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Spontaneous Coronary Artery Dissection Triggered by Chronic Emotional Stress: A Case Report

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Abstract | **Case Report**

Spontaneous coronary artery dissection (SCAD) is a non-atherosclerotic cause of acute coronary syndrome that predominantly affects women, with emotional or physical stress recognized as one of its most consistent precipitating factors [1],[2]. We report a case of extensive SCAD, involving the left main coronary artery extending into the proximal-mid left anterior descending artery, in a 30-year-old man with a one-year history of widowhood and a background of tobacco smoking. He presented with three months of progressive exertional angina before diagnosis was confirmed on coronary angiography and intravascular ultrasound (IVUS). Given the absence of significant atherosclerotic risk factors beyond smoking, and the presence of sustained psychological distress following the loss of his spouse, chronic emotional stress is proposed as the most probable trigger in this case. This report discusses the pathophysiological link between emotional stress and SCAD, and argues for greater clinical attention to psychosocial history in young patients presenting with unexplained chest pain.

Keywords: Spontaneous coronary artery dissection, Acute coronary syndrome, Left main coronary artery, Young male, Emotional stress, Intravascular ultrasound.

INTRODUCTION

Spontaneous coronary artery dissection occurs when a tear develops within the coronary arterial wall, or when blood accumulates spontaneously between the wall's layers, creating a false lumen that compresses the true lumen and restricts blood flow. Unlike typical atherosclerotic coronary disease, SCAD is not caused by plaque buildup, and it tends to affect a different population, mostly women between 30 and 50 years old, often around the time of childbirth or in the setting of underlying arteriopathies such as fibromuscular dysplasia [5].

Among the recognized triggers for the acute dissection event, two stand out repeatedly in the literature: intense physical exertion and severe emotional stress [7]. The latter is thought to act through a surge of catecholamines that places abnormal shear stress on the coronary wall, in a manner mechanistically related to, but distinct from, stress-induced [takotsubo] cardiomyopathy [4].

Male SCAD is uncommon and, when it occurs, is more often traceable to one of these identifiable triggers rather than to hormonal or connective tissue factors [6]. We describe a young male patient whose only conventional cardiovascular risk factor was smoking, but who carried a significant and sustained psychosocial burden the loss of his wife one year before presentation which we believe to be the most plausible driver of his dissection.

CASE PRESENTATION

A 30-year-old man, widowed for one year, with a past medical history notable for chronic tobacco smoking, presented to the emergency department for evaluation of chest pain.

His symptoms had started three months earlier in the form of anginal chest pain provoked by physical effort. The pain was retrosternal, radiating to the interscapular region, and consistent with Canadian Cardiovascular Society [CCS] class II angina. It was reliably brought on by exertion and eased with analgesia. Notably, the pain was not relieved by lying supine, nither

improved by leaning forward, though in this patient it occurred against a background of exertional, effort-related pain more typical of an ischemic mechanism.

The psychosocial picture in this patient deserves emphasis rather than treatment as a passing detail: this was a young man, recently widowed, carrying that loss for a full year by the time his heart began giving him trouble. The chronic, sustained emotional strain, rather than a single acute shock, is increasingly recognized as capable of driving the same physiological stress response implicated in SCAD.

Physical examination on admission:

- Heart rate: 89 bpm
- Blood pressure: 130/79 mmHg
- Chest: normal contour
- Apex beat: palpable at the 5th intercostal space, normally located
- Cardiac auscultation: no murmurs
- No clinical signs of right heart failure
- Peripheral pulses : intact and symmetrical
- Neurological status: GCS 15/15, no focal deficits

Nothing on his physical exam pointed toward an acute catastrophe no murmur, no failure, no neurological compromise which is itself a useful teaching point, since SCAD can present with a deceptively unremarkable exam in the absence of an evolving myocardial infarction.

Investigations

Electrocardiogram (ECG):

Sinus rhythm, rate approximately 75 bpm, normal axis, normal PR interval. Flat T waves were present in the high lateral leads, as well as in leads III and aVF subtle repolarization changes rather than dramatic ST elevation, consistent with a relatively stable clinical course over three months rather than an acute occlusive event (Fig.1).

