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Middle-Aged Male with NSTEMI Triggered by SCAD Following Prolonged Khat Chewing

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Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a rare, yet potentially life-threatening, condition involving the spontaneous tearing or separation of the coronary artery wall. This occurs in the absence of atherosclerosis, trauma, or iatrogenic causes. SCAD can lead to myocardial injury through coronary artery obstruction, often caused by intramural hematoma formation or intimal disruption.

While the exact etiology of SCAD remains unclear, it predominantly affects younger women, especially those in the peripartum period or with fibromuscular dysplasia. Other identified risk factors include extreme physical exertion, emotional stress, and certain substance use, including illicit drugs.

Khat

Khat (Catha edulis) is a shrub whose leaves contain active compounds, primarily cathinone and cathine. Cathinone, structurally similar to amphetamines, acts on the central nervous system to produce stimulant effects.

Khat use is particularly common in regions such as Yemen, where its consumption is a cultural norm, especially among men. The leaves are chewed for prolonged periods (often 2-4 hours) to induce stimulation, euphoria, increased alertness, and heightened energy, effects analogous to those of amphetamines. However, regular use is associated with an elevated heart rate, increased blood pressure, and decreased appetite.

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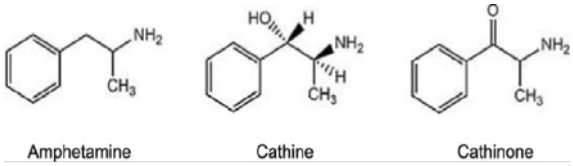


Case presentation

A 51-year-old male presented to the emergency department with complaints of chest pain and shortness of breath persisting for two days. The patient reported that the pain had initially been more severe but had since diminished. He attributed the onset of symptoms to an extended session of khat chewing, lasting over 10 hours. The patient had no significant past medical history, was not on any medications, and had no family history of cardiovascular disease.

Vital signs on admission

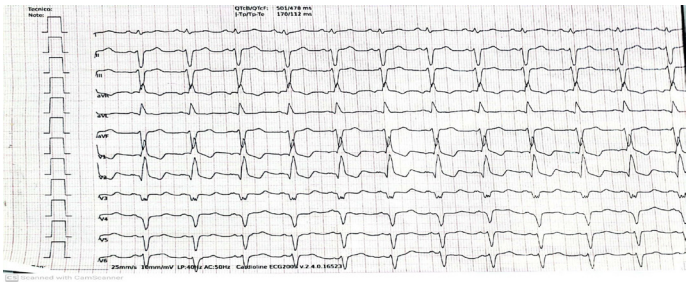
Blood pressure: 123/76 mmHg.

Heart rate: 112 bpm.

Oxygen saturation: 97% (room air).

Respiratory rate: 27 breaths per minute.

Initial Electrocardiography (ECG) revealed a Right Bundle Branch Block (RBBB). As no prior ECG was available for comparison, this finding was noted without immediate conclusion.



Despite the patient's distress and ongoing chest pain, physical examination revealed no abnormal heart sounds, murmurs, or respiratory findings. A chest X-ray was unremarkable.

Laboratory results

High-sensitivity Troponin I: 5 ng/mL (normal: < 0.039 ng/mL).

CK-MB: 149.2 ng/mL (normal: < 5 ng/mL).

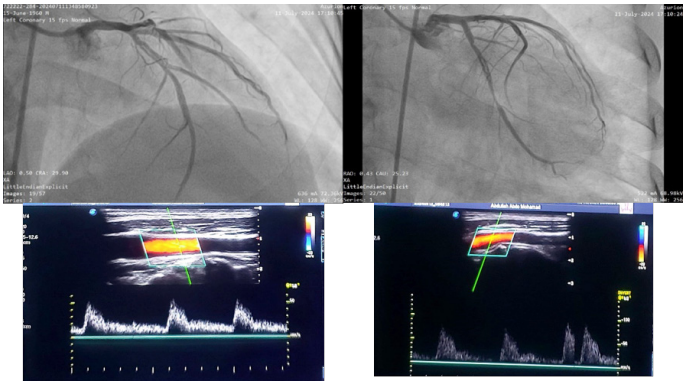
NT-proBNP: 700 pg/mL (elevated).

D-dimer: Normal.

Bedside echocardiography performed in the emergency department showed severe anterior wall hypokinesia, likely in the territory of the Left Anterior Descending artery (LAD), with a reduced left ventricular ejection fraction (LVEF) of 34%. There were no signs of valvular dysfunction or pericardial abnormalities.

The patient was diagnosed with Non-ST Elevation Myocardial Infarction (NSTEMI) and admitted to the Coronary Care Unit (CCU) with a Global Registry of Acute Coronary Events (GRACE) score of 104. Initial treatment included Low Molecular Weight Heparin (LMWH), Dual Antiplatelet Therapy (DAPT), atorvastatin, beta-blockers, nitroglycerin, and low-dose lisinopril.

On the second day of hospitalization, the patient showed clinical improvement. However, due to the persistence of RBBB-an indicator of increased risk in Acute Coronary Syndrome (ACS)-A Coronary Angiogram (CAG) was performed. The CAG demonstrated dissection in both the LAD and the first segment of the circumflex artery (LCX) with TIMI grade 2 flow in the LAD.



A systemic vascular screening, including the carotid and renal arteries, was normal.

Given the confirmed diagnosis of SCAD, conservative management was continued, and atorvastatin was discontinued. Over the following three days, the patient's condition steadily improved, with resolution of chest pain and normalization of CK-MB levels. Troponin I level also began to decline.

The patient was discharged after five days, symptom-free, and scheduled for follow-up in 6 to 8 weeks. At the four-week follow-up, the patient remained asymptomatic, with no chest pain or dyspnea. He was advised to maintain his current medication regimen and make lifestyle modifications, including avoiding khat. A repeat echocardiogram showed improved LVEF (53%) with mild residual anterior hypokinesia.

Conclusion

SCAD is a rare but significant cause of myocardial infarction, particularly in individuals without traditional cardiovascular risk factors. This case highlights the potential role of prolonged khat use in precipitating SCAD, especially in middle aged male —a demographic less commonly affected by this condition. A thorough history, including habits such as khat use, is essential in accurately diagnosing and managing SCAD. Further research is needed to clarify the long-term cardiovascular effects of khat and guide the development of localized health policies to mitigate its risks.

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