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Mini Review

Acute Coronary Artery Dissection: A Modern Approach to a Rare Condition

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Abstract

Spontaneous coronary artery dissection (SCAD), is a rare but increasingly recognized cause of acute coronary syndrome (ACS). SCAD affects predominantly younger women who may lack traditional cardiovascular risk factors, posing unique diagnostic and therapeutic challenges. Recent advancements in imaging techniques and conservative management strategies have led to significant improvements in patient outcomes. This review highlights current understandings of SCAD, focusing on clinical presentation, diagnostic modalities, therapeutic approaches, and long-term patient outcomes. Additionally, we explore future research directions for this rare condition.

INTRODUCTION

Acute coronary artery dissection is a lesser-known but important cause of acute coronary syndrome (ACS). SCAD accounts for approximately 1-4% of all ACS cases and can occur in otherwise healthy individuals. Up to 35% of SCAD cases occur in women under the age of 50 [1]. SCAD results from a spontaneous tear in the coronary artery wall, leading to the formation of a false lumen that compresses the true coronary artery lumen, restricting blood flow and causing ischemia [2]. This article reviews SCAD's clinical features, diagnosis, treatment, and long-term outcomes while exploring current advancements and challenges in the management of this condition.

PATHOPHYSIOLOGY OF SCAD

The exact cause of SCAD is not fully understood, but several risk factors and conditions have been linked to its development. One of the most well-documented associations is with fibromuscular dysplasia (FMD), a non-atherosclerotic vascular disease that weakens arterial walls [3]. SCAD has also been linked to hormonal factors, particularly pregnancy, with a significant number of cases occurring in the peripartum or postpartum periods [4]. Other contributing factors include physical or emotional stress, and certain genetic predispositions.

The mechanism of SCAD involves the development of a hematoma between the layers of the coronary artery, which compresses the arterial lumen. This reduces blood flow and can lead to myocardial infarction [5]. Unlike atherosclerotic ACS, which is caused by plaque rupture, SCAD occurs without typical signs of coronary artery disease, making diagnosis and treatment particularly challenging.

CLINICAL PRESENTATION

The clinical presentation of SCAD is similar to other forms of ACS. Patients typically present with chest pain, shortness of breath, or symptoms consistent with myocardial infarction. However, SCAD is distinct because it often affects younger women without traditional cardiovascular risk factors such as hyperlipidemia, smoking, or hypertension [6]. This demographic difference can make SCAD more challenging to recognize and often leads to misdiagnosis or delayed diagnosis.

It is also worth noting that SCAD may be precipitated by emotional stress or intense physical activity, especially in younger women. Physicians must maintain a high index of suspicion for SCAD in young female patients presenting with ACS, particularly in the absence of common cardiovascular risk factors.

DIAGNOSTIC MODALITIES

The gold standard for SCAD diagnosis is coronary

angiography, which can reveal classic signs such as a radiolucent intimal flap or diffuse, long narrowing without atherosclerotic lesions [7]. However, coronary angiography alone may not always confirm the diagnosis. In these cases, intracoronary imaging techniques like optical coherence tomography (OCT) or intravascular ultrasound (IVUS) provide additional details regarding the dissection and can help confirm the presence of a false lumen or intramural hematoma [8].

Coronary computed tomography angiography (CCTA) is another non-invasive option used in stable patients. Although less sensitive than coronary angiography, it is increasingly being used for follow-up in SCAD cases. The advancement in imaging technologies, particularly with OCT and IVUS, has significantly improved the accuracy of diagnosing SCAD and understanding the extent of the dissection.

MANAGEMENT STRATEGIES

SCAD treatment has shifted from aggressive interventional approaches to more conservative management, particularly in patients who are hemodynamically stable [9]. Research indicates that the majority of SCAD lesions heal spontaneously, with favorable long-term outcomes in most cases. Thus, for stable patients without ongoing ischemia, conservative therapy that includes beta-blockers, aspirin, and careful monitoring is often recommended [10]. Beta-blockers help reduce arterial wall stress, which may lower the risk of SCAD recurrence.

For patients with ongoing myocardial ischemia, hemodynamic instability, or those with left main coronary artery involvement, invasive strategies such as percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) may be necessary [11]. However, PCI in SCAD cases is challenging due to the fragility of the dissected arteries, and complications like stent malposition or dissection propagation are more frequent [12].

Patients with SCAD also require long-term follow-up to monitor for recurrent episodes. Recurrence rates can be as high as 20%, especially in individuals with underlying conditions such as FMD [13]. Thus, long-term management includes regular cardiovascular assessments, lifestyle modifications, and genetic counseling where appropriate.

Prognosis and Long-Term Outcomes

The long-term prognosis for SCAD patients is generally favorable, especially with conservative management. Most patients recover fully, with minimal long-term complications. Nevertheless, the recurrence rate remains a significant concern. Studies indicate that about 10-20% of SCAD patients experience recurrent dissections, which highlights the importance of regular follow-up and monitoring for signs of recurrence [14].

Several factors influence prognosis, including the location of the dissection, the presence of left main coronary artery involvement, and whether the dissection was treated conservatively or invasively. Patients treated conservatively tend to have better long-term outcomes compared to those undergoing PCI or CABG, due to the high risk of procedural complications in SCAD patients [15].

Future Directions

Future research on SCAD will likely focus on identifying genetic markers and hormonal influences that may predispose individuals to SCAD. Additionally, more studies are needed to refine guidelines on when to intervene invasively and how best to manage recurrent SCAD cases. Genetic studies may offer insight into why certain patients, particularly young women, are more prone to SCAD. Another important area for future research is improving post-SCAD care, with a focus on preventing recurrence and managing long-term cardiovascular health.

CONCLUSION

Spontaneous coronary artery dissection is a rare but important cause of acute coronary syndrome, especially in younger women without traditional cardiovascular risk factors. Advances in imaging and a shift toward conservative management have improved patient outcomes, but challenges remain, particularly regarding recurrence rates and the management of complex cases. Ongoing research will continue to refine our understanding of SCAD and improve both acute and long-term management strategies.

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