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CASE REPORT

CLINICAL CASE

Spontaneous Dissection of a Septal Coronary Artery Mimicking Myocarditis

Mihály Károlyi, MD, PhD,[a] Christian Templin, MD, PhD,[b] Hatem Alkadhi, MD, MPH,[a] Robert Manka, MD[a,b,c]

ABSTRACT

Acute chest pain and dyspnea often raise coronary disease suspicion. When echocardiography and cardiac computed tomography findings appear normal, alternative diagnoses should be explored. We present a case initially suggestive of myocarditis but later revealed as coronary dissection by cardiac magnetic resonance. This case emphasizes the role of advanced imaging in atypical cardiac presentations. (J Am Coll Cardiol Case Rep 2024;29:102151) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 38-year-old male patient was admitted to our institution (University Hospital Zurich, Zurich, Switzerland) for persistent chest pain during exercise and at rest, along with worsening dyspnea. He had a history of sporadic smoking (12 pack-years) and had no known cardiovascular risk factors. One month earlier, the patient sought medical attention at our emergency department for weakness and dry cough, diagnosed as a viral respiratory infection.

This case report received the proper ethical oversight in accordance with the Human Research Ordinance in Switzerland, and the patient gave informed consent to use his data.

LEARNING OBJECTIVES

- To understand the diagnostic challenges associated with detecting coronary artery dissections.
- To emphasize the value of multimodal imaging, including CMR, in cases where clinical and standard imaging findings are inconclusive.
- To raise awareness of the increasing prevalence of SCAD, including involvement of septal branches, and its potential to mimic other cardiac conditions.

PAST MEDICAL HISTORY

The patient had no significant past medical history.

DIFFERENTIAL DIAGNOSIS

On admission, the patient's vital parameters were stable, with a slightly elevated blood pressure of 150/103 mm Hg, a heart rate of 60 beats/min, and peripheral oxygen saturation at 99%. The baseline electrocardiogram (ECG) displayed a maximum 1-mm J-point elevation in leads V_3 to V_5, along with concomitant peaked T waves (**Figure 1**), consistent with previous ECGs taken during a visit for viral infection a month earlier and in September 2020. Repeated ECGs performed during hospitalization remained unchanged, and blood pressure returned to

From the [a]Institute of Diagnostic and Interventional Radiology, University Hospital Zurich, Zurich, Switzerland; [b]Department of Cardiology, University Heart Center, University Hospital Zurich, Zurich, Switzerland; and the [c]Institute for Biomedical Engineering, University and ETH Zurich, Zurich, Switzerland.

