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| RESEARCH ARTICLE

Breaking Down SCAD: A Deep Dive into Spontaneous Coronary Artery Dissection (SCAD)

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| ABSTRACT

Spontaneous Coronary Artery Dissection (SCAD) is a rare, non-atherosclerotic cause of acute coronary syndrome, mainly seen in females with non-traditional cardiovascular risk factors. This case report follows a 44-year-old female who experienced a sudden onset of severe chest pain, which was attributed to SCAD following a coronary angiogram. This case emphasizes the importance of recognizing such presentations within the emergency department, particularly regarding differential diagnosis of chest pain following emotional or physical stressors. It also highlights the need for a multidisciplinary approach in terms of proper management with cardiology, emergency medicine, and mental health care to reduce the risk of reoccurrence. This case report aids in underscoring SCAD's significance as a potential cause of ST Elevation Myocardial Infarction in what is noted as a low-risk population and the critical role of awareness and education in terms of its diagnosis and management.

| KEYWORDS

"STEMI," "Chest Pain," "Stress," "SCAD," and "Angiogram"

| ARTICLE INFORMATION

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1. Introduction

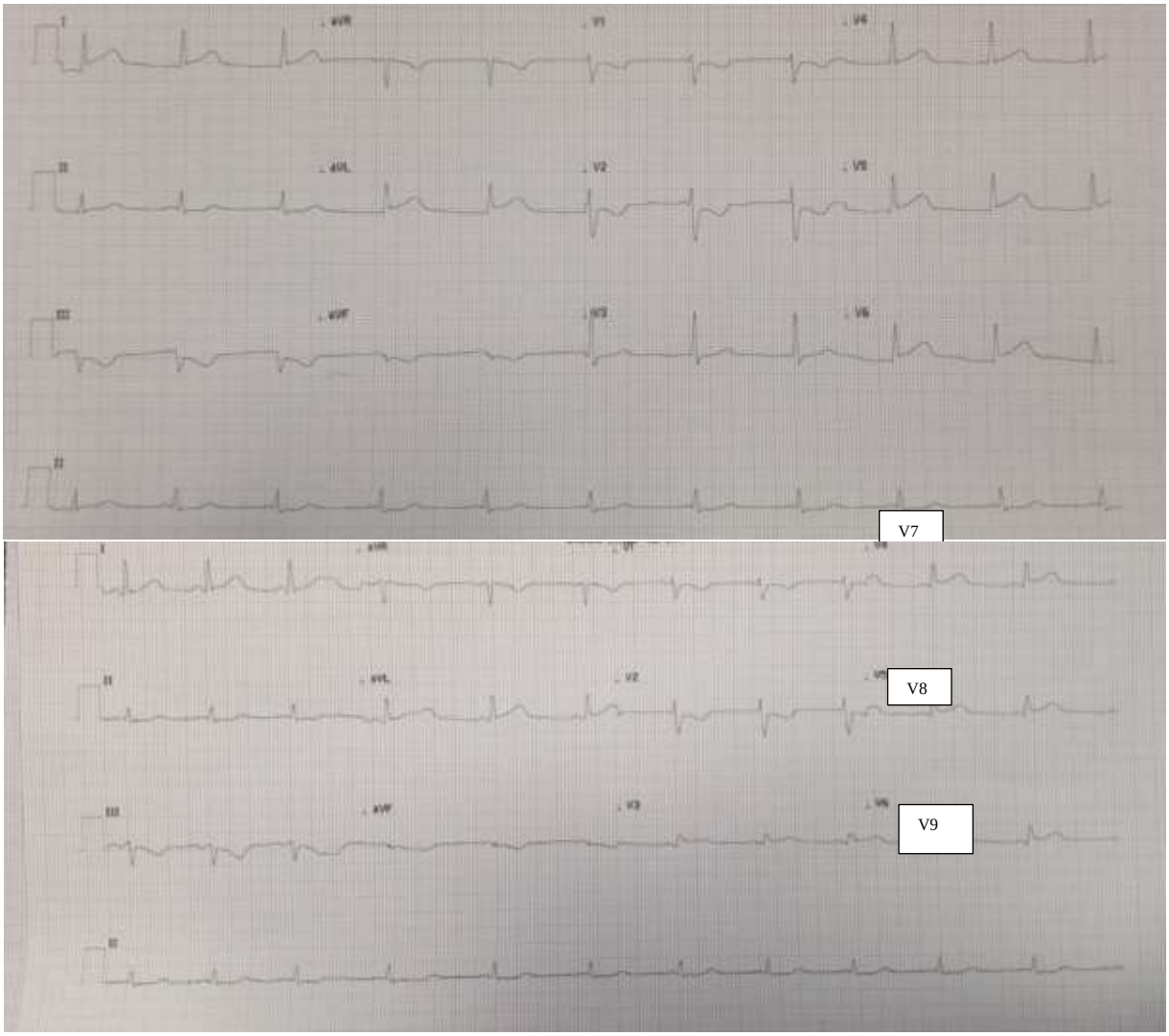
Spontaneous Coronary Artery Dissection (SCAD) is a non-atherosclerotic cause of acute coronary syndrome (ACS) that occurs when a tear forms in the coronary artery wall, leading to blockage of blood flow and myocardial ischemia (1). It is a rare condition but an important cause of myocardial infarction in young females without traditional cardiovascular risk factors (1). Emotional stress like (grief and anxiety) or physical stress like (vigorous exercise) is often associated with sudden onset of SCAD (1-2). While the exact mechanisms aren't fully understood, stress may induce vascular changes, such as heightened blood pressure and increased vascular wall shear stress, contributing to artery dissection (2). This case is significant because it highlights a rare but potentially fatal cardiovascular event that was precipitated by acute emotional stress in a 44-year-old female having chest pain with associated symptoms of ACS, like sweating, nausea, and mild shortness of breath. As stress-related cardiovascular events are often under-recognized, this case underscores the importance of considering SCAD in younger patients. It represents a significant cause of cardiovascular morbidity in low-risk populations.

