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Case Report

Recurrent Myocardial Infarction Due to Spontaneous Coronary Artery Dissection in a Young Female with Rheumatoid Arthritis: A Case Report

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Abstract

Spontaneous coronary artery dissection (SCAD) is an uncommon but significant cause of myocardial infarction (MI) in young women who do not present with traditional cardiovascular risk factors. This case report details the presentation of a 36-year-old female with recurrent myocardial infarctions, characterized by chest pain, ECG abnormalities, and angiographic evidence of coronary dissection. Concurrently diagnosed with rheumatoid arthritis (RA), her repeated cardiac events were attributed to SCAD. The chronic inflammatory state associated with RA, along with autoimmune vasculitis, connective tissue abnormalities, and possible effects of RA medications, likely contributed to the development of SCAD. This case highlights the critical need to recognize SCAD as a potential diagnosis in young women, particularly those with autoimmune conditions, and underscores the importance of considering autoimmune disorders as a contributing factor in the pathogenesis of SCAD.

Keywords: Spontaneous Coronary Artery Dissection, Myocardial Infarction, Rheumatoid Arthritis, Coronary Angiography

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INTRODUCTION

Acute myocardial infarction (MI) is commonly associated with coronary artery disease (CAD). However, in an increasing number of younger female patients who lack traditional cardiovascular risk factors such as hypertension, diabetes, or hyperlipidemia, a less commonly recognized condition, spontaneous coronary artery dissection (SCAD), may be the underlying cause. The American Heart Association (AHA) defines SCAD as a non-atherosclerotic disruption of the coronary artery intima that occurs spontaneously due to weakening or tearing of the arterial wall. This disruption allows blood to seep into the vessel wall, creating an intramural hematoma that compromises myocardial blood supply, leading to ischemia and infarction [1,2].

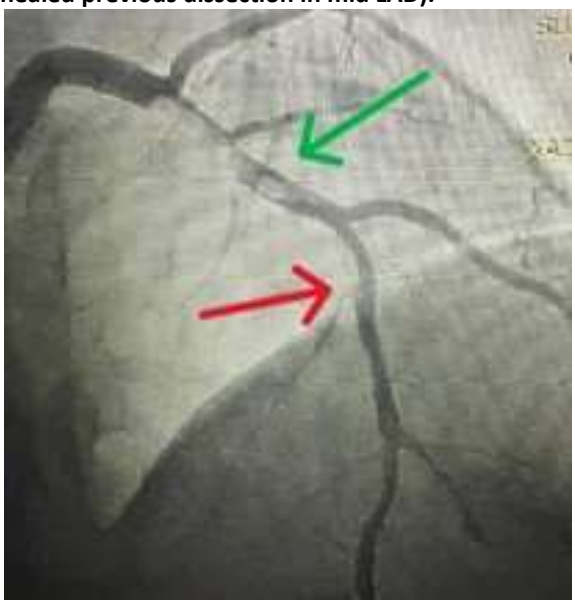
The precise etiology of SCAD remains uncertain, but suspected contributing factors include underlying connective tissue disorders such as fibromuscular dysplasia, hormonal changes associated with pregnancy or perimenopause, and significant physical or emotional stressors [3]. SCAD has emerged as a leading cause of MI in women under 50 years of age, accounting for up to 4% of MI cases with angiographic evidence of the condition [4]. Clinically, SCAD presents with symptoms similar to those of typical MI, including chest pain, dyspnea, and ECG abnormalities, making a high index of suspicion essential for accurate and timely diagnosis and appropriate management.

CASE PRESENTATION

Patient Information: A 36-year-old female presented to the emergency room with acute onset of severe chest pain radiating to her left arm, accompanied by profuse sweating and multiple episodes of vomiting over the past four hours. She had experienced a previous episode of acute coronary syndrome (ACS). On detailed inquiry, she reported a thirteen-year history of morning pain, redness, and swelling of the wrists and small joints of the hands, which was relieved by activity but not associated with mouth ulcers, skin rashes, or photosensitivity.

Clinical Findings: On general physical examination, the patient was mildly obese with a puffy and plethoric face, hirsutism, and pink abdominal striae. Vital signs were within normal limits. Systemic examination revealed wasting of the thenar and hypothenar muscles, with swelling and tenderness in both wrists and proximal interphalangeal joints of the hands, accompanied by restricted movements. There were no additional abnormalities detected during the examination.

