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| RESEARCH ARTICLE

Spontaneous Coronary Artery Dissection in a Young Female: A Rare Cardiac Catastrophe Without Traditional Risk Factors

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| ABSTRACT

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome, predominantly affecting young women without traditional cardiovascular risk factors. We report a 32-year-old female who presented with sudden-onset, severe retrosternal chest pain radiating to the left arm, associated with diaphoresis and mild dyspnea. She had no history of hypertension, diabetes, dyslipidemia, smoking, or family history of premature coronary artery disease. Initial evaluation revealed subtle ST-segment changes on electrocardiogram and modestly elevated high-sensitivity troponin I levels. Coronary angiography demonstrated a type 2 SCAD of the mid-left anterior descending artery, confirmed by intracoronary imaging showing an intramural hematoma with preserved distal flow. Laboratory workup, echocardiography, and computed tomography angiography for extracoronary vascular assessment were unremarkable. Given hemodynamic stability and maintained coronary perfusion, the patient was managed conservatively with dual antiplatelet therapy, beta-blockers, and close monitoring, with plans for follow-up imaging to assess vascular healing. This case underscores the importance of considering SCAD in young women presenting with acute chest pain, highlights the critical role of coronary imaging in diagnosis, and demonstrates that tailored conservative management can lead to favorable outcomes while avoiding unnecessary interventions.

| KEYWORDS

Spontaneous coronary artery dissection, SCAD, acute coronary syndrome, young female, intramural hematoma, conservative management, coronary angiography, non-atherosclerotic coronary disease, cardiac emergency, chest pain

| ARTICLE INFORMATION

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Introduction

Spontaneous coronary artery dissection (SCAD) represents a distinctive and non-atherosclerotic etiology of acute coronary syndrome, characterized pathologically by a spontaneous tear or hemorrhage within the coronary arterial wall, resulting in the formation of an intramural hematoma and a false lumen that compromises coronary perfusion and obstructs blood flow [1]. First described in 1931 in a middle-aged woman who suffered sudden cardiac death [2], SCAD has since been recognized as a significant and increasingly appreciated cause of myocardial infarction among young and otherwise healthy individuals—predominantly women—who lack the conventional risk factors associated with atherosclerotic coronary artery disease [2,3]. In contrast to plaque rupture, which arises from disruption of lipid-rich atherosclerotic lesions, SCAD involves either an intimal tear or rupture of the vasa vasorum leading to hemorrhage within the vessel wall and subsequent dissection [1]. This unique mechanism sets SCAD apart from traditional coronary artery disease, as the event typically occurs in angiographically normal vessels and in patients without a history of hypertension, hyperlipidemia, diabetes mellitus, or tobacco use [1,4]. Clinically, SCAD presents with the classic features of acute myocardial infarction—namely chest pain, electrocardiographic changes, and biomarker elevation—yet the underlying pathology and patient demographic are notably distinct [2,4]. The precise pathophysiology remains an active subject of investigation, and two principal theories have been proposed: the "inside-out" hypothesis, wherein an intimal disruption permits luminal blood to penetrate the arterial wall, and the "outside-in" hypothesis, which posits that spontaneous rupture of the vasa vasorum produces an intramural hematoma that secondarily compromises the true lumen [1]. Increasing evidence from advanced imaging modalities supports the latter mechanism, suggesting that primary intramural bleeding may represent the initiating event in most cases [1]. Regardless of the initiating factor, the pathologic consequence is an expanding hematoma within the vessel wall that compresses the true lumen, precipitating myocardial ischemia or infarction. Predisposing conditions appear multifactorial, involving both hormonal and structural influences; estrogen-mediated alterations in vascular tone and remodeling may underlie the marked female predominance and the strong association with pregnancy [1]. Indeed, pregnancy-associated SCAD (P-SCAD) is frequently severe, and SCAD constitutes one of the leading causes of pregnancy-related myocardial infarction [1]. Another crucial predisposing substrate is fibromuscular dysplasia (FMD), a non-inflammatory arteriopathy affecting medium-sized arteries, with numerous studies reporting a very high prevalence—often exceeding 80%—of FMD or other vascular abnormalities among SCAD patients [1]. Conversely, inherited connective-tissue disorders such as Ehlers–Danlos or Marfan syndromes are only rarely encountered [1], and the majority of cases remain idiopathic apart from these associations. Additional proposed triggers include intense emotional or physical stress and exposure to sympathomimetic agents, though in many patients no precipitating factor can be identified. Epidemiologically, SCAD remains an uncommon contributor to the overall spectrum of acute coronary syndromes, accounting for approximately 1–5% of all cases [1]. Nevertheless, its relative burden is disproportionately high among young women, representing an estimated 20–35% of myocardial infarctions in women under 50 years of age [1]. In large clinical series, 80–95% of patients are female, with a median age in the mid-40s [1]. Thus, clinicians evaluating a younger or middle-aged woman presenting with acute coronary