1.1 Case Report:

A 44-year-old Middle Eastern female with no known medical illnesses was visiting her relative in the intensive care unit, where she started to experience a sudden onset of chest pain. It was central, heavy in nature, and associated with mild shortness of breath, nausea, and sweating. Patient vitals at the time of presentation showed a heart rate of 83 beats per minute, blood pressure of 163\88, oxygen saturation of 100% on room air, Respiratory rate of 18 breaths per minute, and temperature of 37 degrees. The patient looked distressed and in pain during the physical examination. An electrocardiogram (ECG) was ordered, and the initial 12 Lead ECG showed ST elevation in lateral and high lateral leads with ST Depression in V1 and V2, and associated positive R waves. Posterior ECG showed ST-segment elevation in lateral leads associated with ST-Segment Depression in Pericardial Lead. The ECG can be found in Image 1. Initial blood work was taken, which was unremarkable, including creatinine kinase, lactic dehydrogenase, and troponin. As per the guideline with ST Elevation Myocardial infarction, the Cardiac center in the Kingdom of Bahrain was contacted, where the patient was accepted for further management.

Image 1: Electrocardiogram Taken at the Time of Presentation (Anterior and Posterior).

Anterior ECG:



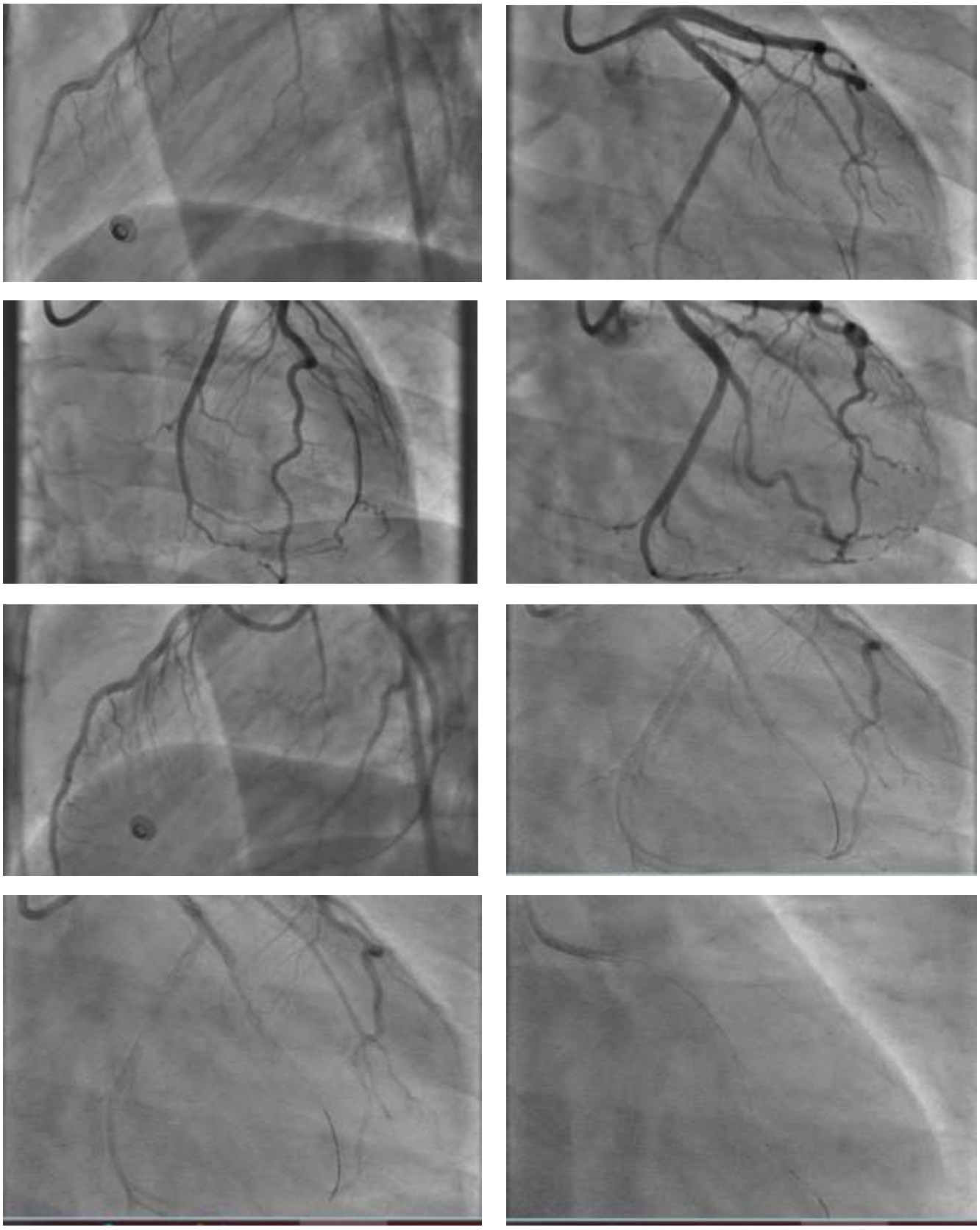
Posterior ECG:

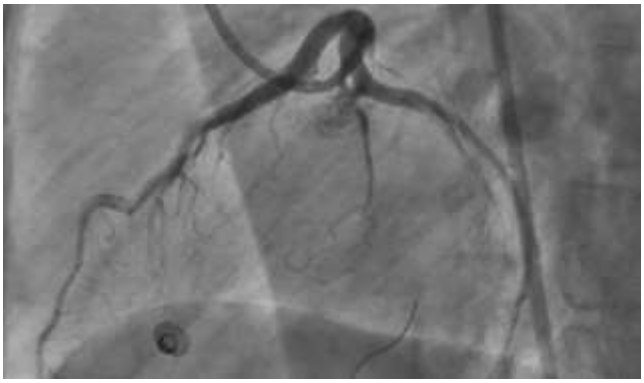
Once presented to the center, a coronary angiogram was performed. The following findings were presented (Image 2):

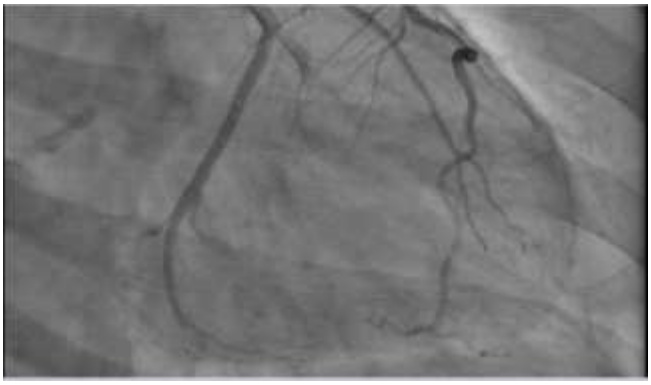
1. Left Main Artery: large, short, and disease-free.