Figure 1: Angiogram (Green arrow showing Grade 4 thrombus in proximal LAD, Red arrow showing healed previous dissection in mid LAD).



Timeline

- **Initial Presentation:** The patient experienced her first episode of ACS, which led to an urgent coronary angiography revealing a thrombus-laden left anterior descending artery (LAD) causing critical mid-course disease. A 2-D echocardiogram showed an ejection fraction (EF) of 35%, ischemic heart disease with moderate left ventricular (LV) systolic dysfunction, Grade I diastolic dysfunction, mild mitral regurgitation, and an organized apical clot.

- **Second Episode:** Approximately five months later, the patient presented with a second episode of ACS. Repeat angiography revealed subtotal occlusion in the proximal part of the LAD with a heavy thrombus extending into the left main stem. Thrombolysis in myocardial infarction (TIMI) grade II partial perfusion was noted, with a fallen EF of 35% and LV apical clot on echocardiography. Surprisingly, the previous angiographic findings in the mid LAD had healed completely, prompting a working diagnosis of Unstable Anginal Pain (USAP) or Non-ST

Elevation Myocardial Infarction (NSTEMI) secondary to Spontaneous Coronary Artery Dissection (SCAD).

- **Diagnostic Clarification:** A comprehensive immune profile revealed a raised ANA titer, Rheumatoid Factor (RF), and Anti-CCP Antibody, leading to a diagnosis of rheumatoid arthritis. Carotid, renal, and lower limb Doppler studies were normal, ruling out fibromuscular dysplasia. Late Gadolinium Enhancement (LGE) images and conventional coronary angiography showed dramatic resolution of previous findings.

Diagnostic Assessment: Initial diagnostic assessment began with an ECG, which was indicative of acute coronary syndrome (ACS). Bedside echocardiography revealed regional wall motion abnormalities (RWMA). Coronary angiography conducted during the first episode of ACS showed a thrombus-laden left anterior descending artery (LAD) with critical disease in the mid-course. On the second episode, angiography demonstrated subtotal occlusion of the proximal LAD, with a significant thrombus and TIMI grade II partial perfusion. A 2-D echocardiogram identified an LV apical clot and a reduced ejection fraction (EF). The immune profile tests revealed a positive antinuclear antibody (ANA) titer of 1:160, elevated rheumatoid factor (RF) of 99.3 IU/ml, and anti-cyclic citrullinated peptide (Anti-CCP) antibody greater than 500 U/ml, suggesting rheumatoid arthritis. Other autoimmune markers, including those for systemic lupus erythematosus (SLE), scleroderma, and myositis, were negative. Doppler studies of carotid, renal, and lower limb arteries were normal, effectively ruling out fibromuscular dysplasia. Late Gadolinium Enhancement (LGE) imaging further showed the presence of the LV apical clot and subendocardial to transmural myocardial enhancement.

Therapeutic Intervention

- **Initial Management:** During the first ACS episode, the patient was stabilized with angiography, which alleviated chest pain. She was started on tirofiban, an antiplatelet medication, and discharged on a regimen including dual antiplatelet therapy (DAPT), rivaroxaban as an anticoagulant, and guideline-directed medical therapy (GDMT) including beta-blockers, mineralocorticoid receptor antagonists, angiotensin receptor neprilysin inhibitors, and sodium glucose cotransporter-2 inhibitors for a duration of four weeks.
- **Management After Second Episode:** Upon diagnosing SCAD, the patient was anticoagulated during her hospital stay and discharged on DAPT, anticoagulants, GDMT, and immune modulators such as methotrexate with folic acid for her rheumatoid arthritis. Steroids were avoided due to their potential risk in exacerbating dissection.

Figure 2: 2D Echo showing Left Ventricular apical clot.