symptoms in the absence of traditional risk factors should maintain a high index of suspicion for SCAD. Notably, while many patients describe recent postpartum status, emotional upheaval, or minor exertion preceding the event, up to half have no recognizable trigger [1]. A detailed clinical history and comprehensive vascular assessment, including evaluation for FMD, are therefore essential. Definitive diagnosis relies upon coronary imaging, with invasive coronary angiography remaining the gold standard; typical angiographic appearances include multiple lumens or long, smooth, tubular stenoses that differ from irregular atherosclerotic lesions [1]. The Saw classification (types 1–3) provides a systematic framework for interpreting angiographic findings, with type 1 lesions demonstrating contrast staining of a false lumen, type 2 exhibiting diffuse narrowing, and type 3 mimicking focal atherosclerosis [1]. When angiography is inconclusive, intravascular imaging techniques such as intravascular ultrasound (IVUS) or optical coherence tomography (OCT) can directly visualize the intramural hematoma and intimal flap, confirming the diagnosis [1]. Noninvasive modalities such as coronary CT angiography or cardiac MRI may be useful adjuncts for follow-up, although their spatial resolution is inferior to catheter-based methods. Importantly, therapeutic management of SCAD differs substantially from that of atherosclerotic myocardial infarction. Because the majority of SCAD lesions undergo spontaneous healing within weeks, a conservative medical strategy is preferred whenever feasible [1]. Registry data indicate that over 85% of patients can be successfully managed without revascularization, demonstrating complete or near-complete vessel healing on follow-up imaging [1]. Percutaneous coronary intervention (PCI) is often technically challenging and fraught with risk, as guidewires or stents may propagate the dissection or create additional intimal injuries, and optimal stent sizing is difficult in vessels distorted by hematoma [1]. Accordingly, PCI is reserved for patients with persistent ischemia, hemodynamic compromise, or involvement of the left main coronary artery. When revascularization is necessary, specialized approaches such as cautious low-pressure stenting or cutting-balloon fenestration under IVUS/OCT guidance are employed to minimize procedural complications [1]. In anatomically unsuitable cases, urgent coronary artery bypass grafting may be required. Medical therapy typically aligns with acute coronary syndrome principles, with aspirin and beta-blockers forming the mainstay, although the ideal duration of antiplatelet therapy and the role of statins remain areas of uncertainty [1]. Recurrent SCAD represents a significant concern, with observational studies reporting recurrence rates of several percent per year [1]. Owing to its rarity and unpredictable course, management strategies are largely empirical, and affected patients frequently experience substantial psychological distress related to fear of recurrence. The medical literature contains numerous illustrative case reports that underscore the heterogeneity of SCAD presentations. Paraskevaidis et al. described SCAD in a middle-aged individual undergoing unrelated surgery, emphasizing its status as a rare yet critical cause of acute ischemia, most often seen in young or peripartum women [3]. Other authors have reported cases occurring in women without pregnancy or typical precipitating factors, highlighting the disorder's unpredictable nature. Collectively, these reports reinforce that SCAD commonly affects otherwise healthy women without conventional cardiovascular risk factors and that its diagnosis should not be excluded on the basis of a benign risk profile [3]. The present case aligns with this paradigm: our patient, a young woman with acute myocardial infarction symptoms and angiographic evidence of coronary dissection, exhibited no identifiable trigger—no recent pregnancy, connective-tissue disorder, vasculopathy, or family history of similar disease. Such idiopathic cases, although uncommon and sparsely documented, illustrate the potential for SCAD to occur spontaneously in individuals without discernible risk factors. By detailing this case, we aim to emphasize that SCAD can present as a truly idiopathic and catastrophic event, underscoring the necessity of maintaining a high level of clinical suspicion for SCAD in all patients presenting with acute coronary syndrome, irrespective of their conventional cardiovascular risk profile.

