CASE REPORT

Rapid Healing of a Spontaneous Coronary Artery Dissection Detected by Computed Tomography Angiography

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Summary
Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and little is known about characteristics, treatment, and follow-up of patients with SCAD. Computed tomography angiography (CTA) plays an important role in the diagnosis and management of SCAD. Herein, we describe a case of a patient with SCAD who underwent successful conservative management, and in whom CTA was able to detect rapid healing of the dissection, 4.5 hours post-dissection.

Key words: Myocardial infarction, Coronary dissection

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potaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and little is known about characteristics, treatment, and follow-up of patients with SCAD. Advances in imaging techniques and better recognition of SCAD have led to several new insights into this understudied condition.1-3) Computed tomography angiography (CTA) plays an important role in the diagnosis and management of SCAD.

Herein, we describe a case of a patient with SCAD who underwent successful conservative management and in whom rapid healing of the dissection was detected by CTA.

Case Report
A 42-year-old man, a hospital employee, with a history of current smoking experienced acute onset of chest pain while working at the hospital. An electrocardiogram (ECG) showed ST-segment elevation in lead I, aVL, V5, V6, and V7. The patient was transferred to the Kokura Memorial Hospital (Kitakyushu, Japan). Emergency coronary angiography showed significant stenosis in the proximal segment of the left anterior descending artery (LADA) with thrombolysis in myocardial infarction (TIMI) 3 flow, occlusion of the first diagonal branch with TIMI 1 flow (Figure 1A), and diffuse (>20 mm) smooth narrowing between the proximal LADA and the left main coronary artery (LMCA) (Figure 1B). There were no signs of atherosclerosis of the other segments of the coronary arteries. Left ventriculography revealed marked anterolateral and apical wall hypokinesia with moderately impaired left ventricular ejection fraction, which identified the infarct-related arteries as being the proximal LADA and the diagonal branch, in agreement with the ECG changes. Given the diffuse narrowing extending to the LMCA, intracoronary imaging with optical coherence tomography or intravascular ultrasound was not performed because of the potential risks of vessel occlusion/dissection. Intra-aortic balloon pumping (IABP) was used to prevent the development of cardiogenic shock and to increase the coronary blood flow. The time from the onset of symptoms to IABP was 2.5 hours. Immediately after IABP, the patient was free of chest pain and the ECG changes resolved; the percentage resolution of ST-segment elevation between ECGs obtained before and 30 minutes after IABP was 45%. Therefore, coronary revascularization was not performed.

Because of the dubious angiographic appearance of the infarct-related arteries, multidetector 64-row coronary CTA was performed 2 hours after IABP and revealed a false lumen with intramural hematoma between the proximal LADA and the LMCA and between the proximal LADA and the first diagonal branch (Figures 2A, 2B, 2C), with no significant stenosis in the proximal LADA (Figures 2B, 2C) and restoration of coronary flow in the first diagonal branch (Figures 2B, 2C), demonstrating that the ST-segment elevation myocardial infarction in this case was caused by SCAD.

Since the dissection extended to the LMCA, the patient underwent invasive coronary angiography the following day, which showed resolution of the dissection with no significant stenosis in the proximal LADA, no occlusion of the first diagonal branch, and no diffuse smooth narrowing between the proximal LADA and the LMCA; TIMI 3 flow had been established (Figures 1C, 1D). Two days after admission, the intra-aortic balloon was removed.
with no complications. Creatine kinase peaked at 5322 IU/L (normal, 30-180 IU/L). CTA on the 8th day post-dissection revealed the false lumen with intramural hematoma between the proximal LADA and the LMCA and between the proximal LADA and the first diagonal branch (Figure 2D). The patient was discharged with aspirin, angiotensin-converting enzyme inhibitor, nitrate, and statin therapy on the 14th day post-infarction. There were no recurrent ischemic events or other complications during the hospitalization.

At the 3.5- and 10-month follow-ups, the patient was free of any symptoms of ischemia on medical therapy and CTA revealed disappearance of the false lumen, which indicated complete vessel healing of SCAD (Figures 2E, 2F).