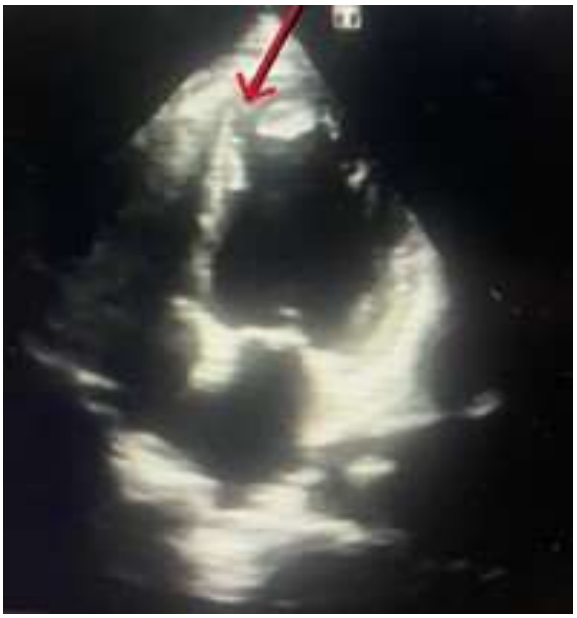


Figure 3: Angiogram Showing SCAD.

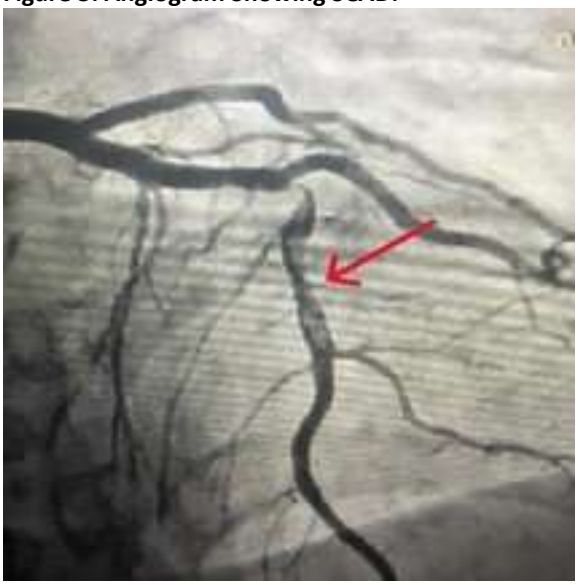
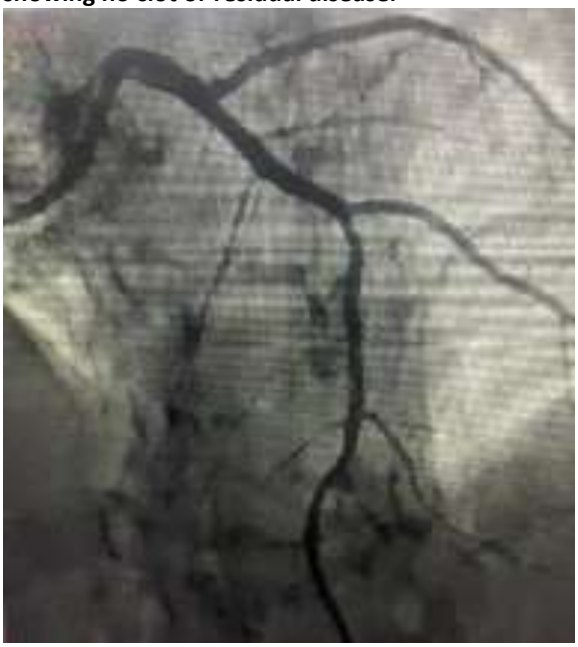


Figure 4: Angiogram after anticoagulation follow up showing no clot or residual disease.



Follow-up and Outcomes

- **Initial Follow-up:** Two months after the first episode, echocardiography showed improvement in EF to 50% and resolution of the LV apical clot.
- **Post-Second Episode Follow-up:** The EF dropped back to 35%, with reappearance of the LV apical clot and subtotal occlusion of the LAD observed on angiography. However, by the time of the third angiography, all previous findings had resolved dramatically, leading to the discontinuation of further invasive diagnostic procedures.

The patient was discharged with continued medical therapy and regular follow-up, showing stable symptoms and ongoing management of her condition.