Case Presentation

Patient's history and Physical Examination

This case describes a 34-year-old previously healthy female who presented to the emergency department with an acute onset of severe, central chest pain radiating to her left arm and jaw, which began abruptly while she was performing light household chores approximately two hours prior to hospital admission. The pain was described as crushing, pressure-like, and persistent, rated 9 out of 10 in intensity, and was associated with marked diaphoresis, nausea, and a profound sense of impending doom. She denied any recent physical trauma, emotional stress, or strenuous exertion preceding the event. There was no history of hypertension, diabetes mellitus, dyslipidemia, or smoking, and she reported no prior cardiac disease or use of hormonal contraceptives, illicit drugs, or stimulant medications. Her menstrual cycles were regular, and she had delivered one child six years earlier without peripartum complications. Family history was negative for premature coronary artery disease, connective tissue disorders, or sudden cardiac death. On arrival, the patient appeared acutely ill, anxious, and pale, clutching her chest in distress. She was afebrile with a blood pressure of 92/58 mmHg, heart rate of 118 beats per minute, respiratory rate of 20 breaths per minute, and oxygen saturation of 96% on ambient air. Cardiovascular examination revealed a rapid but regular rhythm, normal heart sounds without murmurs, rubs, or gallops, and no jugular venous distension. Peripheral pulses were palpable, symmetrical, and without delay. The lungs were clear on auscultation, and the abdominal and neurological examinations were unremarkable. Her skin was cool and clammy, and mild pallor was noted. The intensity and persistence of chest pain in a young woman devoid of conventional cardiovascular risk factors prompted diagnostic uncertainty and a broad differential, including acute coronary syndrome, spontaneous coronary artery dissection, pulmonary embolism, and aortic

pathology. The striking incongruity between her age, risk profile, and the severity of presentation underscored the need for immediate cardiac evaluation and advanced imaging to exclude rare but life-threatening causes of acute myocardial ischemia.

Investigations

Initial investigations were undertaken with high clinical suspicion for an acute coronary event. The 12-lead electrocardiogram (ECG) obtained on arrival demonstrated 2 mm of ST-segment elevation in the anterior precordial leads (V2–V4) with subtle reciprocal depression in the inferior leads, consistent with an evolving anterior wall myocardial infarction. Bedside cardiac monitoring revealed sinus tachycardia without arrhythmias. Serum high-sensitivity troponin I levels were markedly elevated at 1,425 ng/L (reference <14 ng/L) on admission and rose to 5,680 ng/L within six hours, confirming ongoing myocardial injury. Other laboratory parameters, including complete blood count, electrolytes, renal and liver function tests, coagulation profile, and lipid panel, were within normal limits. Pregnancy test and toxicology screen were negative, excluding hormonal or drug-related etiologies. A portable chest X-ray demonstrated no mediastinal widening or pulmonary congestion, and D-dimer assay was normal, making aortic dissection and pulmonary embolism less likely. Transthoracic echocardiography revealed mild left ventricular systolic dysfunction with a regional wall motion abnormality involving the mid-to-apical anterior and septal segments, without pericardial effusion or valvular pathology. Given her age, absence of risk factors, and the acute coronary pattern, she underwent urgent coronary angiography via radial access after initial stabilization with oxygen, low-dose aspirin, and unfractionated heparin. The angiogram revealed a long, smooth, tapering, non-atherosclerotic narrowing extending from the mid-left anterior descending artery to its distal segment, accompanied by a radiolucent intimal flap and contrast staining within the arterial wall—findings highly suggestive of a type 2 spontaneous coronary artery dissection (SCAD). No thrombotic occlusion or atheromatous plaque rupture was visualized in any coronary territory. Intravascular optical coherence tomography (OCT) confirmed the presence of an intramural hematoma compressing the true lumen without evidence of calcified plaque, consolidating the diagnosis of SCAD. Based on these imaging features, and in the absence of hemodynamic instability, the patient was managed conservatively with close monitoring, dual antiplatelet therapy, and beta-blockade, avoiding percutaneous intervention to reduce the risk of iatrogenic vessel extension.

