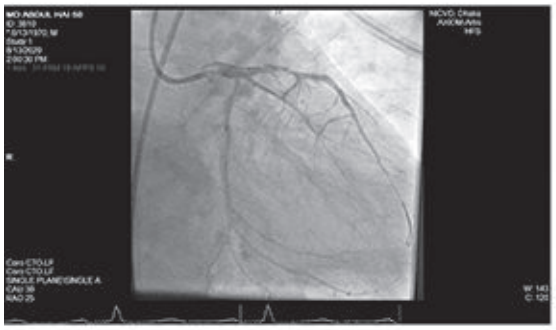
Due to ongoing symptom and compromised coronary flow PCI to LAD was planned. Extra back up coronary catheter (6 FR) was engaged in the left main coronary ostium. 0.014 inch floppy guidewire was passed across the lesion. Adequate precaution was taken to keep the guide wire in the true lumen. IVUS was done(Figure:1B) to differentiate SCAD from atherosclerotic plaque, and to demonstrate the extent of false lumen thrombosis. Pre-dilatation was done with 2.5×15 mm noncompliant PTCA balloon. 3×48 mm Everolimus-Eluting stent (Xience Xpedition) 3X48 mm was deployed in the target lesion with adequate coverage of the two edges. Post-dilatation was done with 3.5×15 mm noncompliant PTCA balloon. TIMI III was achieved immediately with resolution of chest pain .(Figure: 1C-F)



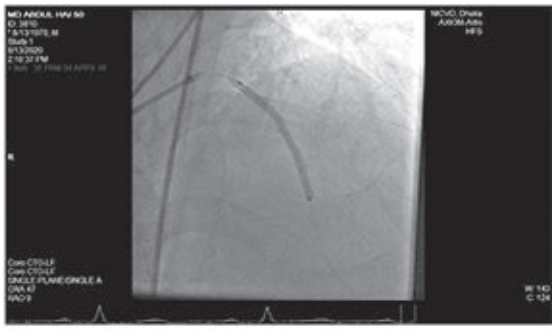
A) An spiral radiolucent linear defect in proximal LAD



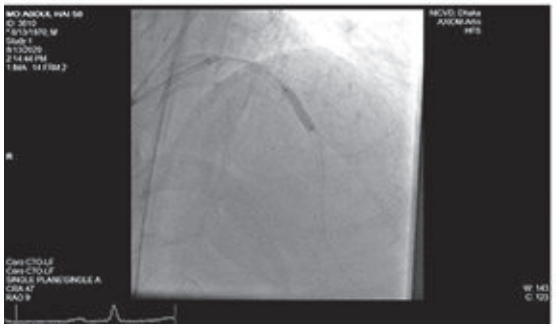
B) IVUS: Showing dissection flap with true & false lumen



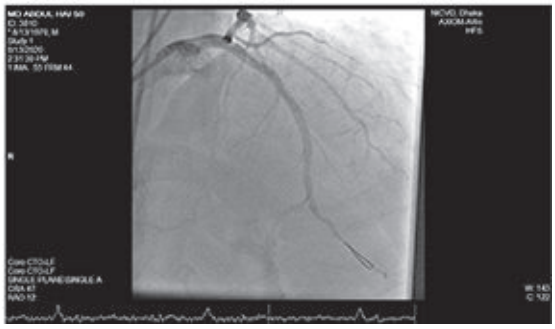
C) Predilatation with 2.5X15 mm SC balloon