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ABBREVIATIONS AND ACRONYMS

CMR = cardiac magnetic resonance

CT = computed tomography

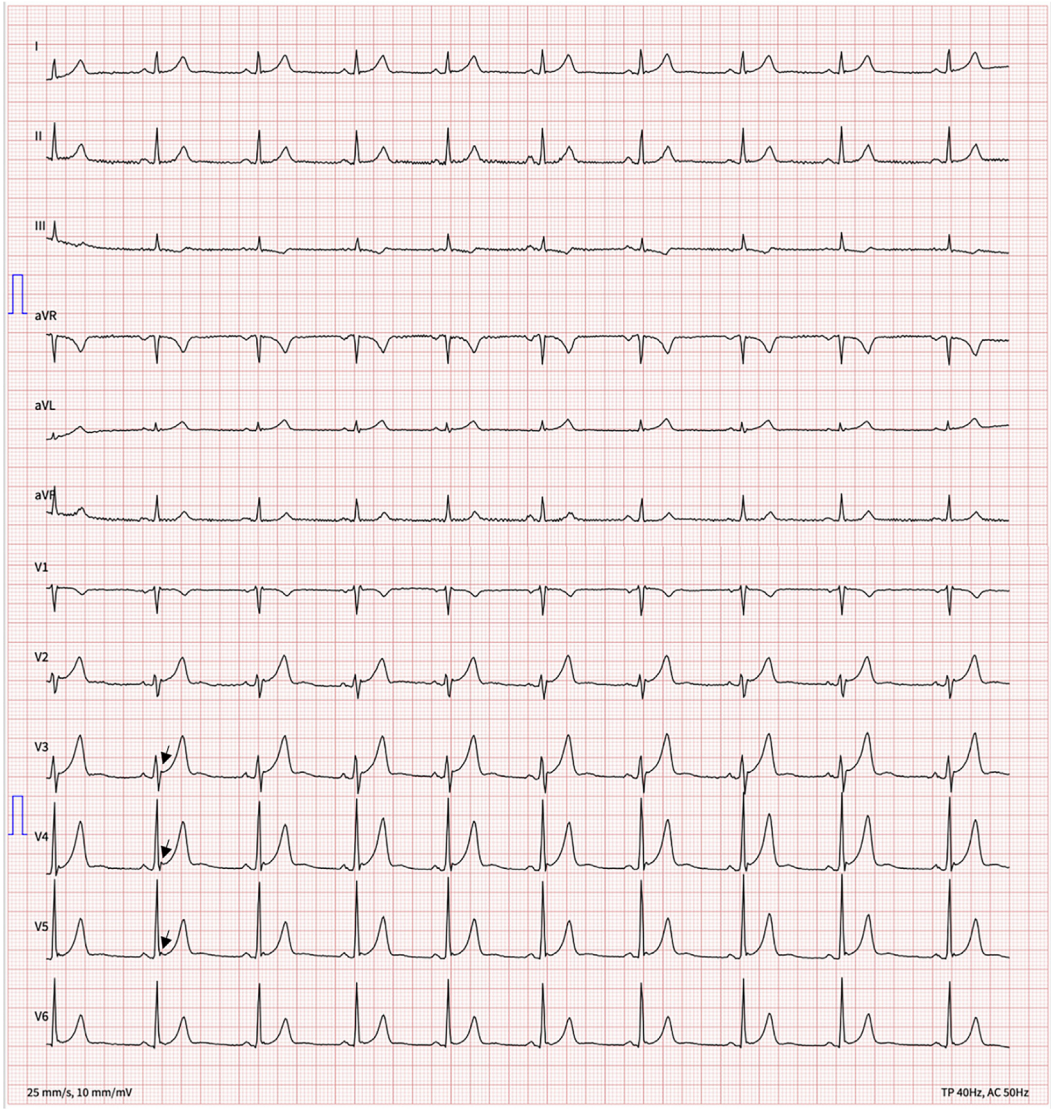
ECG = electrocardiogram

SCAD = spontaneous coronary artery dissection

TTE = Transthoracic echocardiography

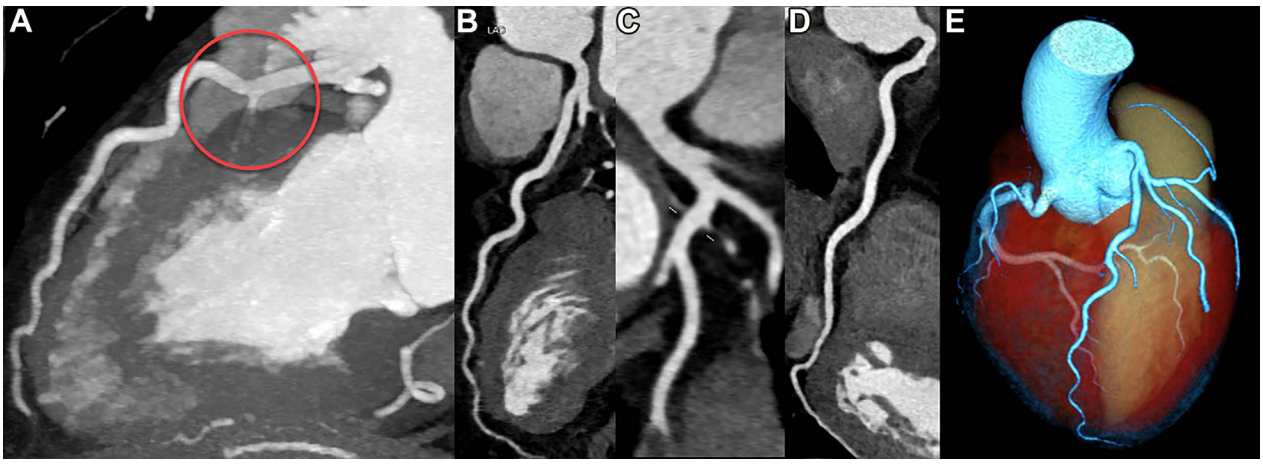
normal. Initial evaluation also included a negative D-dimer test result, excluding pulmonary embolism, normal inflammatory parameters, and negative polymerase chain reaction for SARS-CoV-2. Cardiac biomarkers however were elevated, leading to an initial diagnosis of acute myocarditis (creatine kinase, 219 U/L; high-sensitivity troponin T, 101 ng/L, with a dynamic increase in the first 24 hours of hospitalization up to 350 ng/L; and N-terminal pro-B-type natriuretic peptide, 182 ng/L). Pericarditis was considered unlikely given the absence of typical ECG changes, a lack of relevant pericardial effusion visible in focused transthoracic echocardiography (TTE), and a lack of elevated inflammatory markers. Consecutive TTE findings were normal, left ventricular function and wall motion were preserved, and especially, no wall motion abnormalities of the septal segments were described.

