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Acute Myocardial Infarction Caused by Oral-Contraceptive – Related Spontaneous Coronary Artery Dissection: a Case Report

Background	Spontaneous coronary artery dissection (SCAD) is an increasingly recognized cause of acute myocardial infarction (AMI) in young women, with hormonal factors potentially playing a significant role.
Case summary	A 32-year-old woman taking combined oral contraceptives for four months presented with acute chest pain and ST-segment elevation. Coronary angiography revealed diffuse LAD narrowing that improved after intracoronary nitroglycerin. Follow-up optical coherence tomography at 6 weeks confirmed intramural hematoma consistent with SCAD. Conservative management was pursued. At one-year follow-up, the patient had recovered with normalized left ventricular function.
Conclusion	Clinicians should be highly suspicious of SCAD in young women with ACS or AMI, particularly those using oral contraceptives. Early identification with appropriate imaging can lead to favorable outcomes.
Keywords	Spontaneous coronary artery dissection; acute myocardial infarction; oral contraceptives; optical coherence tomography
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Introduction

Acute myocardial infarction (AMI) in young women without traditional cardiovascular risk factors is uncommon, accounting for less than 3% of all AMI cases [1]. Spontaneous coronary artery dissection (SCAD) has emerged as a significant non-atherosclerotic cause of AMI, disproportionately affecting women who account for more than 90% of SCAB cases [2].

SCAD is defined as spontaneous separation of the coronary arterial wall, creating a false lumen that can compromise coronary blood flow [3]. Several predisposing factors have been identified, including fibromuscular dysplasia, pregnancy, and hormonal factors [4]. Combined oral contraceptives (COCs) have been implicated in several SCAD cases, with a recent Danish cohort study demonstrating that COC use was associated with a 1.9-fold increased risk of myocardial infarction [5].

The diagnosis of SCAD has been revolutionized by optical coherence tomography (OCT), which offers superior resolution for visualizing vessel wall pathology [6]. Management of SCAD differs from atherosclerotic coronary disease, with conservative approach generally favored in stable patients [7].

Case Presentation

Patient information and clinical findings

A 32-year-old woman presented to the emergency department with acute-onset left-sided chest discomfort that began while working (Table 1). The initial episode was accompanied by neck tightness, left arm numbness, and brief loss of consciousness. She had no prior medical history, was a non-smoker, and did not consume alcohol.

Detailed history revealed the patient had been taking COCs (compound levonorgestrel/ethinyl-estradiol tablets) for four months before admission. She had no pre-

Table 1. Timeline of clinical events

Time	Event	Key Findings
Day –2	Initial symptoms	Chest pain, LOC; Normal ECG/biomarkers
Day 0	Hospital admission	Acute chest discomfort; STEMI; Troponin 0.036 ng/ml
Day 0	Emergency angiography	LAD 90–95% stenosis; Improved with NTG
Day 3	Vasovagal episode	Sinus arrest; BP 49/27 mmHg
Day 10	Discharge	Stable on DAPT + CCB
Week 6	Follow-up coronary angiography + OCT	LVEF 71%; OCT confirms SCAD
Month 12	Long-term follow-up	LVEF 62%; Asymptomatic

LOC, loss of consciousness; ECG, electrocardiogram; STEMI, ST-segment elevation myocardial infarction; LAD, left anterior descending artery; NTG, nitroglycerin; BP, blood pressure; DAPT, dual antiplatelet therapy; CCB, calcium channel blocker; OCT, optical coherence tomography; SCAD, spontaneous coronary artery dissection; LVEF, left ventricular ejection fraction.