D) Stenting with Xience Xpedition 3X48 mm



E) Postdilatation with 3.5X15 mm NC balloon



F) TIMI-III flow achieved

Fig.-1: *Steps of Percutaneous Coronary Intervention in Cath lab*

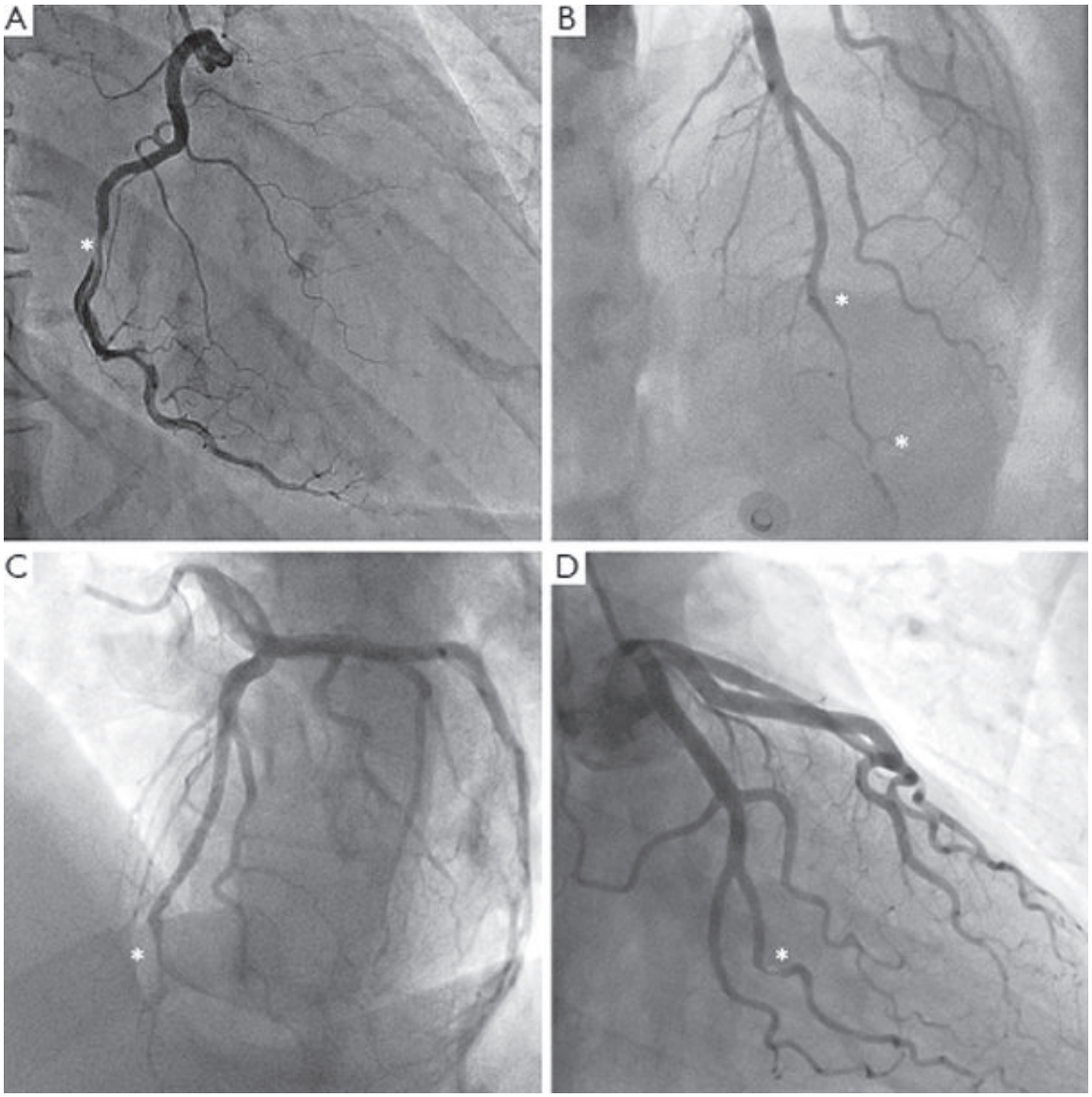


Fig.-2 : *Angiographic classification of SCAD. (A) shows Type 1 SCAD of the right coronary artery, characterised by a double lumen illustrated by contrast hold-up. (B) and (C) show Type 2 SCAD of the left anterior descending artery, which involves abrupt narrowing of the coronary artery with a diffuse tubular stenosis, either for a section of the artery in Type 2a (B), or to the distal end of the artery in Type 2b (C). (D) shows Type 3 SCAD of the second obtuse marginal branch of the left circumflex artery, mimicking atherosclerotic disease. In this case, SCAD was confirmed by optical coherence tomography. Asterisks denote the locations of dissection. SCAD, spontaneous coronary artery dissection*

Discussion:

Definition: SCAD is defined as a non-iatrogenic, non-atherosclerotic coronary artery dissection, resulting in formation of a false lumen or intramural haematoma in the coronary artery wall that compresses the true lumen, often compromising myocardial blood flow.[1]

Epidemiology: SCAD is an under-diagnosed entity and accounts for 1.7% to 4% of ACS cases.[2] It was first described in 1931.[3] The true prevalence of SCAD remains uncertain. Missed diagnoses are due to low suspicion of ACS in young women even in the presence of classic presenting symptoms, limitations of current

coronary angiographic techniques, and lack of clinician familiarity with the condition.[4] It predominantly affects young female patients with few or no traditional cardiovascular risk factors.

