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Spontaneous Coronary Artery Dissection Unmasking Occult Systemic Lupus Erythematosus in a Young Woman: The Importance of Systematic Screening – A Case Report

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Abstract **Case Report**

Background: Spontaneous Coronary Artery Dissection (SCAD) is the leading cause of acute coronary syndrome (ACS) in women under 50. While Systemic Lupus Erythematosus (SLE) is a recognized cause of vascular fragility, it is classically associated with overt mucocutaneous or musculoskeletal symptoms. SCAD occurring as the sole, inaugural manifestation of "silent" SLE represents a major diagnostic pitfall. ***Case Presentation:*** We report the case of a 36-year-old female, non-smoker with moderate obesity (BMI 31 kg/m²), admitted for acute retrosternal chest pain. Electrocardiogram showed anterior ischemic changes consistent with NSTEMI. Coronary angiography revealed a Type 1 SCAD of the Left Anterior Descending (LAD) artery. Despite the patient fitting the "typical" demographic profile for idiopathic SCAD, a systematic etiological workup was performed. Surprisingly, while the patient had no history of rash, photosensitivity, or arthralgia, immunological testing was strongly positive for Antinuclear Antibodies (ANA 1:640) and anti-dsDNA, confirming active SLE. ***Management:*** The patient was managed conservatively for the dissection (Beta-blockers, Aspirin) and initiated on specific immunosuppressive therapy (Corticosteroids, Hydroxychloroquine) to treat the underlying vascular inflammation. The outcome was favorable with no recurrence at 3 months. ***Conclusion:*** This case challenges the common practice of labeling SCAD as "idiopathic" in young women without obvious comorbidities. It supports the systematic screening for connective tissue diseases, as unmasking an occult Lupus drastically alters long-term management and prognosis.

Keywords: Spontaneous Coronary Artery Dissection (SCAD); Occult Systemic Lupus Erythematosus; Women's Heart Health; Systematic Screening; Case Report.

1. INTRODUCTION

Spontaneous Coronary Artery Dissection (SCAD) is defined as a non-traumatic, non-iatrogenic separation of the coronary arterial wall [1]. It has emerged as a significant cause of myocardial infarction in young women, accounting for up to 35% of ACS cases in women under 50 [2]. While the pathophysiology is often multifactorial (hormonal, stress-related, or associated with Fibromuscular Dysplasia), autoimmune etiologies remain a crucial differential diagnosis.

Systemic Lupus Erythematosus (SLE) typically presents with a constellation of clinical symptoms [3]. However, vascular inflammation can occasionally be the primary driver of the disease. We present a case of SCAD in a young woman where the cardiac event was the *only* indicator of an underlying, clinically silent Lupus. This case emphasizes that the "typical" demographic profile of a SCAD patient should not discourage clinicians from performing a complete immunological workup.

2. CASE PRESENTATION

Patient Information and Clinical Findings

A 36-year-old woman presented to the emergency department with sudden, intense, constrictive chest pain.

Her cardiovascular risk profile was notable for moderate obesity (Body Mass Index: 31 kg/m²). She was a non-smoker and had no history of hypertension,

diabetes, or dyslipidemia. She reported no recent emotional or physical stress.

Review of systems was negative: she specifically denied joint pain, skin rashes, hair loss, oral ulcers, or Raynaud's phenomenon.

On admission, the physical examination was unremarkable (Blood Pressure 115/70 mmHg, Heart Rate 88 bpm). There was no malar rash and no synovitis.

Diagnostic Assessment

The 12-lead Electrocardiogram (ECG) showed normal sinus rhythm with T-wave inversion in anterior leads (V1-V5).

Transthoracic Echocardiography demonstrated hypokinesis of the anterior wall with preserved Left Ventricular Ejection Fraction (LVEF 60%).

Laboratory analysis revealed a significant myocardial injury with High-sensitivity Troponin I elevated at 50 times the upper limit of normal. Kidney function and blood count were normal.

Coronary Angiography

The angiogram revealed a Type 1 Spontaneous Coronary Artery Dissection of the mid-Left Anterior Descending (LAD) artery, characterized by a radiolucent intimal flap and contrast staining of the arterial wall. The other coronary arteries were smooth and angiographically normal [4].

Etiological Investigation

Given the diagnosis of SCAD, a systematic search for secondary causes was initiated despite the absence of systemic symptoms.

- **Fibromuscular Dysplasia (FMD) screening:** Renal and iliac arteries were normal.
- **Autoimmune screening:** Unexpectedly positive.
  - **Antinuclear Antibodies (ANA):** Positive at **1:640**.
  - **Specific Antibodies:** Positive Anti-dsDNA and **Anti-Sm**.
  - **Complement:** Low C3 and C4 levels (indicating active consumption).

The diagnosis of Systemic Lupus Erythematosus (SLE) was confirmed. The SCAD was attributed to lupus-related vascular wall inflammation.

Therapeutic Management

- **Coronary Strategy:** Conservative management (no stenting) was chosen due to hemodynamic stability and TIMI-3 flow. Treatment included Bisoprolol 2.5mg and Aspirin 75mg.
- **Systemic Strategy:** Prompt initiation of Prednisone (0.5 mg/kg/day) and Hydroxychloroquine (200mg BID) to control the autoimmune flare.
- **Outcome:** The patient remained chest pain-free. At 3-month follow-up, she was asymptomatic, and inflammatory markers had normalized.

