Journal of Cardiology Cases 20 (2019) 1–3

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

A case of spontaneous coronary artery dissection with early de novo recurrence

Takafumi Okura (MD)[a,*], Koji Takahashi (MD)[a], Tomoki Sakaue (MD)[a], Sou Ueda (MD)[b], Daijiro Enomoto (MD)[c], Daichi Yamamoto (MRT)[c], Daisuke Sasaki (MRT)[c], Taizo Kono (MRT)[c], Shigeki Uemura (MD)[c]

[a] Department of Community and Emergency Medicine, Ehime University Graduate School of Medicine, Ehime, Japan
[b] Ehime University Hospital, Ehime, Japan
[c] Yawatahama City General Hospital, Ehime, Japan

ARTICLE INFO

Article history:
Received 20 October 2018
Received in revised form 20 December 2018
Accepted 5 January 2019

Keywords:
Spontaneous coronary artery dissection
De novo recurrence
Hormonal therapy
Acute coronary syndrome

ABSTRACT

Spontaneous coronary artery dissection (SCAD) is a relatively rare cause of acute coronary syndrome compared with atherosclerotic plaque rupture and predominantly occurs in young women. SCAD is associated with various conditions, such as emotional stress, pregnancy, hormonal therapy, collagen diseases, fibromuscular dysplasia, or vasospasm. Long-term cardiovascular events are common including the recurrence of SCAD. We report a case of SCAD with de novo recurrence at only 4 days after the first attack.

<**Learning objectives:** Spontaneous coronary artery dissection (SCAD) is a relatively rare cause of acute coronary syndrome (ACS) compared with atherosclerotic plaque rupture, but if young to middle-aged women develop ACS, a high suspicion of SCAD is warranted. Recurrence of SCAD is common with 4- to 10-year follow-up. However, SCAD recurred early as in our case.>

Introduction

Spontaneous coronary artery dissection (SCAD) is a relatively rare cause of acute coronary syndrome (ACS) compared with atherosclerotic plaque rupture. It is now estimated that SCAD is the underlying cause of ACS in 1.7 to 4% of cases [1]. SCAD typically occurs in patients without conventional cardiovascular risk factors, and these patients are predominantly women (82–98% of SCAD cases) in their early 40s and 50s [2,3]. The prevalence of SCAD among women presenting with ACS is reported to be between 15 and 30% in those <60 years old [4]. Therefore, a high suspicion of SCAD is warranted in young to middle-aged women presenting with ACS.

SCAD patients have an in-hospital mortality rate <5%, and a rate of in-hospital major adverse cardiac events (MACE), including recurrent myocardial infarction and the need for urgent revascularization, that ranges from 5 to 10% [4]. However, MACE during long-term follow-up are frequent, ranging from 10 to 20%. Recurrence of SCAD is common, with intermediate-term recurrence rates of 15% within two years and longer-term recurrence rates as high as 7% at four to five years [5,6]. The definition of recurrent SCAD included both patients with extension of the first coronary artery dissection and those with de novo SCAD. De novo SCAD usually recurred 30 days beyond the first SCAD [5].

Here, we report a case of SCAD in a 38-year-old woman who presented with ACS. She had recurrence of de novo SCAD in only four days after the first ACS.

Case report

A 45-year-old woman was taken to the emergency room of our hospital at 11 pm due to severe chest pain that she had never experienced before. She had been treated with dienogest for endometriosis for 3 years. She had neither a habit of smoking nor a habit of drinking. Her symptoms disappeared by the time she arrived at 11:30 pm. A physical examination revealed a regular pulse of 65 beats/min and a blood pressure of 129/69 mmHg. The electrocardiogram (ECG) showed no remarkable abnormalities including right ventricular load. Echocardiography reveled hypokinesis of the posterior wall. Blood gas analysis showed hypoxemia

* Corresponding author at: Department of Community and Emergency Medicine, Ehime University Graduate School of Medicine, Shitsukawa, Toon City, Ehime, 791-0295, Japan.
E-mail address: okura@m.ehime-u.ac.jp (T. Okura).