FIGURE 1 The 12-Lead Electrocardiogram on Hospital Admission Showing No Signs of Acute ST-Segment Elevation Myocardial Infarction and No Repolarization Abnormalities Suggestive of (Myo)pericarditis



Note the J-point elevations in leads V_3 to V_5 up to 1 mm, accompanied by peaked T waves, which were in line with electrocardiograms obtained during previous visits for a viral infection a month earlier and in September 2020.

FIGURE 2 Cardiac Computed Tomography Images Initially Interpreted as Normal, With No Significant Coronary Artery Disease



Retrospectively, considering the results of cardiac magnetic resonance, a subtotal occlusion of a perforating septal branch could be discerned. (A) Left anterior descending coronary artery and the perforating septal artery on maximum intensity projection (red circle). Curved planar reformatted images of (B) the left anterior descending coronary artery, (C) the left circumflex artery, and (D) the right coronary artery. (E) A 3-dimensional volume-rendered image of the heart.

INVESTIGATIONS

Coronary computed tomography (CT) angiography was performed, thereby ruling out significant coronary artery disease of the main coronary arteries (Figures 2A to 2E). Cardiac magnetic resonance (CMR) imaging revealed a transmural myocardial scar in the basal anteroseptal and inferoseptal myocardial segments. This scar was accompanied by akinesia, myocardial edema, microvascular obstruction, and elevated T1 and T2 mapping values in this region, which were 5 SDs above normal tissue (T1: 1,145 ms [normal: 988.7 ± 27.8 ms]; T2: 63 ms [normal: 48.4 ± 3.1 ms]) (Figures 3A to 3G). These findings are consistent with myocardial injury, most likely stemming from a (sub-)acute infarction in the territory supplied by the first septal branch of the left anterior descending coronary artery.

Concomitant invasive coronary angiography confirmed subtotal stenosis of the first septal perforator (reference diameter of 1.5 mm), likely caused by a spontaneous coronary artery dissection (SCAD) (Figures 4A and 4B). No thrombosis was observed, and an interatrial shunt, a known rare cause of paradoxical coronary artery embolism, was excluded with contrast echocardiography.[1] The subtotal occlusion of the perforating septal branch could be revealed on coronary CT angiography retrospectively, knowing the CMR and invasive coronary angiography findings (Figures 2A to 2E).