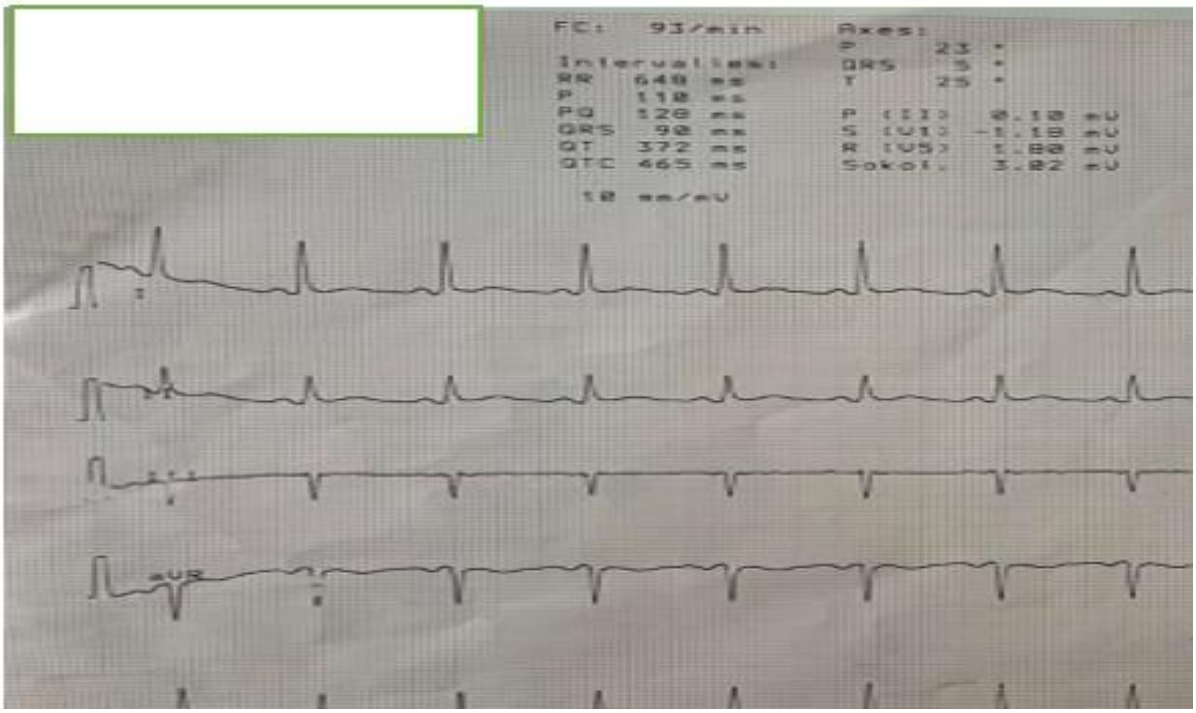

Fig. 1

Transthoracic echocardiography (TTE):

No regional wall motion abnormalities identified. Preserved Left ventricular ejection fraction at 60%, reassuring in that no established infarction or significant ischemic damage had yet occurred.

Exercise stress ECG:

The test was terminated early due to a positive result on both clinical grounds [reproduction of anginal symptoms] and electrocardiographic grounds [ischemic changes], confirming a genuine ischemic basis for his exertional chest pain.

Laboratory investigations:

Unremarkable, with the single exception of troponin, elevated at 226 ng/L, indicating some degree of myocardial injury in keeping with a partially compromised but not fully occluded coronary segment.

Coronary angiography:

Demonstrated a dissection originating in the left main coronary artery and extending into the proximal-to-mid segment of the left anterior descending artery a long and hemodynamically significant segment of disease, given the myocardial territory this vessel supplies.

Intravascular ultrasound (IVUS):

Confirmed the diagnosis, visualizing the intramural hematoma and false lumen characteristic of spontaneous coronary artery dissection, and excluding atherosclerotic plaque rupture as the underlying cause.

Management

The dissection involving the left main coronary artery and extending into the proximal-mid left anterior descending artery the decision was made to proceed with surgical revascularization rather than conservative medical management alone. This approach is consistent with current practice for SCAD involving the left main trunk, where the volume of myocardium at risk if the vessel were to occlude outweighs the general preference for conservative management seen in most other SCAD distributions.

The patient underwent coronary artery bypass grafting (CABG). The procedure was well tolerated, and his postoperative course was uncomplicated, with good clinical recovery and preservation of left ventricular systolic function. He was discharged in stable condition, with good clinical status and a preserved left ventricular ejection fraction.

Outcome and Follow-up

The patient was seen in the outpatient cardiology clinic one month after discharge for routine post-surgical follow-up. He reported doing well clinically, with no recurrence of chest pain and no new recurrent chet pain or cardiovascular symptoms. Repeat transthoracic echocardiography at this visit confirmed a left ventricular ejection fraction above 60%, indicating full preservation of systolic function with no evidence of interval deterioration.

This favorable early trajectory symptom resolution, uncomplicated surgical recovery, and normal ventricular function on follow-up imaging is a reassuring outcome for a dissection that had involved a high-risk anatomical segment, and supports surgical revascularization as an appropriate strategy in left main SCAD.

DISCUSSION

SCAD remains an uncommon cause of ACS in the general population, but is common proportion of young women who present with MI, up to 35% in women younger than 50 years [8]. The risk factors for SCAD include most common female sex, acute stressors, predispose patients to arteriopathy as well as hormonal and hemodynamic changes associated with pregnancy fibromuscular dysplasia, are thought to trigger dissection. [9]. The differential diagnosis to consider in ACS presentations similar to SCAD may be Takotsubo, The management modalities for SCAD such as conservative measurements, percutaneous coronary intervention (PCI), and CABG. Those patients that develop SCAD are expected to manifest a wide variety of symptoms during multiple out-patient clinic follow-up. Cardiovascular-related symptoms that occur include recurrent chest pain, anxiety, depression, recurrent myocardial infarctions, congestive heart failure, stroke, and recurrent SCAD [10].

The decision to proceed with CABG rather than conservative management reflects the general consensus that left main involvement is an exception to the usual conservative-first approach in SCAD, given the myocardial territory that would be jeopardized by occlusion of an unprotected left main segment. The patient's uncomplicated recovery and preserved ejection fraction at one-month follow-up support this as a reasonable and effective strategy in this specific anatomical scenario.

CONCLUSION

This case reinforces that SCAD should remain on the differential for young male patients with anginal symptoms even when traditional atherosclerotic risk factors are minimal. It further suggests that chronic emotional stress — here, a full year of unresolved grief following the loss of a spouse — may act as a slower, sustained trigger for coronary dissection, distinct from but mechanistically related to the acute emotional triggers more commonly described in the literature. Greater attention to psychosocial history in young cardiac patients may improve recognition of this important and likely underdiagnosed condition.

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