https://doi.org/10.1016/j.jccase.2019.01.006
1878-5409/

(PO_2, 56.1 mmHg; PCO_2, 20.9 mmHg). A blood test showed increased white blood cells ($9.0 \times 10^3/\mu L$) but no elevation of creatine kinase (CK), CK-MB, troponin I, and D-dimer. Immediate coronary angiography was performed because of the suspicion of ACS. Coronary angiography revealed long and diffuse narrowing [Thrombolysis In Myocardial Infarction (TIMI) grade III flow] of the posterolateral (14-PL) branch in the left circumflex (LCX) coronary artery (Fig. 1A) even after 1.5 mg of isosorbide dinitrate given by intracoronary administration denying coronary artery spasm. This case was recognized as type 2 SCAD. The right coronary artery was normal. Because the imaging findings suggested SCAD with preserved flow, she was treated medically without coronary intervention or intravascular imaging. Dienogest was stopped, and she was treated with a vasodilator, statin, beta blocker, and antiplatelet agent. At 2 am on the fourth day of admission, she had the same severe chest pain as before. The ECG showed ST-segment elevation in leads I and aVL, and terminal T inversion in leads II, III, and aVF. Immediate coronary angiography was performed again. Angiography showed total occlusion at another 14 PL branch in the LCX (Fig. 1B). After guidewire (SION Blue and X-Treme XT-R, ASAHI INTECC, CO., LTD, Aichi, Japan) insertion, blood flow was restored in the 14-PL branch of the LCX (TIMI grade III flow/myocardial blush grade 3, Fig. 1C). This coronary angiography in the 14-PL branch of the LCX revealed a radiolucent lumen, indicating type 1 SCAD. There were no remarkable changes at the site of the previous SCAD. However, there was a new focal stenosis (TIMI grade III flow) in the second diagonal branch of the left anterior descending coronary artery (LAD), indicating type 3 SCAD (Fig. 2A) that was not seen in the first coronary angiography (Fig. 2B). Therefore, no further intervention was performed. The time taken for coronary angiography was 60 min. The radiation dose and contrast volume were 356 mGy and 75 mL, respectively. CK and CK-MB were elevated with a peak of 853 U/L and 561 U/L, respectively. During the 7 days preceding discharge after the second coronary angiography, she had no chest pain with ST-T changes, and her blood pressure control was good.

To estimate the complication of fibromuscular dysplasia (FMD), abdominal magnetic resonance angiography was performed, and

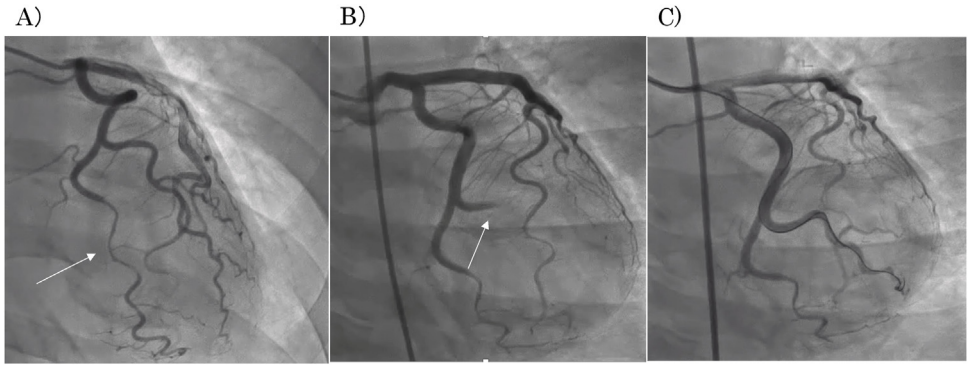

Fig. 1. (A) The first coronary angiography demonstrating diffuse narrowing of the posterolateral (14-PL) branch in the left circumflex (LCX) coronary artery (white arrow) and a type 2 spontaneous coronary artery dissection (SCAD) (anterior-posterior/cranial 30°). (B) The second coronary angiography demonstrating total occlusion at another 14-PL branch in the LCX (white arrow) (right anterior oblique 30°/cranial 30°). (C) After wiring in the 14-PL branch in the LCX, blood flow was restored. This coronary angiography in the 14-PL branch of the LCX revealed a radiolucent lumen (white arrow), indicating a type 1 SCAD (right anterior oblique 30°/cranial 30°).

