Clinical Case Report

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A case report of spontaneous coronary artery dissection and the role of intravascular imaging for accurate diagnosis and successful management

Chun-Chao Chen, MD[a], Ju-Chi Liu, MD, PhD[a,b], Li-Chin Sung, MD, PhD[a,b,*]

Abstract

Rationale: Acute myocardial infarction (AMI) secondary to spontaneous coronary artery dissection (SCAD) is a rare condition. SCAD can be underdiagnosed on a coronary angiography (CAG). Therefore, the application of intravascular imaging including intravascular ultrasound (IVUS) and optical coherence tomography (OCT) is crucial for ensuring an accurate diagnosis and treatment.

Patients concerns: A 72-year-old woman had an evolving AMI with ST elevation in the inferior leads (II, III, and aVF).

Diagnoses: An emergent CAG showed that a double lumen had developed in the middle portion of the left circumflex artery. An IVUS examination revealed a coronary artery dissection and intramural hematoma.

Interventions: First, the patient was treated with conservative management. We later placed a stent in response to the progression of the intramural hematoma observed during the IVUS follow-up.

Outcomes: The patient remained symptom free after discharge. CAG with OCT at the 1-year follow-up after stent implantation showed in-stent restenosis with dissection flap with residual false lumen at the proximal site of stent. We treated this lesion with another stent.

Lessons: From this case, we learned that in patients with AMI, SCAD should be considered as a possible diagnosis and that intravascular imaging tool can successfully guide clinical decision making and the treatment strategies.

Abbreviations: ACS = acute coronary syndrome, AMI = acute myocardial infarction, CAG = coronary angiography, ECG = electrocardiography, IVUS = intravascular ultrasound, LCX = left circumflex artery, OCT = optical coherence tomography, SCAD = spontaneous coronary artery dissection.

Keywords: acute myocardial infarction, intravascular imaging, intravascular ultrasound, optical coherence tomography, spontaneous coronary artery dissection

1. Introduction

An acute myocardial infarction (AMI) is usually caused by atherosclerotic plaque rupture with thrombus formation in the coronary artery lumen, but conditions such as a coronary spasm or aortic dissection can also cause an AMI. In rare circumstances, especially for patients without traditional atherosclerotic risk factors, spontaneous coronary artery dissection (SCAD) should also be considered.[1–3] Because a coronary angiography (CAG) might fail to show an obstruction, image modalities are important for ensuring accurate diagnosis and evaluation of severity.[4–8] We present a case of an AMI caused by SCAD, where intravascular imaging modality was decisive in choosing between treatment strategies.

2. Case report

A 72-year-old woman with a history of type 2 diabetes mellitus and hypertension presented to the emergency department after experiencing chest pain for several days. Electrocardiography (ECG) revealed sinus rhythm and no major changes in the ST-T segment. A chest x-ray showed no active lung lesions. Her laboratory tests showed normal levels of serum creatine phosphokinase at 274 U/L with MB fraction 24.26 ng/mL, and a slightly elevated troponin-I level at 0.67 ng/mL. Under the impression that she had a non-ST-elevation AMI, the patient was admitted to the intensive care unit for further management. Dual antiplatelet and anticoagulation therapy with low-molecular-weight heparin were prescribed. A follow-up serial cardiac enzyme measurement showed gradually decreasing levels. However, during the first night of hospitalization, she had a

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[a] Division of Cardiology, Department of Internal Medicine, Shuang Ho Hospital, [b] Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei City, Taiwan.

[*] Correspondence: Li-Chin Sung, Division of Cardiology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University, No. 291, Zhongzheng Rd., Zhonghe District, New Taipei City, 23561, Taiwan (e-mail: 10204@s.tmu.edu.tw).