2. Left Anterior Descending Artery (LAD): moderate calibrate vessels with medical calibers diagonal branches and multiple septal perforators. LAD and the diagonal branches were normal.
3. Circumflex Artery (LCx): Large nondominant vessel with moderate caliber OM1, large OM2, and a medium caliber posterolateral branch. OM1 and circumflex were disease-free, while the OM2 was occluded.
4. Right Coronary Artery: moderate caliber co-dominant vessels with high bifurcation into medium caliber PDA and moderate caliber posterolateral branches. All were normal and diseased.

Images 2: Diagnostic Coronary Angiography Results.







Therefore, a decision was made to perform a percutaneous coronary intervention (PCI) to the occluded OM2. During the PCI, after intubating the left main with the guiding catheter, the BMW wire was advanced into the LCx and was negotiated to OM2. The wire easily crossed the total occlusion and was parked in the distal part of the vessel. Wire passage established a faint flow into the distal part of the vessel with a critical lesion in the proximal part. This was dilated with a 2.5mm balloon at an inflation pressure of 6 ATMs. At this stage, we could see a large dissection flap in the area with TIMI-I flow distally. Large doses of intracoronary Adenosine and Nitrates did not help with persistent ST elevation and ongoing chest pain. An IVUS run was performed for the vessel, which revealed a large caliber OM2 with no atheromatous disease. There was a significant dissection of the intima with subintimal hematoma, confirming the diagnosis of spontaneous coronary artery dissection. As there was ongoing chest pain, the entire vessel was treated with a 2mm Cutting balloon at an inflation pressure of 6 ATMs. There was an excellent angiographic result with no residual stenosis and TIMI-III flow to the distal LAD and the jailed diagonal branch. After Cutting balloon dilatation, TIMI-III flow was established in the vessel with complete resolution of ST elevation and relief of chest pain. Given SCAD, the vessel was not stented. The patient was admitted to the Cardiac Care Unit (CCU) for observation and discharged a week later with proper follow-up for her condition.

2. Discussion:

As mentioned earlier, SCAD is a rare, non-atherosclerotic cause of ACS, characterized by a spontaneous tear within the coronary artery wall (1-2). This dissection creates a false lumen that can obstruct blood flow, potentially leading to myocardial infarction or sudden cardiac death (3). SCAD predominantly affects young, healthy women of age between 30-50 years old who lack traditional cardiovascular risk factors, making it a diagnostic challenge (3). Although the exact etiology of SCAD remains unclear, increasing evidence suggests that acute emotional or physical stress can serve as a significant trigger. Emotional stressors such as grief or anxiety, as well as physical exertion, have been implicated in weakening arterial walls and increasing the likelihood of coronary artery dissection (3-4). In cases where emotional stress acts as a trigger, patients often experience a sudden onset of myocardial infarction symptoms, including chest pain, dyspnea, and diaphoresis, as seen in the case presented above. Regarding SCAD, two main pathogenetic mechanisms have been described (3, 5-6):

- A vasa vorsum ruptures with consequent arterial wall bleeding, which affects the external arterial layers.
- Intima-tearing and false lumen formation lead to media hemorrhage and true lumen compression, which affects the internal arterial layers.

However, it is hypothesized that a sudden surge in catecholamines triggered by intense stress leads to increased shear forces on the coronary vessels, which, in susceptible individuals, can result in dissection. This acute elevation in blood pressure, heart rate, and vascular tone may strain the coronary arteries, particularly in patients with underlying vascular abnormalities such as fibromuscular dysplasia (FMD), leading to vessel wall rupture (6-7). Furthermore, stress-related hormonal changes may weaken arterial walls, further increasing susceptibility to dissection (4, 6-7).