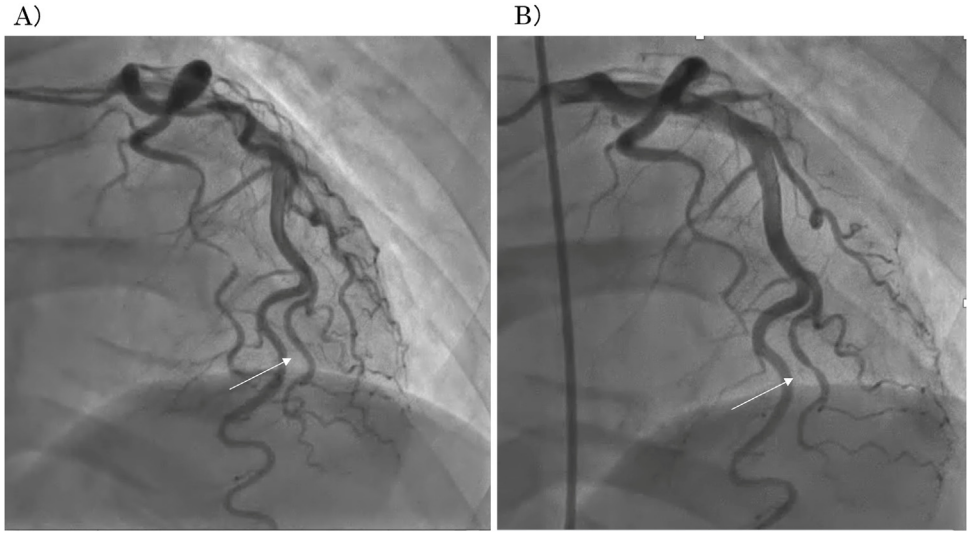

Fig. 2. The second coronary angiography demonstrating focal stenosis in a type 3 spontaneous coronary artery dissection in the second diagonal branch of the left anterior descending coronary artery (white arrow) (anterior-posterior/cranial 30°). (B) The first coronary angiography demonstrating a normal second diagonal branch of the left anterior descending coronary artery (white arrow) (anterior-posterior/cranial 30°).

no FMD lesion was detected. At six months after discharge, she remains free of chest pain.

Discussion

A strong association between SCAD and FMD has been reported. Saw et al. reported that 70–86% of patients with SCAD have noncoronary FMD [1]. However, in our patient, magnetic resonance angiography revealed no FMD.

Other potential predisposing conditions for SCAD include a history of systemic inflammatory disease, connective tissue disease, pregnancy, and hormonal therapy [1]. Our patient had been taking dienogest, an oral progestogen that binds to and activates the progesterone receptor. Progesterone excess is associated with elastic fiber disarray, loss of acid mucopolysaccharide ground substance, and impaired collagen synthesis [7,8]. Long-term exposure to exogenous progesterone is postulated to cause chronic impairment of arterial wall and changes in coronary arterial architecture, leading to be an important risk factor for SCAD [4]. Another possible risk factor for SCAD in this patient is vasospastic angina (VSA). VSA has been reported as the etiology of SCAD [3]. Although there was no evidence of VSA in our patient, she had chest pain twice during sleep. Therefore, we could not deny the possibility that coronary spasm caused SCAD.

SCAD is classified angiographically into three types: type 1, type 2, and type 3 [1]. Type 1 angiographic SCAD appears as the classic contrast dye staining of arterial wall with multiple radiolucent lumens, with or without the presence of dye hang-up or slow contrast clearing from the lumen. Type 2 angiographic SCAD appears as diffuse (typically 20–30 mm) and smooth narrowing that can vary in severity. Type 3 angiographic SCAD mimics atherosclerosis with focal or tubular stenosis. According to this classification, lesions in the 14-PL branch of the LCX, in the second diagonal branch of the LAD and in another 14-PL branch of the LCX in our case were type 2, type 3, and type 1 SCAD, respectively.

