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Spontaneous coronary artery dissection secondary to Japanese Encephalitis: A rare case report

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Abstract
Background: Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome predominantly affecting young women. While various precipitating factors have been identified, viral infections as triggers for SCAD remain extremely rare, with Japanese encephalitis (JE) never previously reported as a causative factor.
Case Summary: We report a 53-year-old diabetic male smoker who developed multivessel SCAD following recovery from JE viral meningoencephalitis. The patient initially presented with high-grade fever and altered mental status with positive neck rigidity. CSF examination confirmed viral meningoencephalitis with positive JE virus testing. Following recovery from the acute neurological syndrome, the patient developed NYHA Class III dyspnea. Cardiac evaluation revealed elevated cardiac biomarkers (troponin 0.04 ng/mL, BNP 800 pg/mL), left bundle branch block, and severely reduced ejection fraction (30%) with global left ventricular hypokinesis1. Coronary angiography demonstrated extensive multivessel SCAD involving proximal LAD with long-segment dissection and distal subtotal occlusion, non-flow limiting dissection in major obtuse marginal, and flow-limiting dissection causing 90% stenosis in proximal RCA. Viability testing revealed non-viable myocardium in LAD territory and mixed viability in LCx and RCA territories. Comprehensive workup for traditional SCAD etiologies including autoimmune markers, genetic testing, and fibromuscular dysplasia screening was negative.
Conclusion: This represents the first reported case of JE-induced SCAD, expanding our understanding of viral infections as precipitating factors for coronary artery dissection. The temporal relationship between viral encephalitis and subsequent cardiac presentation, combined with absence of other identifiable risk factors, strongly suggests a causal relationship through inflammatory or direct viral mechanisms.

Keywords: Spontaneous coronary artery dissection, Japanese encephalitis, viral meningoencephalitis, case report, coronary angiography, myocardial viability, cardiac complications, viral cardiopathy, neurogenic heart disease, multivessel dissection

Introduction

Case Summary

A 53-year-old male with longstanding diabetes mellitus and smoking history presented to the emergency department with high-grade fever and altered mental status. Physical examination revealed neck rigidity consistent with meningeal irritation. Non-contrast computed tomography of the head was normal. Cerebrospinal fluid examination demonstrated findings consistent with viral meningoencephalitis, including lymphocytic pleocytosis (150 cells/mm³), elevated protein (80 mg/dL), and normal glucose levels. CSF testing confirmed Japanese encephalitis virus positivity, establishing the diagnosis of JE viral meningoencephalitis. The patient received supportive care as per standard protocols and showed gradual clinical improvement with complete recovery from the acute neurological syndrome 2. Several weeks following discharge, the patient presented to cardiology outpatient department with New York Heart Association Class III dyspnea. Cardiac biomarkers revealed troponin elevation at 0.04 ng/mL, myoglobin at 77 ng/mL, and significantly elevated B-type natriuretic peptide at 800 pg/mL. Electrocardiogram showed left bundle branch block pattern with normal sinus rhythm.

Echocardiography demonstrated severely reduced left ventricular ejection fraction of 30% with global left ventricular hypokinesis affecting LAD territory more than LCx and RCA territories, along with mild mitral regurgitation. Given the non-acute presentation with heart failure symptoms, coronary angiography was performed, revealing extensive multivessel SCAD: proximal LAD showed long-segment SCAD with distal subtotal occlusion, major obtuse marginal branch had non-flow limiting dissection, and proximal RCA demonstrated flow-limiting dissection causing approximately 90% long-segment stenosis.

Myocardial viability assessment revealed non-viable myocardium in LAD territory and mixed viability in LCx and RCA territories. Comprehensive investigation for traditional SCAD risk factors included negative autoimmune markers (ANA, ANCA, rheumatoid factor), negative HLA-B27, and whole exome sequencing showing no significant pathogenic variants associated with SCAD or connective tissue disorders.

Given the patient's asymptomatic status and predominantly non-viable myocardium in LAD territory, revascularization was deferred. The patient was initiated on guideline-directed medical therapy including beta-blockers, which have been shown to reduce SCAD recurrence rates, and is currently doing well with plans for repeat coronary angiography with intravascular imaging.

Discussion

SCAD is associated with various predisposing and precipitating factors. The most commonly identified risk factor is fibromuscular dysplasia, present in 25-86% of SCAD patients depending on screening methodology [1, 2]. In the largest cohort study, 86% of SCAD patients had concomitant FMD affecting noncoronary territories [3]. Other established risk factors include pregnancy (particularly peripartum period), connective tissue disorders such as Ehlers-Danlos syndrome and Marfan syndrome, emotional stress, physical exertion, and certain medications [4, 5].