Patients with SCAD often present with symptoms indistinguishable from those of acute coronary syndrome. The most common presenting symptom is chest pain, typically described as intense, pressure-like, and radiating to the back or arms. Other symptoms include shortness of breath, nausea, vomiting, and diaphoresis, closely mimicking myocardial infarction. What distinguishes stress-induced SCAD is the presence of a significant emotional or physical event before symptom onset. Despite the acute onset of symptoms, many of these patients lack traditional cardiovascular risk factors like hypertension, diabetes, or hyperlipidemia, making the diagnosis particularly challenging in the long term (7-8).

While SCAD has been historically underdiagnosed, emerging evidence highlights specific risk factors that predispose individuals to this form of acute coronary syndrome. In cases where stress serves as a primary trigger, understanding the interaction between psychological, physiological, and structural factors is crucial. Below is a detailed exploration of the key risk factors for SCAD related to stress (7-9):

1. Emotional stress may be associated with comorbidities such as generalized anxiety disorder and post-traumatic stress disorder.
2. Physical exertion, including sudden trauma and heavy exercise.
3. Pregnancy and post-partum state have been associated with physiological and hormonal changes. This may be a contributing factor to the formation of the dissection. Moreover, childbirth may be noted as the physical trauma precipitating the occurrence of SCAD.
4. Fibromuscular Dysplasia due to preexisting weakened vessels and abnormalities.
5. Connective tissue disorders are associated with structural abnormalities in the vessels affected.
6. Autonomic dysfunction and associated catecholamine surge will increase the stress response within the body, leading to increasing blood pressure and heart rate, which were associated with dissection occurrence.
7. Hormonal fluctuations associated with the menopause state and the use of oral contraceptives. These changes were associated with weakening of the arterial walls in the long term.
8. Vascular inflammation and endothelial dysfunction will lead to structural damage and weakening.

Another interesting association that was recently highlighted by Madias JE is the relationship of SCAD with Takotsubo cardiomyopathy. Takatsubo syndrome (TTS) is characterized by transient left ventricular (LV) dysfunction with a clinical presentation that mimics acute myocardial infarction (MI) (9-10). LV wall motion abnormalities in TTS occur in the absence of culprit coronary artery disease and typically extend beyond a single coronary distribution (9). In most TTS cases, a pattern of apical LV hypokinesis and ballooning with hyperdynamic basal LV segments is observed to disproportionally affect mostly postmenopausal women, is often preceded by emotional or physical stressors, and is associated with outcomes that are like or more like MI (10-11). Takotsubo syndrome and SCAD are distinct clinical entities characterized by chest pain, elevated troponin, and ECG changes.

Atherosclerotic disease with plaque rupture was the most common pathophysiology behind ACS; it is now recognized that women may have other underlying pathophysiology, such as plaque erosion and SCAD (8, 19, 12). The exact prevalence of SCAD remains unclear, and the reported prevalence ranges from 0.1% to 25% (1, 12). Although Spontaneous Coronary Artery Dissection (SCAD) is relatively rare, it has emerged as a leading cause of heart attacks in women under 50, particularly those without the typical risk factors for cardiovascular disease, highlighting the need for greater awareness of its prevalence and risk factors.

Standard diagnostic tools, including ECG and cardiac biomarkers, may show nonspecific or subtle findings in SCAD. While some patients present with ST-segment elevation, suggestive of acute myocardial infarction, others may have more atypical findings, such as ST-segment depression or nonspecific changes (2-5). Coronary angiography remains the gold standard for diagnosis; however, the angiographic findings of SCAD can be subtle, with some cases showing a dissection flap, while others may only exhibit a smooth tapering of the vessel (12). Advanced imaging modalities, such as intravascular ultrasound (IVUS) or optical coherence tomography (OCT), are often required to confirm the diagnosis, revealing features like a false lumen or intramural hematoma (12-13). Based on radiological findings, SCAD is categorized into three types (13-14):

- Type 1 (about 25% of SCAD cases) has typical contrast dye staining of the arterial wall and multiple radiolucent luminal abnormalities, with or without dye hang-up.
- Type 2 (about 70%) has diffused, smooth narrowing of the coronary artery, with the left anterior descending artery the most frequently affected.
- Type 3 (about 5%) mimics atherosclerosis, with focal or tubular stenosis

In this case, the patient experienced type 2, characterized by diffuse narrowing of the coronary artery due to an intramural hematoma without a visible dissection flap. This was confirmed by intravascular ultrasound (IVUS) during angiography.