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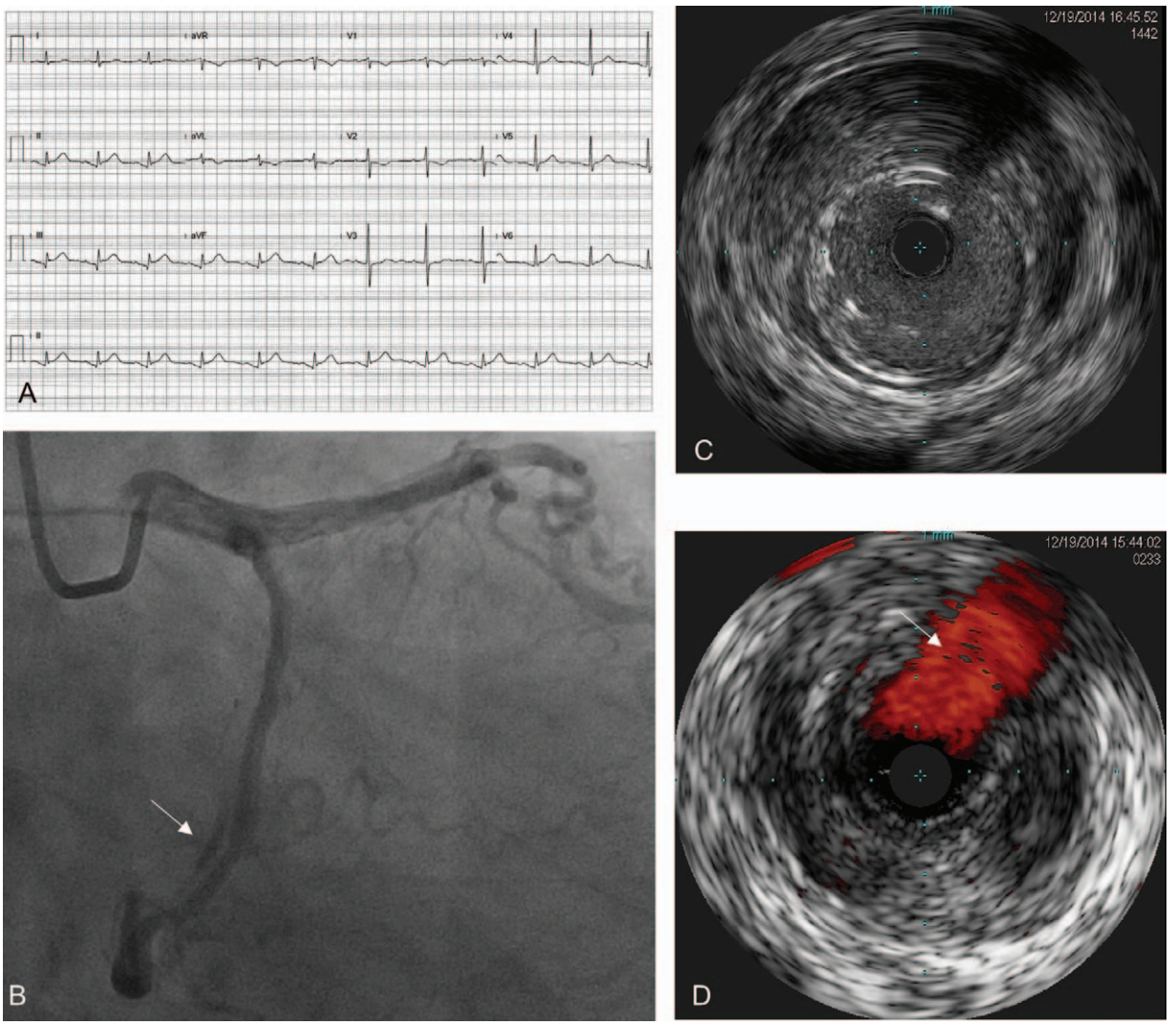

Figure 1. (A) An electrocardiogram recorded on the night of admission showed newly developed ST-segment elevation at leads II, III, and aVF. (B) Coronary angiography (RAO 30°, caudal 15°) showed a double lumen in the middle portion of the left circumflex coronary artery (white arrow). (C) Intravascular ultrasound (IVUS) revealed a false lumen with a maximal diameter of nearly 7 mm. (D) IVUS with ChromaFlo mode revealed intima tearing with unobstructed flow between the true and false lumen (white arrow).