3. DISCUSSION

3.1 The Diagnostic Trap: Overlooking Autoimmunity in "Typical" SCAD

Because this patient fit the "typical" SCAD demographic (young female, few risk factors), the risk of labeling the condition as "idiopathic" or hormonal was high [1, 2]. Our case illustrates the danger of this confirmation bias. The patient had no external stigmata of SLE. Without proactive immunological screening, her condition would have been misdiagnosed. This supports the stance of Garcia *et al.,* who advocate that SCAD should be considered a potential vascular marker of occult connective tissue disorders [5].

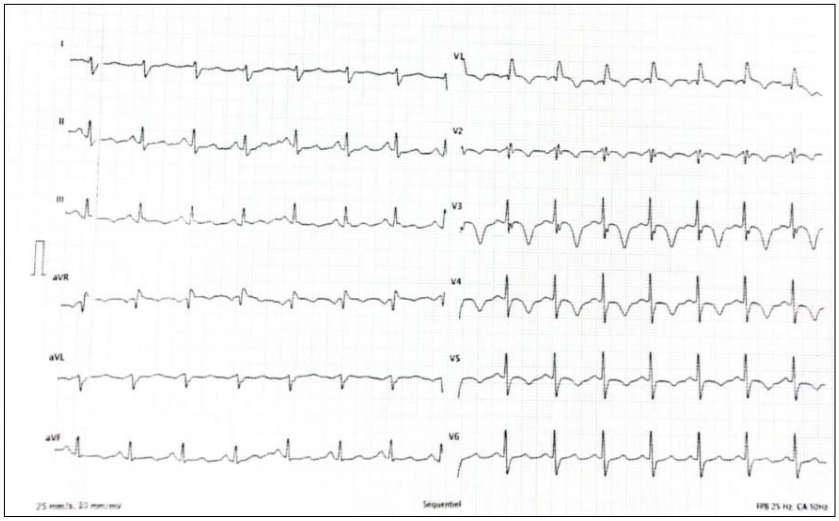

Figure 1: 12-lead Electrocardiogram on admission showing sinus rhythm with T-wave inversion in anterior leads (V1–V5), consistent with anterior ischemia

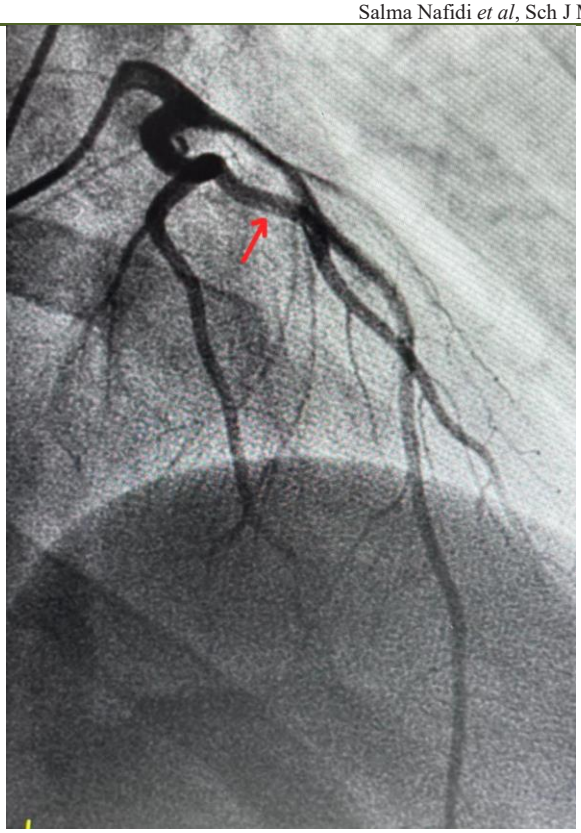

Figure 2: Coronary Angiography (RAO cranial view) demonstrating a Type 1 Spontaneous Coronary Artery Dissection of the mid-Left Anterior Descending artery. The red arrow points to the radiolucent intimal flap separating the true and false lumens

3.2 Pathophysiological Mechanisms

The link between SLE and SCAD differs fundamentally from atherosclerosis. In SLE, the vascular injury is driven by systemic inflammation. Two mechanisms are described:

1. **Vasa Vasorum Vasculitis:** Immune complexes target the microvessels in the adventitia, causing intramural hemorrhage that compresses the true lumen [6].
2. **Structural Degradation:** Pro-inflammatory cytokines (TNF-α, IFN-α) degrade collagen and elastin fibers [7]. This creates a "locus minoris resistentiae" (weak point), rendering the vessel susceptible to dissection under normal shear stress [8, 9].

3.3 Therapeutic Implications

Managing SCAD in the context of SLE requires a dual strategy.

- **Avoiding Revascularization:** In line with AHA guidelines, we avoided Percutaneous Coronary Intervention (PCI), which carries a high failure rate (up to 53%) in friable vessels [1, 10, 11]. Thrombolysis was also strictly avoided as it can expand the intramural hematoma [12].
- **Targeting the Root Cause:** Beta-blockers alone are insufficient if the driver is vasculitis. Controlling SLE activity with immunosuppressants is critical to prevent recurrence [13].

3.4 Recommendation for Systematic Screening

Based on this case and similar reports [14], we propose that a basic immunological panel (ANA, CRP) should be part of the routine workup for any patient presenting with SCAD, regardless of the presence of systemic symptoms.

4. CONCLUSION

In young women with SCAD, the absence of extra-cardiac symptoms does not rule out connective tissue diseases. This case advocates for a low threshold to perform immunological screening in all SCAD patients. Diagnosing "Silent" Lupus allows for targeted immunosuppressive therapy, transforming the prognosis of these patients.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest: The authors declare that they have no competing interests.

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Authors' Contributions: All authors contributed equally to the manuscript and approved the final submitted version.

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