Management course

The patient was immediately placed on continuous cardiac monitoring and administered supplemental oxygen at 4 liters per minute via nasal cannula to maintain oxygen saturation above 96%. Intravenous access was secured with two large-bore cannulas, and gentle crystalloid resuscitation was initiated with 500 milliliters of isotonic normal saline over one hour to correct relative hypotension while avoiding excessive preload that could exacerbate coronary wall stress. Analgesia was provided using titrated intravenous morphine in 2 mg increments to achieve comfort while maintaining hemodynamic stability. Antiplatelet therapy was initiated with oral aspirin 300 mg chewed, followed by a maintenance dose of 75 mg daily. Given the angiographically confirmed spontaneous coronary artery dissection and absence of significant atherosclerotic disease, a conservative management strategy was pursued in accordance with current expert consensus to minimize procedural risk. Dual antiplatelet therapy was continued with clopidogrel 75 mg once daily (loading dose 300 mg), and anticoagulation was withheld after diagnostic angiography to prevent propagation of the intramural hematoma. Beta-blockade was commenced with intravenous metoprolol 5 mg administered slowly over five minutes, repeated once after 10 minutes, then transitioned to oral metoprolol tartrate 25 mg twice daily to reduce heart rate and shear stress across the dissected coronary segment. Blood pressure control was reinforced with oral perindopril 2 mg daily to optimize afterload reduction. Serial cardiac enzymes were monitored every six hours, showing a gradual downtrend, and continuous telemetry revealed no arrhythmias. Echocardiography was repeated after 48 hours, demonstrating stable left ventricular function with no extension of wall motion abnormalities. The patient remained hemodynamically stable, chest pain subsided within 24 hours, and there were no recurrent ischemic episodes. A cardiothoracic consultation confirmed that revascularization was not indicated given preserved distal flow and lack of ongoing ischemia. She was transferred to the coronary care unit for close observation, with activity restriction and counseling to avoid emotional or physical exertion. Lipid-lowering therapy with rosuvastatin 10 mg nightly was added to promote endothelial recovery, and low-dose nitrates were prescribed as needed for residual discomfort. The patient's hospital course remained uncomplicated. She was discharged on hospital day six with prescriptions for aspirin 75 mg daily, clopidogrel 75 mg daily for six months, metoprolol 25 mg twice daily, perindopril 2 mg daily, and rosuvastatin 10 mg nightly. Detailed education was provided regarding avoidance of high-intensity exercise, recognition of recurrent symptoms, and adherence to follow-up imaging. Outpatient cardiac magnetic resonance imaging was scheduled for three months to reassess myocardial recovery and confirm vessel healing, with cardiology follow-up arranged at two and six weeks post-discharge.

Discussion

Spontaneous coronary artery dissection (SCAD) represents an infrequent but increasingly recognized cause of acute coronary syndrome (ACS), predominantly affecting young to middle-aged females without traditional atherosclerotic risk factors [1,2].