DISCUSSION

Spontaneous Coronary Artery Dissection (SCAD) is a rare but significant cause of acute myocardial infarction (MI), particularly affecting young women. SCAD typically arises from either intramural hematoma formation due to spontaneous rupture of the vasa vasorum or intimal tearing, which creates a false lumen that disrupts or impedes myocardial blood flow, leading to myocardial injury [5]. Unlike other causes of acute coronary syndrome (ACS), SCAD is not associated with atherosclerosis or traditional cardiovascular risk factors. It commonly presents in otherwise low-risk individuals, such as women under 50, and accounts for approximately 0.1% to 4% of ACS cases and 35% of MI cases. Additionally, SCAD is a leading cause of pregnancy-associated MI [6,7].

Factors contributing to SCAD include multifocal fibromuscular dysplasia, connective tissue disorders, systemic inflammatory diseases, and hormonal changes, which can weaken the arterial wall [3]. Emotional and physical stressors, such as recurrent Valsalva maneuvers and certain drug use, may trigger SCAD, though in many cases, no specific trigger is identified. The prognosis for SCAD varies; as demonstrated by this patient, severe symptoms can improve significantly with appropriate medical management, underscoring the importance of timely diagnosis, effective treatment, and ongoing monitoring.

Diagnostic and investigative strategies for SCAD involve several modalities. Coronary angiography is essential for revealing the extent and location of dissection, guiding treatment decisions. Optical coherence tomography (OCT) and intravascular ultrasound (IVUS) offer detailed visualization of the arterial wall, helping to differentiate SCAD from atherosclerosis by identifying multiple radiolucent lumens and patterns of luminal narrowing or stenosis [8]. Echocardiography is useful for assessing cardiac function, detecting complications such as left ventricular thrombus, and monitoring therapeutic responses. Laboratory tests, including inflammatory markers, autoimmune profiles, and connective tissue disease markers, aid in identifying underlying conditions contributing to SCAD.

In this patient, the clinical presentation initially suggested myocardial infarction but was complicated by recurrent episodes, raising concerns for other potential diagnoses. Differential diagnoses considered included atherosclerotic coronary artery disease (CAD), Takotsubo cardiomyopathy, coronary artery embolism, Prinzmetal's angina, and myocarditis [9]. These conditions were ruled out based on the absence of typical atherosclerotic plaques and stenotic patterns on angiography, lack of apical ballooning on echocardiography, evolving dissection patterns, and normal inflammatory markers except for an elevated ESR [5,10].

Additionally, it is plausible that chronic inflammatory states, autoimmune vasculitis, connective tissue abnormalities, and potential medication effects secondary to rheumatoid arthritis contributed to the development of SCAD in this patient.

Management of SCAD generally involves conservative medical therapy, lifestyle modifications, and occasionally percutaneous interventional procedures (PCI). Initial treatment typically includes a combination of dual antiplatelet therapy (DAPT), anticoagulants, and guideline-directed medical therapy (GDMT) to reduce myocardial oxygen demand, prevent thrombus formation, and improve heart failure prognosis, particularly in patients without flow-limiting dissections [11]. As per the American Heart Association (AHA) 2021 Revascularization Recommendations, PCI or coronary artery bypass grafting (CABG) may be considered for patients refractory to conservative management, who have persistent ischemia or hemodynamic instability [2]. However, PCI is often associated with poor outcomes due to the risk of iatrogenic dissection, particularly in the presence of increased vascular fragility, worsening intramural hematoma, and potential stent malapposition, especially since SCAD frequently occurs in distal coronary segments that are challenging to treat with intervention [2,5] .

In this patient, methotrexate, a non-biological Disease-Modifying Antirheumatic Drug (DMARD), was initiated as the first-line treatment for rheumatoid arthritis according to American College of Rheumatology guidelines. Folic acid was also prescribed to mitigate methotrexate toxicity and support folic acid rescue therapy [12].

CONCLUSION

This case underscores the complexities involved in diagnosing and managing Spontaneous Coronary Artery Dissection (SCAD), especially in the context of underlying autoimmune conditions. The patient's presentation and evolving clinical course highlight the importance of a comprehensive, multidisciplinary approach that integrates cardiology, rheumatology, and vigilant patient monitoring. Timely and coordinated care is essential for optimizing patient outcomes, effectively managing symptoms, and preventing recurrence. This case exemplifies the need for ongoing research and awareness to better understand SCAD and improve treatment strategies for patients with similar challenges.

AUTHORS' CONTRIBUTION

UF, AHA, SS, IF, and SA: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. UF, AHA, SS, IF, and SA: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

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