Recurrent SCAD has been reported in up to 30% of cases with 4- to 10-year follow-up in different series [2,3]. However, the definition of recurrent SCAD included both patients with extension of the first coronary artery dissection and those with de novo SCAD. Extension of dissection involved expansion of the intramural hematoma at either edge and usually occurred within 30 days of the first SCAD event [9]. On the other hand, de novo SCAD usually recurred 30 days beyond the first SCAD and was observed in 10–20% of cases [9]. De novo SCAD recurs in previously unaffected arteries 77%–100% of the time [6]. Eleid et al. reported that coronary artery tortuosity is highly prevalent in the SCAD population and is associated with recurrent SCAD [10]. Coronary artery tortuosity may be associated with systemic vasculopathy. In our case, coronary angiography in both the LAD and LCX showed strong tortuosity. Although we do not know the precise reason why de novo SCAD recurred only four days after the first attack in our case, one possibility is the hormonal therapy. Dienogest may have induced systemic vasculopathy in this patient.

A scientific statement from the American Heart Association (AHA) recommended a conservative (non-revascularization) treatment strategy for acute SCAD in low-risk patients without ongoing or recurrent chest pain, ischemia, left main dissection, or ventricular arrhythmias [6]. Iatrogenic catheter-induced coronary artery dissection by coronary angiography is reported to be increased in patients with SCAD [6]. Furthermore, intracoronary imaging procedures for the diagnosis of SCAD have potential risks, including extending the coronary dissection and catheter-induced occlusion of the true lumen. Therefore, the AHA recommends that intracoronary imaging be limited to the proximal segment of dissection [6]. Thus, we did not perform intravascular imaging to avoid further progression of dissection. Since intravascular imaging was not performed in this case, VSA cannot be completely denied even though the vasodilatation could not be obtained by intracoronary administration of isosorbide dinitrate.

Useful medications to prevent the recurrence of SCAD have not been fully elucidated. Only beta blocker therapy was reported to be able to reduce the recurrence of SCAD [1]. Our patient is now taking a beta blocker, aspirin, and a statin, and SCAD has not recurred after discharge.

Conflict of interest

None of the authors have a conflict of interest with regards to the contents of this article.

References

[1] Saw J, Humphries K, Aymong E, Sedlak T, Starovoytov A, Mancini GBJ. Spontaneous coronary artery dissection. Clinical outcomes and risk of recurrence. J Am Coll Cardiol 2017;70:1148–58.
[2] Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012;126:579–88.
[3] Saw J, Aymong E, Sedlak T, Buller CE, Starovoytov A, Ricci D, et al. Spontaneous coronary artery dissection: association with predisposing arteriopathies and precipitating stressors and cardiovascular outcomes. Circ Cardiovasc Interv 2014;7:645–55.
[4] Saw J, Mancini GB, Humphries K. Contemporary review on spontaneous coronary artery dissection. J Am Coll Cardiol 2016;68:297–312.
[5] Nakashima T, Noguchi T, Haruta S, Yamamoto Y, Oshima S, Nakao K, et al. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: a report from the angina pectoris-myocardial infarction multicenter investigators in Japan. Int J Cardiol 2016;207:341–8.
[6] Hayes SN, Kim ESH, Saw J, Adlam D, Arslanian-Engoren C, Economy KE, et al. Spontaneous coronary artery dissection: current state of the science. A scientific statement from the American Heart Association. Circulation 2018;137:e523–57.
[7] Madu EC, Kosinski DJ, Wilson WR, Burket MW, Fraker TD, Ansel GM. Two-vessel coronary artery dissection in the peripartum period. Case report and literature review. Angiology 1994;45:809–16.
[8] Manalo-Estrella P, Barker AE. Histopathologic findings in human aortic media associated with pregnancy. Arch Pathol 1967;83:336–41.
[9] Main A, Prakash R, Strarovoytov A, Sabbaghan A, Aymong E, Mancini GBJ, et al. Characteristic of extension and de novo recurrent spontaneous coronary artery dissection. EuroIntervention 2017;13:e1454–59.
[10] Eleid MF, Guddeti RR, Tweet MS, Lerman A, Singh M, Best PJ, et al. Coronary artery tortuosity in spontaneous coronary artery dissection. Angiographic characteristics and clinical implication. Circ Cardiovasc Interv 2014;7:656–62.