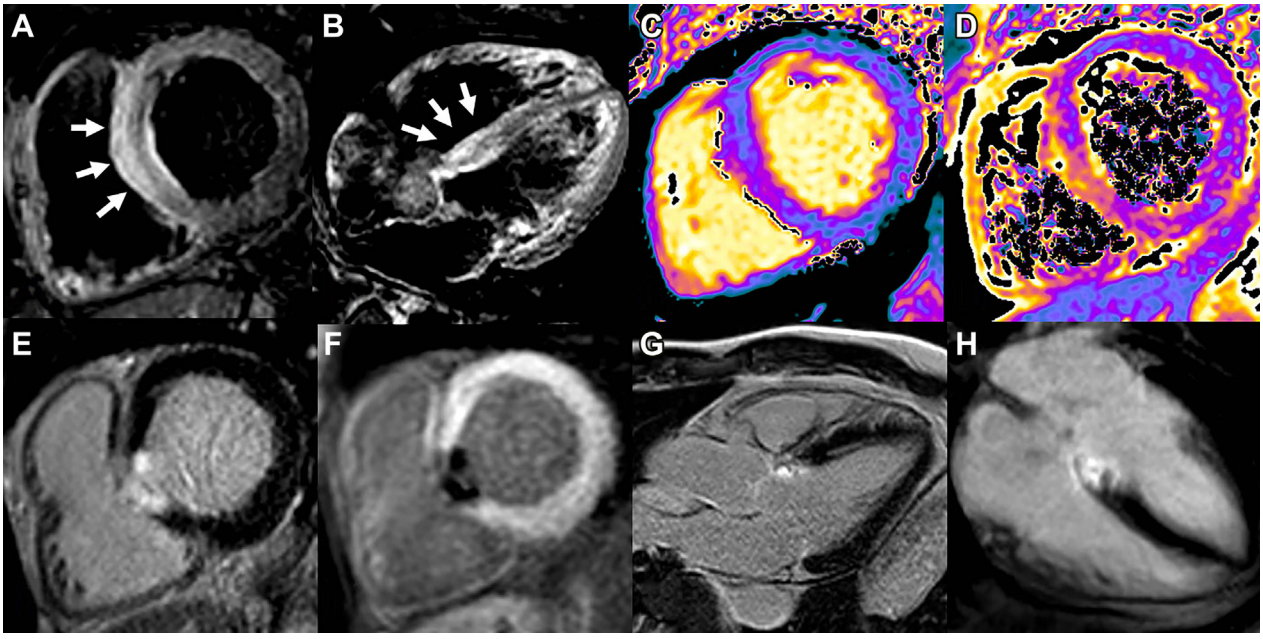
MANAGEMENT (MEDICAL/INTERVENTIONS)

Conservative antithrombotic therapy with aspirin was initiated because of the small diameter of the septal branch, along with additional rosuvastatin, as a result of newly recognized dyslipidemia in this patient. Cardiac biomarkers normalized within 5 days, and the patient was discharged in good condition. Follow-up with invasive coronary angiography as part of an internal register for SCAD showed normal coronary arteries, with no detectable stenosis in the first septal branch (Figures 4C and 4D). Control echocardiography findings and laboratory values were within normal ranges.

DISCUSSION

This case presents a diagnostic challenge in the evaluation of acute chest pain and dyspnea. Initially, the patient's clinical presentation, previous viral infection of the upper airways, unremarkable ECG findings, and lack of significant pericardial effusion pointed toward acute myocarditis. However, subsequent CMR revealed a transmural myocardial scar, edema, and microvascular obstruction in the myocardial septum, a pattern consistent with myocardial injury. This unexpected finding led to the suspicion of coronary artery involvement, despite initially normal-appearing coronary CT angiography findings.

FIGURE 3 Cardiac Magnetic Resonance Images Displaying Myocardial Edema in the Septal Region With Correspondingly Elevated T1 And T2 Mapping Times and Late Gadolinium Enhancement



These findings are consistent with a (sub-)acute myocardial infarction, most likely resulting from a coronary dissection of a septal branch. T2-weighted images in the (A) short-axis and (B) 4-chamber views, showing hyperintense signal in the septum indicative of edema (arrows). (C) Native T1 and (D) native T2 mapping images in short-axis orientations. Late gadolinium enhancement images in the (E and F) short-axis, (G) 3-chamber, and (H) 4-chamber views, illustrating myocardial fibrosis (scar) in the basal septum.

SCAD is an increasingly recognized cause of acute coronary syndromes, particularly in young individuals with few or no conventional risk factors.[1] SCAD primarily affects women,[2] but as demonstrated in this case, it can also occur in men. In a previous study, up to 22% of the affected patients were men.[3] Although SCAD is a relatively rare phenomenon, its prevalence may be underestimated because it can mimic other conditions, thus leading to potential misdiagnosis.

The combination of advanced imaging modalities, such as incorporating CMR alongside an anatomical test of the coronary arteries, has proved invaluable in differentiating SCAD from other cardiac disorders. CMR provides detailed information about myocardial tissue characteristics, including the presence of myocardial edema, microvascular obstruction, and late gadolinium enhancement patterns. These features aid in the identification and characterization of myocardial injury and help to establish the diagnosis of SCAD.[4]

The case we present here highlights the importance of considering SCAD in the differential diagnosis of acute coronary syndromes, especially when clinical presentation, ECG, and laboratory findings do not align with the working diagnosis. Coronary CT angiography, albeit effective in visualizing coronary anatomy, may not detect dissections in small coronary branches, thereby underscoring the value of additional imaging modalities, such as CMR.