Pathophysiology: The pathophysiology of SCAD remains unknown.[5] It is likely that a combination of predisposing factors increase susceptibility such that a relatively minor trigger event is sufficient to precipitate SCAD. The vast majority of SCAD patients (90%) are women. The associated conditions are pregnancy, fibromuscular dysplasia, inflammatory conditions, connective tissue disorders.[5] The precipitants for SCAD are intense exercise, emotional stress, Valsalva, pregnancy, toxins.[5]

Clinical presentations: Although there are wide ranges of clinical presentations and severities of SCAD, patients who survive and present for initial evaluation almost universally experience ACS and increased levels of cardiac enzymes. Among available series of patients presenting for evaluation, 26% to 87% of patients with SCAD present with ST-segment–elevation MI, and 13% to 69% present with non–ST-segment–elevation MI.[9,10] Presenting symptoms are consistent with atherosclerotic ACS, with chest pain being the most prevalent (95.9%).[11] Ventricular arrhythmias or sudden cardiac death account for SCAD presentation in 3% to 11% of reported series.[9,10] Delayed diagnosis is common and SCAD should be actively considered in the differential diagnosis of ACS presentations in low risk patients.

Differential diagnosis: The differential diagnosis for SCAD includes atherosclerotic ACS, coronary artery spasm, Takotsubo cardiomyopathy, coronary thromboembolism, and myocardial infarction with non-obstructed coronary arteries (MINOCA).

Diagnostic modalities: There are no currently identified specific blood biomarkers for SCAD. Coronary angiography represents the principal tool for the diagnosis of SCAD in clinical practice. Intracoronary nitroglycerin should be given, where blood pressure allows, to ensure complete vasodilation and to rule out the possibility of associated coronary spasm. With experience most SCAD cases can be diagnosed on angiography alone, with intracoronary imaging reserved for cases where diagnostic uncertainty exists.[12] In the Saw angiographic SCAD classification, type 1 refers to the classic appearance of multiple radiolucent lumens or arterial wall contrast staining (Figure 2A).[8,13] Type 2 refers to the presence of diffuse stenosis that can be of varying severity and length (usually >20 mm; Figure 2B): Variant 2a is diffuse arterial narrowing bordered by normal segments proximal and distal to the IMH, and variant 2b is diffuse narrowing that extends to the distal tip of the artery(Figure 2C). [8, 13] Type 3 is focal or tubular stenosis, usually <20 mm in length, that mimics atherosclerosis (Figure 2D); intracoronary imaging is required to confirm the presence of intra mural haematoma and to diagnose SCAD. [8, 13]

Most SCAD can be diagnosed angiographically and in scenarios where a conservative approach to management is feasible, coronary instrumentation should, if possible, be avoided. However, where diagnostic uncertainty exists or to guide coronary intervention when required, careful intracoronary imaging can be invaluable and appears safe. Intravascular ultrasound (IVUS) and OCT provide tomographic images of the vessel wall and the coronary lumen that have proved to be of major value in the diagnosis of SCAD.[14, 15, 16] Intracoronary imaging can also help to guide decision-making on stent size. Although there are relative advantages to each technology, OCT is generally favored for SCAD imaging because of its higher spatial resolution.[14, 15, 16]

Management:

Acute management

Previously, the treatment of SCAD was largely extrapolated from management of atherosclerotic coronary disease, although this has now been brought into question. Although an early invasive strategy with revascularization is widely advocated in ACS secondary to atherosclerotic disease, there are no randomised data to support coronary revascularisation with percutaneous coronary intervention (PCI) in ACS caused by SCAD.