Interestingly, traditional cardiovascular risk factors such as diabetes, smoking, and hypertension are less prevalent in SCAD patients compared to those with atherosclerotic coronary disease [4, 6]. Our patient's presentation with diabetes and smoking history is somewhat atypical for SCAD, though these factors do not exclude the diagnosis.

The association between viral infections and SCAD has gained attention recently, particularly following reports of COVID-19-related SCAD cases [7]. The proposed mechanisms include direct viral injury to coronary arteries, systemic inflammatory response leading to endothelial dysfunction, and immune-mediated vascular damage [7, 8].

SARS-CoV-2 infection has been hypothesized to trigger SCAD through several pathways: infiltration and activation of T-cells in the adventitia, production of inflammatory cytokines and proteases, stimulation of vasa vasorum proliferation with subsequent rupture, and direct viral invasion of coronary arteries via ACE2 receptors [7]. The virus can prompt inflammation in the vessel wall, causing endothelial cell death and damage to the hemostatic system and vascular tone, ultimately making the vessel wall fragile and resulting in dissection [7].

Japanese encephalitis virus can cause systemic complications beyond the central nervous system [9]. The virus affects brainstem centers responsible for cardiovascular regulation, potentially leading to neurogenic cardiac complications [9, 10]. The case fatality rate for JE ranges from 14-30%, with stroke being the most common long-term neurological complication, followed by epilepsy, encephalopathy, and parkinsonism [10-12].

The pathophysiology of JE-induced cardiac complications may involve direct viral effects on the myocardium, neurogenic mechanisms through brainstem involvement, or systemic inflammatory responses [10]. In our case, the temporal relationship between JE infection and subsequent SCAD development, combined with the absence of other identifiable risk factors, strongly suggests a causal relationship.

Although the majority of SCAD patients have a favorable prognosis, there is still controversy about the type of management for SCAD patients, which may vary depending on the type and severity of the presentations. It is generally recognized that conservatively managed SCAD frequently heals and that percutaneous coronary intervention for SCAD is associated with high rates of technical failure or unsatisfactory outcomes, due in part to false lumen extension caused by tracking of the hematoma during stent deployment [13, 14].

Conservative management of patients with SCAD has shown better clinical outcomes than invasive management, with lower rates of death, myocardial infarction, and target vessel revascularization [13, 14]. PCI complications occur in approximately 38.6% of cases, with 13.0% experiencing serious complications and procedural failure rates as high as 53% [13].

Indications for revascularization may be considered in the following circumstances: complete vessel occlusion with thrombolysis in myocardial infarction with zero flow, which is unlikely to resolve with medical treatment alone, ongoing ischemia, left main stem involvement, recurrent angina, hemodynamic instability, and sustained ventricular arrhythmias [13, 14]. Revascularization should be considered only in high-risk patients, defined according to angiographic and clinical characteristics: persistent chest pain, persistent ST-segment elevation, hemodynamic or electrical instability, proximal location or multiple dissections, left main dissection, and TIMI 0 and 1 coronary flow [13].

In those treated with conservative management, prolonged hospitalization should be considered [13, 14]. Current recommendations support an extended inpatient monitoring period of 3-5 days for conservatively managed SCAD patients, given that the majority of patients readmitted for recurrent AMI occurred within the first 48 hours following discharge [13].

SCAD recurrence remains a significant concern, with rates ranging from 10-19% within 2-3 years [3, 13]. Predictors of SCAD recurrence include fibromuscular dysplasia (5-fold increased risk), migraine headaches (3.4-fold increased risk), and hypertension [3].

Importantly, beta-blocker therapy has been associated with reduced recurrence rates (hazard ratio 0.36, $p = 0.004$) [3]. In the largest prospective SCAD cohort, hypertension increased the risk of recurrent SCAD (hazard ratio 2.46), while beta-blocker use was protective against recurrence [3]. This supports the use of beta-blockers in our patient's management strategy.

Myocardial viability assessment plays a crucial role in management decisions for patients with SCAD and reduced

ejection fraction [6]. In our case, viability testing revealed predominantly non-viable myocardium in the LAD territory, supporting the decision for conservative management. The presence of mixed viability in the LCx and RCA territories suggests potential for functional recovery with optimal medical therapy [13].