sudden onset of severe chest pain and a repeat ECG revealed ST segment elevation at leads II, III, and aVF (Fig. 1A). An emergency CAG was performed and showed right coronary artery hypoplasia, a patent left anterior descending artery, and the appearance of a double lumen at the middle of the left circumflex artery (LCX) with thrombolysis in myocardial infarction II flow. Coronary perforation with dye extravasation at the LCX was highly suspected according to CAG (Fig. 1B). Intravascular ultrasound (IVUS) showed a massive intramural hematoma with true lumen compression. ChromaFlo modality demonstrates colorized blood flow, which is helpful in detecting blood flow outside the vessel. The Chromaflo mode showed intimal perforation with continuous flow between the true and false lumen, and the maximal false lumen was approximately 6 to 7 mm (Fig. 1C and D). A cardiovascular surgeon was consulted and he suggested surgical intervention if pericardial tamponade developed. We initially adopted a supportive and watchful waiting strategy because the hemodynamic status of the patient was stable and the degree of chest pain did not progress. Echocardiography was performed the next day, showing minimal pericardial effusion. After 3 days of hospitalization, coronary computed tomography angiography revealed no progression of the LCX hematoma. However, she still reported symptoms of intermittent angina. On the seventh day of hospitalization, CAG was performed and showed persistent narrowing of the lumen in the middle of the LCX. IVUS with ChromaFlo mode showed that the perforation site remained, and the dissection area was extended at the LCX with progression of the hematoma. One covered stent (GraftMaster 3.50 mm × 26 mm; Abbot Vascular, Abbott Park, IL) was deployed at the LCX. Post-stenting CAG and IVUS revealed minimal dye stasis in the peri-stent area and adequate apposition of the stent (Fig. 2A–C). Dual antiplatelet therapy was administered. Her general condition improved and she was discharged on the ninth day of hospitalization. After 1 year, she was admitted for scheduled follow-up CAG. The result showed in-stent restenosis at the proximal portion of the graft stent (Fig. 3A). IVUS revealed the residual hematoma located at the proximal site of stent (Fig. 3B). Optical coherence