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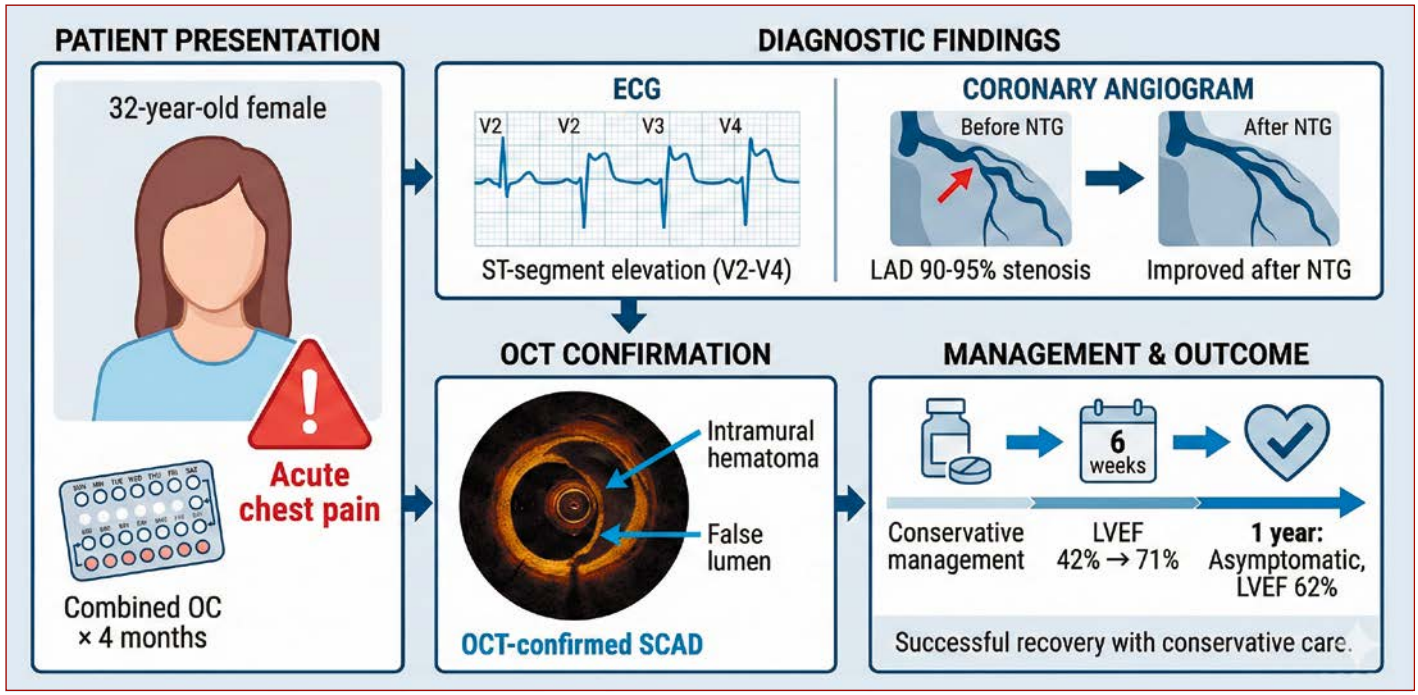
Central illustration. Spontaneous Coronary Artery Dissection (SCAD) Case Flow



vious pregnancies or births and had not used COCs prior to this four-month period. Family history revealed paternal hypertension.

Vital signs: blood pressure, 110 / 70 mm Hg; heart rate, 66 beats / min; respiratory rate, 20 breaths / min; oxygen saturation, 98% on room air. Physical examination revealed an anxious-appearing woman and no other significant findings.

Diagnostic assessment

Initial ECG showed sinus rhythm with hyperacute T waves in V_2-V_4. Ten minutes later, repeat ECG demonstrated marked ST-segment elevation in leads V_2-V_4, consistent with anterior STEMI (Figure 1). Cardiac troponin I was elevated at 0.036 ng/ml (reference <0.023 ng/ml). The patient was immediately administered a loading dose of aspirin (300 mg), ticagrelor (180 mg), and atorvastatin (40 mg).

Interventional management

Emergency coronary angiography revealed diffuse LAD narrowing with 90–95% stenosis and Thrombolysis In Myocardial Infarction (TIMI) flow grade II–III (Figure 2A, arrows indicate areas of narrowing). After intracoronary nitroglycerin (250 μg), significant improvement in LAD caliber occurred with restoration of TIMI grade III flow (Figure 2B, arrows show improved vessel diameter), suggesting coronary prior vasospasm.