Recent studies have shed light on the prevalence and clinical characteristics of SCAD.[5] In a large SCAD registry, a prevalence of 0.7% for spontaneous dissection of the first septal perforator was reported.[6] This study suggests that SCAD, including involvement of septal branches, may be more common than previously thought. Moreover, many SCAD cases were found to be unrelated to atherosclerosis or other traditional risk factors. Highlighting the significance of early diagnosis in SCAD, Sharkey et al[6] reported a case of acute myocardial infarction resulting from an SCAD involving the first septal perforator. Our case similarly underscores the wide variability in clinical presentations associated with SCAD.

The decision of routine follow-up coronary angiography in patients with SCAD warrants careful evaluation, with a focus on the risk-to-benefit ratio. Although angiography can be essential in cases with

clinical indications such as recurrent symptoms, it is important to consider the potential risks associated with the procedure, including radiation exposure and contrast medium use. The decision should be made on a case-by-case basis, considering the individual patient's clinical condition and input from a multidisciplinary team of cardiologists and health care providers.

In summary, this case underscores the significance of advanced imaging techniques, particularly CMR, in diagnosing SCAD, even when clinical presentation and standard imaging findings are inconclusive. Additionally, it emphasizes the importance of considering alternative diagnoses, especially in patients with acute chest pain and dyspnea. Clinicians should be vigilant in recognizing the potential for SCAD because early diagnosis and appropriate management are crucial for optimal outcomes in these patients. Decisions regarding routine follow-up angiography should be individualized by taking into account the specific patient's clinical condition and the risk-to-benefit ratio associated with the procedure.

FOLLOW-UP

The patient underwent a 2-week rehabilitation program and regained normal physical performance.

CONCLUSIONS

This case illustrates the significance of advanced imaging techniques, such as CMR, in diagnosing SCADs, even when clinical and standard imaging findings are inconclusive.

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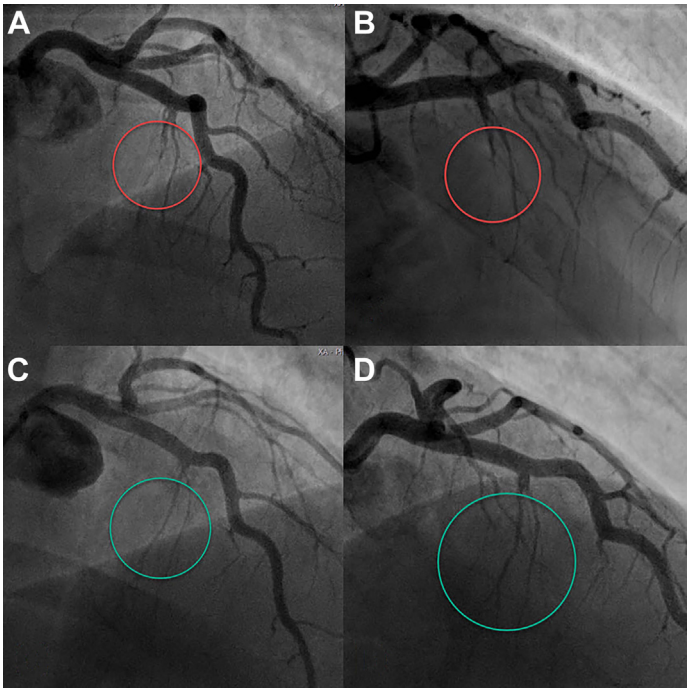
The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

FIGURE 4 Baseline and Follow-Up Invasive Coronary Angiography



(A and B) Baseline invasive coronary angiography images revealing a subtotal occlusion of the first septal perforator branch of the left anterior descending coronary artery (red circles), most likely reflecting spontaneous dissection. (C and D) Follow-up invasive coronary angiography at 1.5 months showing normal coronary arteries with no visible septal branch dissection (green circles).

ADDRESS FOR CORRESPONDENCE: Dr Robert Manka, Department of Diagnostic and Interventional Radiology, University Hospital Zurich, Raemistrasse 100, 8091 Zurich, Switzerland. E-mail: robert.manka@usz.ch.

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KEY WORDS acute coronary syndrome, cardiac magnetic resonance, computed tomography, dissection