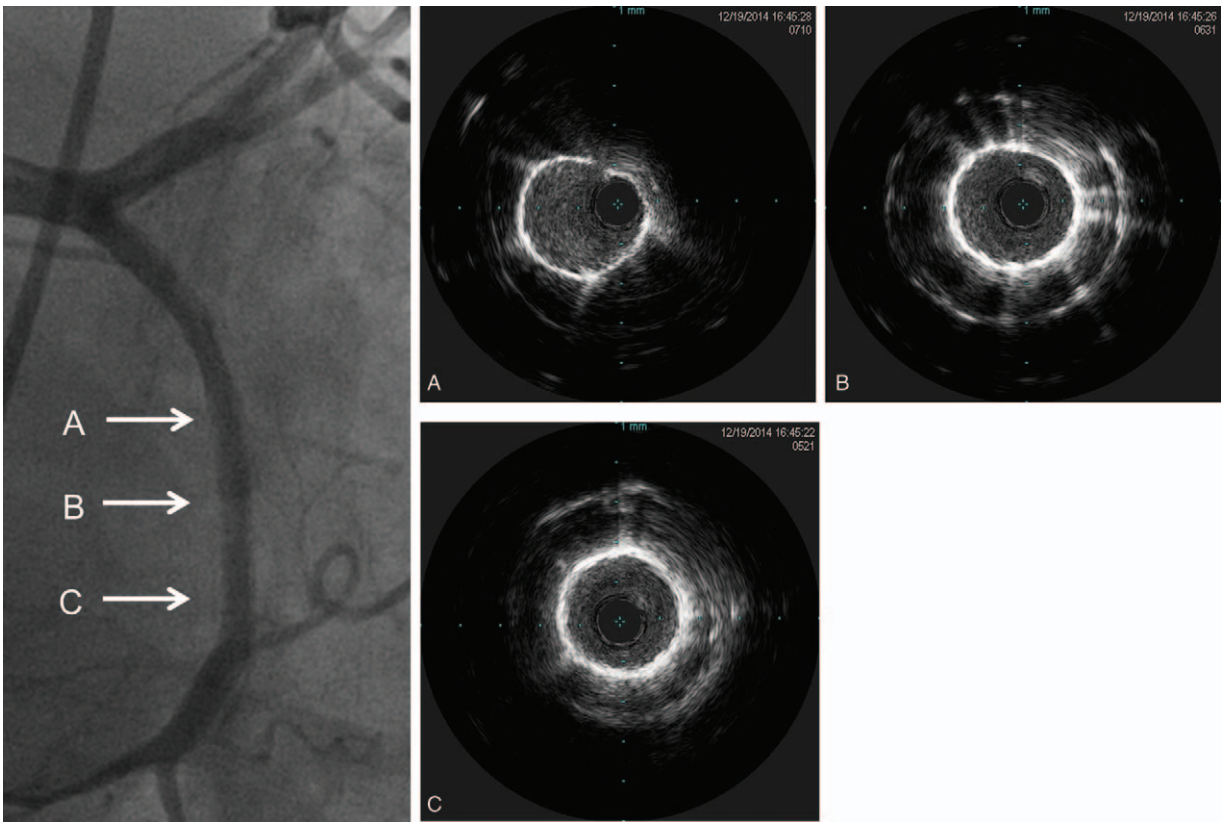

Figure 2. After the deployment of a graft stent, the double lumen was no longer visible in the left circumflex (LCX) coronary artery. (A–C) Intravascular ultrasound images at a corresponding point of the LCX coronary artery in the angiogram (white arrow) showed adequate apposition of the stent.

tomography (OCT) showed the dissection flap with residual false lumen at the proximal site of stent with a length of 8.9 mm extended to proximal portion of LCX (Fig. 3C). One short drug-eluting stent (Resolute integrity 4.0 × 12 mm; Medtronic Vascular, Santa Rosa, CA) was deployed at the proximal LCX with overlapping with the covered stent. Followed OCT showed resolution of the dissection flap, well stent apposition, and stent expansion (Fig. 3D and E). The patient had no more events and received regular outpatient department follow-up.

3. Discussion

AMI is diagnosed according to a combination of clinical symptoms, ECG findings, and positive cardiac enzyme results. The most common cause of ST elevation AMI is coronary atherosclerotic plaque rupture with acute thrombus formation. However, in some patients who have chest pain but do not present with coronary artery thrombus formation, such as those diagnosed with pericarditis, aortic dissection, coronary artery spasm, takotsubo cardiomyopathy, left ventricular aneurysm, and hypertrophic cardiomyopathy, ST elevation was observable in electrocardiograms.

SCAD is a rare but notable cause of acute coronary syndrome (ACS), especially in patients who present with typical symptoms but lack traditional cardiovascular risk factors. Studies have reported the prevalence of SCAD as approximately 0.07% to 1.1%,[1] but its true prevalence may be higher because of the difficulty of diagnosis. The mean age of patients is usually <50 years old and the majority are women.[1–3] Typical symptoms of SCAD include unstable angina, dyspnea, sudden cardiac arrest, ST segment changes in ECG, and elevation of cardiac biomarkers. Its etiology can be idiopathic or include any combination of fibromuscular dysplasia, pregnancy, connective tissue disease, systemic inflammation disease, and coronary artery spasms. Some precipitating stress events are also associated with SCAD, such as extreme stressors, emotion, and exercise.[1,2]

There are 3 types of SCAD. Contrast dye staining clearly shows the presence of a double lumen in the coronary angiography for Type 1 SCAD. However, in Type 2 SCAD, angiography images may reveal only diffuse narrowing of the lumen without the typical appearance of dissection. Moreover, in Type 3 SCAD, the lesion may mimic atherosclerosis and is often misdiagnosed.[2,9] Therefore, intravascular images such as IVUS and optical coherence tomography (OCT) images are crucial tools for accurately diagnosing these diseases, especially in younger women ACS patients.[5] Both IVUS and OCT have been reported as successful in diagnosing SCAD.[4,6] By employing these endovascular imaging tools, more characteristics of SCAD can be confirmed. Maehara et al[6] reported 5 cases of SCAD, none of which showed intima tearing with connection between the true and false lumen, which can explain the negative angiography results for dissection. OCT can provide clearer images of the dissection site than IVUS can, but when the vessel is large or the intramural hematoma is rich in red thrombi, the posterior structure cannot be seen clearly through OCT.[4] IVUS can provide real-time imaging and the ChromaFlo modality can identify the site where the true and false lumen are connected.