Hospital course

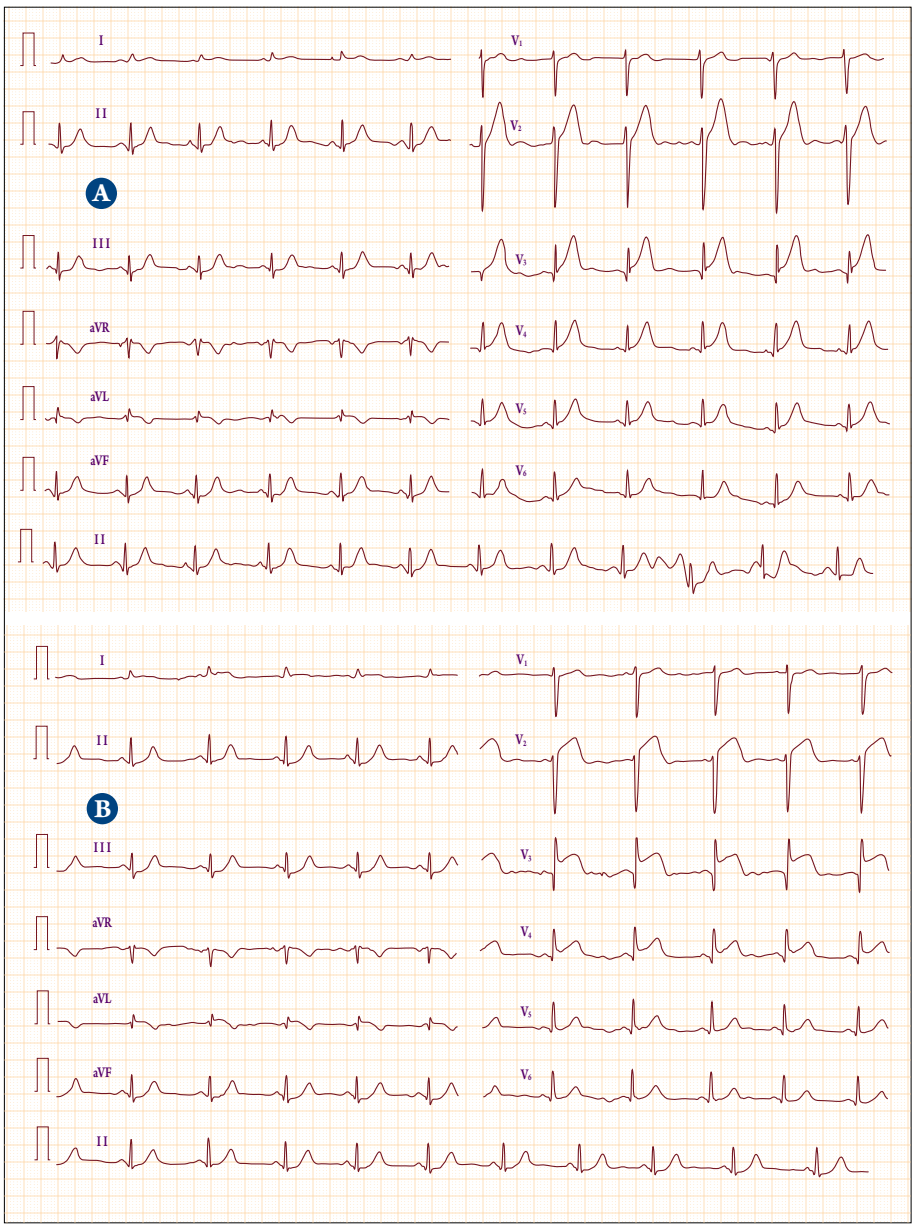
Post-procedure investigations revealed rising cardiac biomarkers with peak troponin I of 3.9 ng/ml, myoglobin >900 ng/ml, and NT-proBNP of 1460 pg/ml. Echocardiography showed anterior wall motion abnormality with left ventricular ejection fraction (LVEF) of 42%. The patient was managed with dual antiplatelet therapy (aspirin 100 mg daily, ticagrelor 90 mg twice daily), diltiazem 90 mg daily, and atorvastatin 40 mg daily.