The management of SCAD due to stress requires a different approach compared to atherosclerotic coronary artery disease (14). Initial treatment is often conservative, as aggressive interventions such as percutaneous coronary intervention (PCI) can increase the risk of extending the dissection. In cases where PCI is unavoidable, extreme caution is exercised to minimize the risk of further vessel damage. Importantly, stenting is typically avoided unless there is ongoing ischemia or hemodynamic instability. Pharmacological management is centered around reducing the workload on the heart and preventing recurrence. Beta-blockers are commonly prescribed to lower heart rate and blood pressure, while dual antiplatelet therapy with aspirin and clopidogrel may be used for patients with ongoing ischemia. In stress-induced SCAD, addressing the underlying stressors is crucial for long-term

management. Stress reduction techniques, psychological support, and counseling are often recommended to prevent recurrence. While the short-term prognosis for SCAD is generally favorable, there is a notable risk of recurrence, with studies reporting recurrence rates as high as 10-20% (10).

In the emergency setting, the recognition and management of Spontaneous Coronary Artery Dissection (SCAD) triggered by stress presents unique challenges. SCAD, particularly in patients without traditional cardiovascular risk factors, can easily be overlooked or misdiagnosed as a more common form of acute coronary syndrome (ACS). This highlights the critical need for emergency physicians to maintain a high index of suspicion for SCAD, especially in younger women and patients presenting with recent emotional or physical stress. Immediate and accurate diagnosis is essential, as the management of SCAD differs significantly from that of atherosclerotic coronary artery disease. While the initial treatment may mirror that of other ACS cases, stabilizing the patient, providing oxygen, and administering antiplatelet therapy—interventional strategies must be carefully considered. Aggressive procedures like percutaneous coronary intervention (PCI) can worsen the dissection, and conservative management may be preferable in stable patients. From an emergency medicine perspective, early identification, stabilization, and swift transfer to a specialized cardiac center are crucial steps in improving outcomes. Additionally, emergency teams must prioritize addressing underlying stressors, providing psychological support when necessary, and initiating discussions about long-term management strategies that include stress reduction and follow-up care. In conclusion, SCAD, due to stress, represents a distinct subset of ACS that demands tailored emergency care, timely diagnosis, and a multidisciplinary approach. Effective management in the acute phase can prevent complications, while long-term strategies focusing on cardiovascular and psychological health can significantly reduce the risk of recurrence. Awareness and preparedness among emergency healthcare providers are key to optimizing outcomes for patients with SCAD.

3. Conclusion:

This case study highlights the complexity and unique challenges of diagnosing and managing Spontaneous Coronary Artery Dissection (SCAD) in the setting of acute emotional stress. A 44-year-old female with no prior history of cardiovascular disease presented with acute chest pain, eventually diagnosed as SCAD. The patient's presentation with typical symptoms of acute coronary syndrome (ACS) mimicked those of more common atherosclerotic events. Yet, the absence of traditional risk factors and the association with emotional stress pointed towards a less typical etiology. SCAD remains an important cause of myocardial infarction, particularly in younger women, and its association with stress underscores the need for awareness in emergency and cardiac care. Prompt recognition, early diagnostic imaging such as coronary angiography, and cautious management, particularly avoiding unnecessary stenting, are vital to reducing complications. In emergency medicine, SCAD should always be considered as a differential diagnosis in younger patients with ACS symptoms, especially when there is a clear emotional or physical stress trigger. Ongoing management should focus not only on cardiac recovery but also on stress reduction and psychological support to mitigate the risk of recurrence. Long-term follow-up, including lifestyle modification, stress management techniques, and routine cardiovascular assessment, will be crucial in ensuring the patient's health and preventing further episodes. This case underscores the importance of a multidisciplinary approach, integrating cardiology, emergency medicine, and mental health care, in the optimal management of SCAD, particularly in cases precipitated by stress.

Authors Contributions:

Conceptualization, Dr. Ali, Dr. Jalal, and Dr. Khurram; Methodology and Case Writing, Dr. Ahmed, Dr. Hawra, and Dr. Reem; Discussion: Imman, Dr. Ali, Dr. Layla, and Dr. Nasser.

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