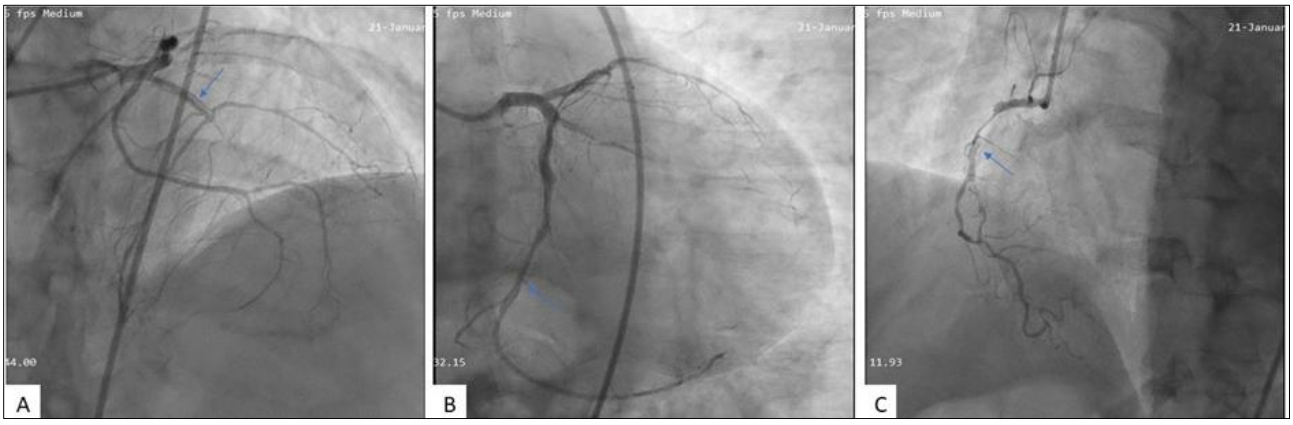
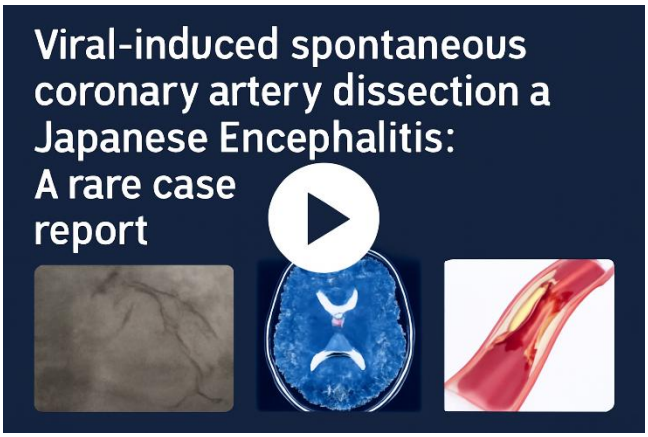


Fig 1: Coronary angiography images demonstrating spontaneous coronary artery dissection (SCAD) in multiple vessels.

Panel A: Long-segment SCAD involving the proximal left anterior descending (LAD) artery with distal subtotal occlusion (arrowhead).

Panel B: Non-flow limiting SCAD involving the major obtuse marginal (OM) branch of the left circumflex artery (arrowhead).

Panel C: Flow-limiting SCAD in the mid-segment of the right coronary artery (RCA) with approximately 90% stenosis (arrowhead).



Supplementary Video 1: Coronary angiography demonstrating multivessel spontaneous coronary artery dissection.

Video 1 URL: https://www.cardiologyjournals.net/uploads/archives/videos/119/1.mp4

Scan me to Watch Video

The video sequentially highlights: (1) Proximal LAD dissection with long-segment involvement and distal tapering; (2) Dissection in the major obtuse marginal branch; and (3) Flow-limiting mid-RCA dissection with severe luminal narrowing.

This case expands our understanding of SCAD pathophysiology by demonstrating that viral encephalitis can serve as a precipitating factor. The mechanism likely involves systemic inflammatory responses, direct viral effects on coronary arteries, or neurogenic cardiac dysfunction secondary to brainstem involvement [7, 10, 14].

Healthcare providers should maintain awareness of potential cardiac complications in patients recovering from viral encephalitis, particularly when patients develop new cardiac symptoms. The conservative approach should be the preferred method of treatment in patients with SCAD, with PCI revascularization reserved for high-risk presentations given the high prevalence of periprocedural complications [13, 14].

Abbreviations

SCAD: Spontaneous Coronary Artery Dissection; JE: Japanese Encephalitis; ACS: Acute Coronary Syndrome; NYHA: New York Heart Association; CSF: Cerebrospinal Fluid; CT: Computed Tomography; BNP: B-type Natriuretic Peptide; ECG: Electrocardiogram; LBBB: Left Bundle Branch Block; EF: Ejection Fraction; LAD: Left Anterior Descending (artery); LCx: Left Circumflex (artery); RCA: Right Coronary Artery; ANA: Antinuclear Antibody; ANCA: Anti-Neutrophil Cytoplasmic Antibodies; HLA: Human Leukocyte Antigen; PCI: Percutaneous Coronary Intervention; AMI: Acute Myocardial Infarction; TIMI: Thrombolysis In Myocardial Infarction; FMD: Fibromuscular Dysplasia; COVID-19: Coronavirus Disease 2019; ACE2: Angiotensin-Converting Enzyme 2; MRI: Magnetic Resonance Imaging; DNA: Deoxyribonucleic Acid.

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Conflict of Interest
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