Following a heavy meal on the third day of hospitalization, the patient experienced substernal discomfort followed by a staring episode with unresponsiveness. Her blood pressure dropped to 49/27 mmHg, and cardiac monitoring showed sinus arrest with junctional escape beats, with the longest R-R interval measuring 2.55 seconds. The ECG also showed bradycardia with heart rate of 35 beats/min prior to return of sinus rhythm.This event was attributed to a vasovagal reflex and resolved within 15 min after treatment with intravenous fluids and atropine. She was discharged after 10 days with clinical improvement. Discharge ECG showed resolution of ST-segment elevation with persistent T-wave inversion in leads V_2-V_4.

Follow-up investigations and management

Six weeks after discharge, the patient underwent scheduled follow-up examination and OCT. She remained asymptomatic with improved LVEF of 71%. Follow-up coronary angiography demonstrated significant improvement in LAD appearance compared to the initial presentation, with residual mild diffuse irregularity and approximately 30% stenosis in the mid-LAD segment. TIMI flow grade III was maintained. Subsequent OCT of the LAD revealed 30% stenosis without atherosclerotic plaque but with persistent intramural hematoma

Figure 1. Serial ECGs demonstrating evolution of ST-segment elevation myocardial infarction



Panel A. Initial ECG at 9:50 AM showing sinus rhythm with hyperacute T waves in leads V_2-V_4. **Panel B.** Follow-up ECG at 10:00 AM same day demonstrating marked ST-segment elevation in the same leads, consistent with AMI in the anterior wall.

(Figure 3), confirming SCAD diagnosis. The angiographic and OCT findings were consistent with healing SCAD with favorable vessel remodeling. Screening for fibromuscular dysplasia with computed tomography angiography of renal and carotid arteries showed no evidence of arteriopathy.

Continued medical management was recommended. The patient was managed with dual antiplatelet therapy (aspirin, 100 mg daily and clopidogrel, 75 mg daily), atorvastatin, 20 mg nightly, sacubitril/valsartan, 25 mg twice daily, and diltiazem, sustained release, 90 mg once daily. The patient was advised to permanently discontinue COCs.

At one-year follow-up, echocardiography showed LVEF 62% with mild anteroseptal thinning. Clopidogrel had been discontinued, at 6 months and she remained on aspirin, low-dose statin (10 mg), and diltiazem 90 mg daily. She remained off oral contraceptives and remains asymptomatic.

Discussion

This case illustrates an association between use of COCs and SCAD in a young woman without traditional cardiovascular risk factors. The combination of coronary vasospasm and arterial wall dissection exemplifies the complex pathophysiology of SCAD.

Multiple mechanisms were likely involved in the pathophysiology of this case. Estrogen promotes a hypercoagulable state through increased clotting factors and enhanced platelet aggregation [8]. Progesterone may impair arterial wall integrity by disrupting collagen synthesis [9]. Both hormones modulate vascular tone through endothelial function, with estrogen generally promoting vasodilation while progesterone may increase vascular reactivity [10].

This case underscores OCT's diagnostic value. While conventional angiography suggested SCAD through diffuse narrowing, the dramatic nitroglycerin response indicated significant vasospasm but did not exclude underlying pathology. OCT's high resolution (10–20 μm) enabled visualization of intramural hematoma, confirming the SCAD diagnosis [11, 12].

Management of SCAD differs from that of atherosclerotic disease. Conservative management is often preferred over percutaneous coronary intervention (PCI) due to technical challenges and potential complications including dissection propagation and wire passage into a false lumen [13]. Our conservative approach aligns with evidence suggesting favorable outcomes in stable SCAB patients with preserved coronary flow [14].