Recent studies show that the majority of SCAD will first stabilize and then heal completely over time if managed conservatively.[17,18] Revascularization in patients with SCAD is very challenging due to the presence of an underlying disrupted and friable coronary vessel wall. This is widely reported to lead to worse outcomes for PCI than in atherosclerotic coronary disease.[17,18] For this reason where revascularization is not mandated (i.e. in haemodynamically stable patients with maintained distal flow in the culprit coronary and without demonstrable ongoing ischaemia) a conservative strategy is generally favoured.[19, 20] The US Mayo Clinic series reported on 59 patients (from 95 managed conservatively) who underwent repeat angiography for a range of reasons a median of 2.4 years after the index event. In all, 73% (43/59) were described as 'healed'.[20]

Percutaneous coronary intervention

Published studies consistently show an increased risk of coronary complications with PCI.[20,11, 22] In the Canadian series, revascularization procedural success was only achieved in 64% of patients and, in addition to that, only 30% of patients maintained durable results at long term follow-up.[19] Where ongoing ischaemia or infarction mandates intervention, interventional cardiologists should be mindful of specific additional risks associated with SCAD interventions.

These include:

- Increased risk of secondary iatrogenic dissection
- Guidewire passage into the false lumen
- Proximal and/or distal false lumen propagation during stent deployment
- Persistent distal dissection
- Major side branch restriction or occlusion by propagation of haematoma

Where stents are deployed, second generation drug-eluting stents (DES) are advised. Significant rates of in-stent restenosis are reported. [5]

Coronary artery bypass grafting

Coronary artery bypass grafting (CABG) in SCAD is generally used as a bail-out strategy either for a failure of PCI with ongoing ischaemia or infarction of a significant at-risk myocardial territory (e.g. failure to wire the true lumen distal to a SCAD occlusion) or because the site and extent of the dissection (usually involving the left main stem or the presence of multiple dissections in different vessels) is felt to pose a prohibitive risk with either a conservative or a PCI strategy.[5]

Medical management

Thrombolysis: Thrombolysis is contraindicated for the acute management of SCAD.[5]

Antiplatelet therapies: The use of antiplatelet therapies and the duration of treatment remains an area of controversy in SCAD.[5] Patients who undergo stenting should receive dual antiplatelet therapy for 12 months and prolonged or lifelong monotherapy (usually with aspirin) in accordance with current ACS guidelines.[23] In patients managed conservatively, high grade stenosis sometimes are associated with true luminal thrombus in SCAD. This provides justification for antiplatelet therapy in the acute phase and most authors advocate acute dual antiplatelet therapy (usually with aspirin and clopidogrel rather than the newer P2Y12 inhibitors and avoiding intravenous antiplatelet therapies).[5, 23] The optimal duration of dual and subsequent monotherapy remains unknown with some authors advocating lifelong aspirin.[5]

Anticoagulant therapies: Anticoagulation should probably be limited to acute administration during revascularization procedures while chronic use should be restricted to situations where there is an unequivocal clinical indication (such as left ventricular thrombus or thromboembolism) which should over-ride what is at present a theoretical risk.[12]

Angiotensin converting enzyme inhibitors, angiotensin receptor antagonists, mineralocorticoid receptor antagonists, beta-blockers, and vasodilator therapies: Medical management of SCAD patients with significant impairment of left ventricular systolic function should follow current guidelines aimed at maximizing angiotensin converting enzyme inhibitors(ACEI) or angiotensin receptor blocker (ARB) and b-blocker doses and adding in a mineralocorticoid receptor antagonist (MRA) as indicated. [23,24]

Statins: The rationale for prescribing statins for a condition whose pathophysiology has no known association with cholesterol is unclear.[5]

Prognosis:

In patients surviving SCAD, long-term mortality is low. In the US Mayo Clinic series 10-year survival from Kaplan-Meier estimates is reported at 92%.[25] The overall major adverse cardiac events (MACE) rate in SCAD patients is significant but with considerable variation between published series. Recurrence in SCAD has been widely reported.[19, 20, 21,22, 25] The US series reported SCAD recurrence in 17% of patients across a median follow-up period of 47 months with a 10-year recurrence rate of 29.4% [the median time to a second event was 2.8 years (ranging from 3 days to 12 years)][6]

Conclusions:

SCAD is a challenging clinical entity that most commonly presents with ACS. Further research must be carried out to establish the ideal pharmacological and interventional management of SCAD, and its underlying predisposing factors, including the importance of genetics. At last, all the cardiologists should remain vigilant and aware of this condition, as patient outcomes and treatment guidelines differ substantially from conventional atherosclerotic ACS.

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