Unlike typical plaque rupture, SCAD involves the formation of an intramural hematoma or intimal tear, which leads to dynamic coronary lumen obstruction, often resulting in myocardial ischemia or infarction [3]. The predilection for women, particularly in the peripartum period, suggests a complex interplay of hormonal, structural, and genetic factors, with estrogen-mediated changes in vascular connective tissue and perivascular inflammation hypothesized to contribute to arterial fragility [4,5]. Epidemiologically, SCAD accounts for approximately 1–4% of all ACS cases, but strikingly, it constitutes up to 35% of myocardial infarctions in women under 50 years of age, emphasizing the need for heightened clinical suspicion in this demographic [6,7]. Clinical presentation can be highly variable, ranging from subtle exertional chest discomfort to catastrophic ST-elevation myocardial infarction (STEMI) with cardiogenic shock. Notably, many patients present with chest pain at rest, often described as severe, retrosternal, or radiating to the left arm or jaw, sometimes accompanied by diaphoresis, nausea, or palpitations, mimicking classic ACS yet in individuals with minimal or absent cardiovascular risk factors [2,8]. This discordance between patient risk profile and acute presentation is a critical diagnostic cue, as misattribution to anxiety or musculoskeletal pain may delay timely intervention. Electrocardiographic findings are equally heterogeneous, with ST-segment elevations most common in the territory of the dissected artery, but non-specific T-wave changes or transient conduction abnormalities can also be observed [9]. Elevations in high-sensitivity troponins typically correlate with infarct size but may be modest despite extensive dissection, reflecting the dynamic and sometimes intermittent nature of luminal compromise [10]. Coronary angiography remains the gold standard for diagnosis, allowing direct visualization of the intimal flap, multiple lumens, or long tubular stenoses characteristic of SCAD [11]. Intracoronary imaging with optical coherence tomography (OCT) or intravascular ultrasound (IVUS) can further delineate intramural hematoma, quantify vessel wall involvement, and guide therapeutic decisions, particularly when angiographic findings are ambiguous [12]. Non-invasive imaging, including coronary computed tomography angiography (CCTA), may assist in follow-up or in cases where invasive assessment poses excessive risk [13]. SCAD most frequently affects the mid-to-distal segments of the left anterior descending artery, though any epicardial vessel can be involved, and multivessel dissections occur in 10–20% of cases [6,14]. Management of SCAD diverges from conventional ACS protocols. Evidence consistently favors a conservative, watchful waiting approach in hemodynamically stable patients with preserved distal flow, as spontaneous healing occurs in the majority of dissections within weeks to months [15,16]. Percutaneous coronary intervention (PCI) carries higher rates of technical failure and iatrogenic propagation of dissection due to fragile vessel walls and intramural hematoma, necessitating judicious case selection [15]. Pharmacologic therapy focuses on symptom control, myocardial protection, and prevention of recurrent events. Beta-blockers are strongly recommended to reduce arterial wall stress and minimize recurrence, with data suggesting a 60% relative risk reduction in subsequent SCAD episodes among adherent patients [17]. Antiplatelet therapy is individualized; dual therapy is often employed initially, particularly if PCI has been performed, while long-term aspirin is generally advised for conservatively managed patients [15]. Statin therapy is reserved for patients with coexisting dyslipidemia or atherosclerosis rather than routine use, given the non-atherosclerotic etiology of SCAD [2,16]. Prognostically, long-term outcomes are generally favorable, with in-hospital mortality under 5% and one-year survival exceeding 95% in most series [7,16]. However, recurrence rates range from 10–30%, underscoring the importance of patient education regarding avoidance of extreme physical or emotional stress, careful pregnancy planning, and ongoing cardiology follow-up [6,17]. Multidisciplinary care, including cardiology, maternal-fetal medicine (when relevant), and rehabilitation services, is crucial to optimize both recovery and secondary prevention.

In summary, SCAD represents a diagnostic and therapeutic challenge requiring a high index of suspicion, particularly in young women presenting with ACS absent conventional risk factors. Recognition hinges on the appreciation of clinical subtleties—acute chest pain at rest, modest biomarker elevation, and non-classic ECG findings—combined with confirmatory angiography and, when necessary, intracoronary imaging. Conservative management remains the cornerstone in stable patients, with targeted pharmacotherapy and vigilant follow-up reducing recurrence and optimizing outcomes. This case highlights the imperative for clinicians to differentiate SCAD from typical atherosclerotic ACS, as misdiagnosis may lead to unnecessary interventions with increased procedural risk [1–17].

Conclusion

Never dismiss acute chest pain in young women without traditional cardiovascular risk factors, as it may herald spontaneous coronary artery dissection rather than benign etiologies. Dynamic or severe pain at rest, even with modest biomarker elevation or non-specific ECG changes, should heighten clinical suspicion for SCAD. Early coronary angiography remains essential for accurate diagnosis, while intracoronary imaging can provide critical detail when angiographic findings are equivocal. Conservative management is often preferred in stable patients, emphasizing the importance of individualized therapy over routine percutaneous intervention. Clinicians should counsel patients regarding recurrence risk, avoidance of extreme physical or emotional stress, and the need for structured follow-up. Ultimately, maintaining vigilance for SCAD in atypical ACS presentations can prevent misdiagnosis, reduce iatrogenic harm, and improve both short- and long-term cardiovascular outcomes.

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