Several similar cases have been reported in the literature, providing important insights into the association between oral contraceptives and SCAD. Del Rio-Pertuz et al. reported a case of a young woman taking low-dose oral contraceptives who presented with non-ST segment elevation myocardial infarction due to SCAD with superimposed thrombosis, high-

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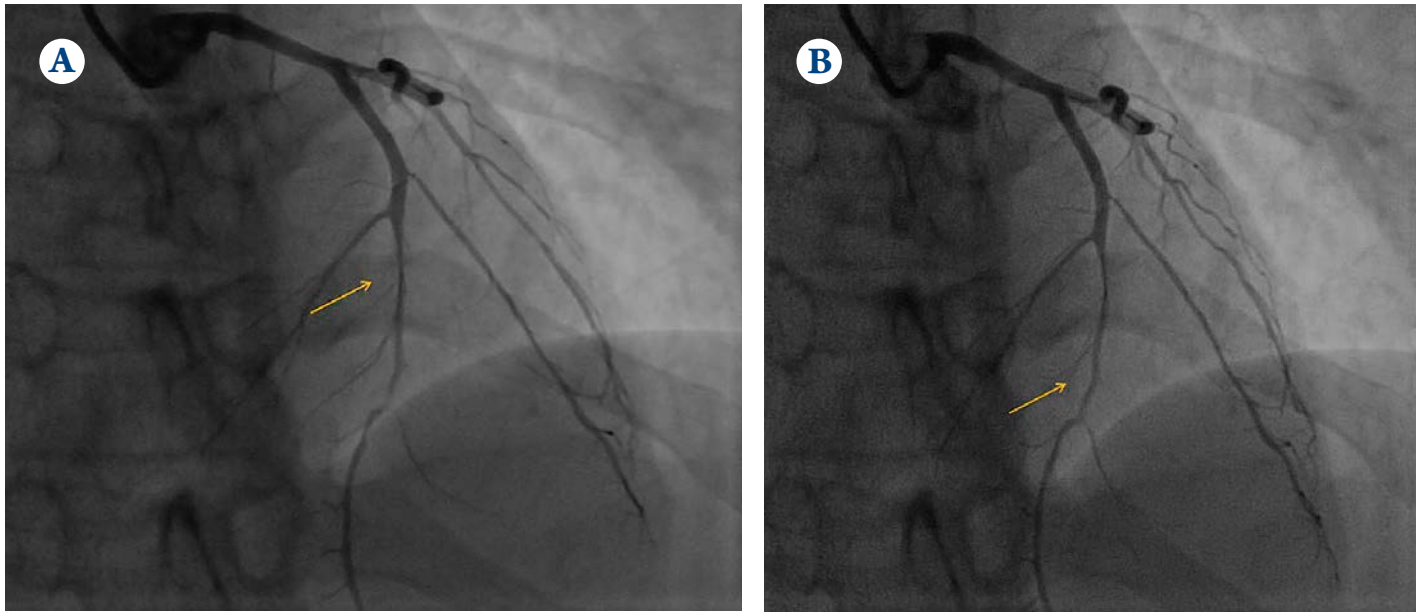
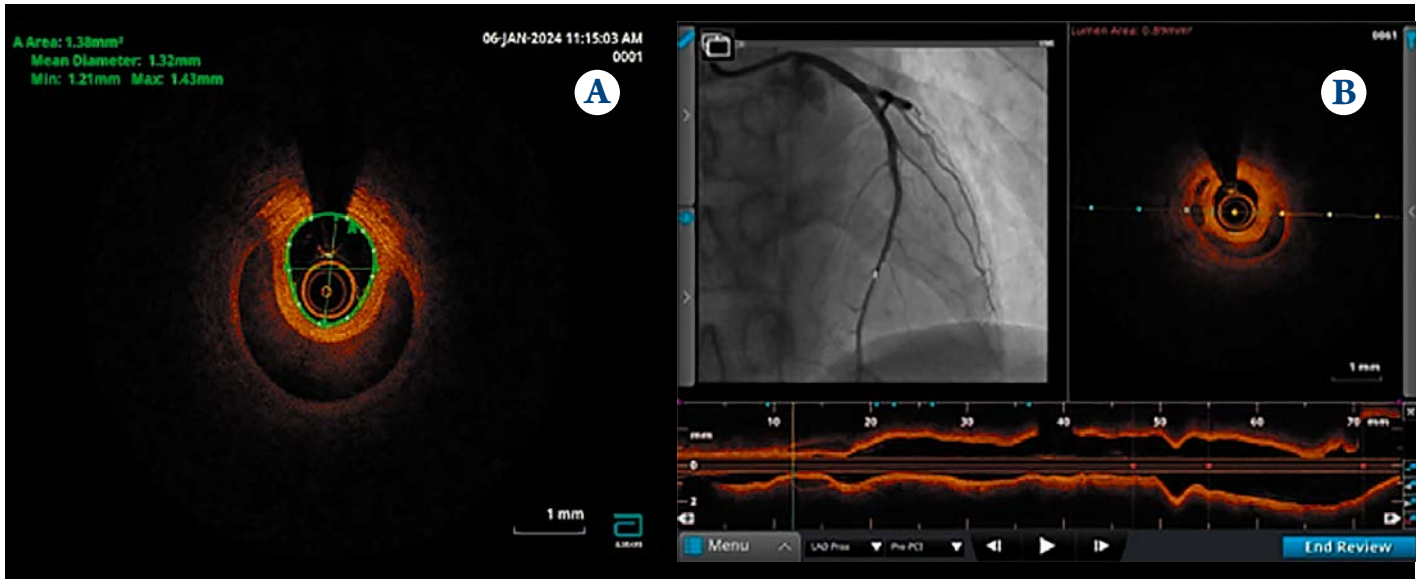
Figure 2. Coronary angiography before and after intracoronary nitroglycerin administration