**Discussion**

In the present case, serial angiographic changes of the infarct-related arteries between the proximal LADA and the LMCA and between the proximal LADA and the diagonal branch could be observed, and these SCAD arteries healed spontaneously with conservative management. In addition, invasive coronary angiography clearly confirmed healing of the dissection on the day after the dissection. Notably, CTA 2 hours after IABP documented no significant stenosis in the proximal LADA and restoration of coronary flow in the first diagonal branch. Given the changes in both symptom and ECG, angiographic healing was achieved within 4.5 hours post-dissection. In a large case series in which repeat coronary angiography was performed, Saw, et al. showed that angiographic healing did not occur < 20 days post-dissection; however, angiographic healing was documented in all patients undergoing repeat angiography ≥ 26 days post-dissection. Considering the time requirement for angiographic healing in this case series, the angiographic healing in our patient was unexpectedly rapid.

Two potential mechanisms of SCAD have been proposed. The first is the intimal tear hypothesis, in which a primary disruption in the intimal-luminal interface creates an entry point for intramural hematoma accumulation inside the false lumen, leading to separation of the arterial wall. The second is the medial hemorrhage hypothesis, according to which a hemorrhage into the arterial wall is the primary mechanism, perhaps due to spontaneous rupture.
RAPID HEALING OF A SCAD DETECTED BY CTA

Figure 2. Computed tomography angiography (CTA) images. CTA 2 hours after intra-aortic balloon pumping revealed a false lumen with intramural hematoma between the proximal segment of the left anterior descending artery (LADA) and the left main coronary artery (LMCA) and between the proximal LADA and the first diagonal branch (white arrow in A and C), with no significant stenosis in the proximal LADA (black arrow in C) and restoration of coronary flow in the first diagonal branch (white arrow head in B and C). CTA on the 8th day post-dissection revealed the false lumen with intramural hematoma between the proximal LADA and the LMCA and between the proximal LADA and the first diagonal branch (white arrow in D). At the 3.5-month follow-up, CTA revealed disappearance of the false lumen, which indicated complete vessel healing of the spontaneous coronary artery dissection (SCAD) (E). At the 10-month follow-up, CTA revealed continuous healing of SCAD (F). Ao indicates Aorta; and PA, pulmonary artery.

from the increased density of the vasa vasorum. Accordingly, accurate and early diagnosis of SCAD is important because the management of SCAD is different from that of atherosclerotic disease. Intracoronary imaging provides complementary details to diagnose SCAD, which requires the presence of intramural hematoma or double-lumen. In a small series of 11 patients with SCAD confirmed by optimal coherence tomography, Alfonso, et al. reported that an intramural hematoma or double-lumen image was visualized in all patients; however, an intimal tear was identified in seven patients (64%). In our patient, emergency coronary angiography did not show arterial wall contrast staining with multiple radiolucent lumens, but it showed diffuse (> 20 mm) and smooth stenosis (type2 SCAD angiographic appearance), which required intracoronary imaging to confirm diagnosis. Although our patient did not perform intracoronary imaging, CTA 2 hours after IABP revealed an intramural hematoma image, and the diagnosis of SCAD could be made. Therefore, CTA was useful as an alternative to intracoronary imaging to diagnose SCAD in our patient.

Because of the low prevalence of this condition, there is no prospective randomized data available to guide the management of patients with SCAD. Management decisions are generally based on clinical presentation and angiographic characteristics. While it cannot be asserted that the IABP contributed to the rapid angiographic healing of this dissection because of the lack of any previous report on the use of IABP, its continuance was likely not deleterious. Conservative management in stable SCAD patients without evidence for ongoing ischemia has a good overall prognosis with a majority showing spontaneous healing of the dissection on follow-up angiography, which is concordant with our patient.

In this case, complete vessel healing with resorption of the intramural hematoma was not observed on the 8th
day post-dissection, but rather at the 3.5-month follow-up. In a small series of 34 patients with SCAD, Roura, et al. reported 24 patients that underwent CTA at a median of 121 days, and observed complete vessel healing in 20 patients (83%), which is consistent with the time for complete vessel healing in our patient.

Conclusion
To the best of our knowledge, this is the first case of a patient with SCAD in whom CTA was able to detect rapid healing of the dissection, 4.5 hours post-dissection. Given the minimally invasive nature of the technique, CTA may be the optimal imaging modality for the follow-up of patients with SCAD. Furthermore, CTA can be used to assess arterial healing after SCAD of larger proximal-mid coronary arteries, as in the present case.

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Disclosures
Conflicts of interest: No conflict of interest exists.

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