(**A**) Initial coronary angiogram demonstrating diffuse narrowing of the left anterior descending (LAD) artery (arrows) with maximum stenosis of 90–95% and TIMI flow grade II–III. (**B**) Post-nitroglycerin angiogram showing significant improvement in LAD caliber (arrows) with restoration of TIMI grade III flow after 250 μg of intracoronary nitroglycerin.

Figure 3. Optical coherence tomography (OCT) findings at follow-up angiography



OCT images obtained 6 weeks after initial event demonstrating: (**A**) Cross-sectional view showing intramural hematoma with approximately 30% luminal stenosis. (**B**) Longitudinal reconstruction showing extent of intramural hematoma, confirming spontaneous coronary artery dissection without atherosclerotic plaque.

lighting the prothrombotic effects of hormonal contraceptives in the setting of SCAD [15]. Their case, similar to ours, demonstrated the challenges in diagnosis and the importance of advanced imaging modalities. Krishnan et al. provided comprehensive insights from histology and OCT in SCAD cases, demonstrating that OCT can identify intramural hematoma, intimal tear, and false lumen with superior resolution compared to conventional angiography [16]. Their review emphasized that OCT-guided diagnosis enables appropriate management decisions, particularly in distinguishing SCAD from atherosclerotic disease. A broader review of published cases found LAD involvement in most patients, with conservative management predominating and favorable outcomes when TIMI flow is preserved [15]. The recent Danish nationwide study provides robust epidemiological support, demonstrating dose-dependent relationships between estrogen content and cardiovascular risk, with combined oral contraceptives associated with a 1.9-fold increased risk of myocardial infarction [5].

Clinical implications include maintaining high suspicion for SCAD in young women who use hormonal contraceptives and present with acute coronary syndrome. Advanced imaging is vital for accurate diagnosis and for individualized management decisions. Hormonal contraceptives should be discontinued, and alternative, non-hormonal alternatives should be considered. Given the 10–20% risk of SCAB recurrence within five years [17], regular follow-up is warranted.

Conclusion

This case highlights the important association between use of COCs and SCAD in young women. OCT proved invaluable in establishing the diagnosis of SCAD and guiding conservative management, resulting in an excellent clinical outcome. Clinicians should be highly suspicious of SCAD in young women presenting with ACS or AMI, especially those using COCs.

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