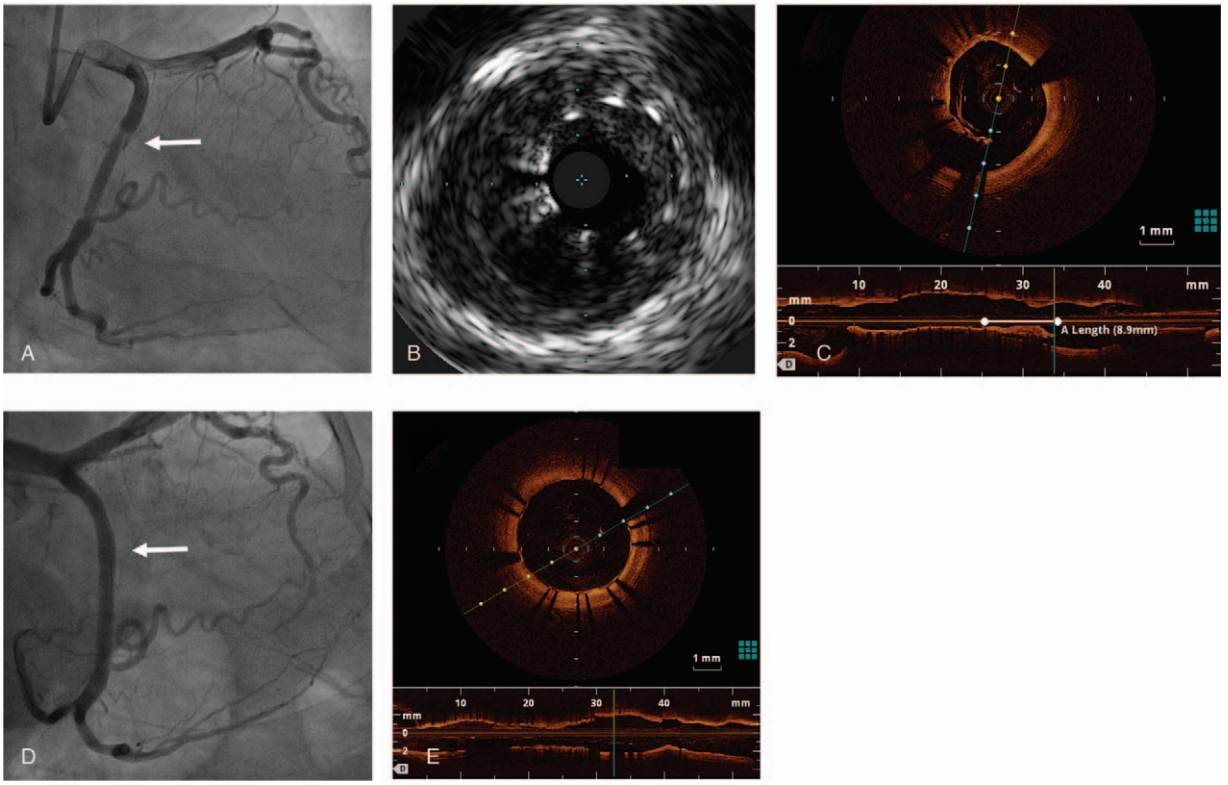

Figure 3. After 1 year of follow-up, coronary angiography showed in-stent restenosis located at stent proximal site. Intravascular imaging with (A) intravascular ultrasound and (B) optical coherence tomography showed the dissection flap with residual false lumen extended to proximal site of the left circumflex artery (C). After deployment of a drug-euling stent, the angiography showed totally resolution of the dissection flap (D) and optical coherence tomography showed the well apposition and expansion of the stent (E).

Because of the different limitations of IVUS and OCT, the combination usage OCT and IVUS could provide more comprehensive lesion information.[7] In addition, the advancement of the invasive and non-invasive image tools such as coronary computed tomography angiography, cardiac magnetic resonance could give accurate diagnosis, especially while the lesion is ambiguous.[8]

To our knowledge, there are no guidelines for managing SCAD; treatment currently depends on the clinical presentation of the patient. Revascularization therapy is suggested for patients with an unstable hemodynamic status, electrical instability such as ventricular tachycardia and fibrillation, recurrent episodes of angina, and left main CAD. Cutting balloon angioplasty has been reported to be successful in creating a connection between the true and false lumen in SCAD patients with ongoing chest pain and persistent ST-segment elevation.[10] In previous studies, it has not been clear whether bare metal stents and drug-eluting stents produce different outcomes.[3,11] Covered stents have been shown to be useful for treating iatrogenic coronary artery dissection or perforation during coronary angioplasty, yet this has not been reported for the treatment of SCAD. Our patient showed good angiographic results and an improvement of symptoms after the implantation of a covered stent. Stent implantation might be an appropriate treatment choice if the dissection site is clearly identified through IVUS with ChromaFlo. For SCAD patients with relatively stable clinical situations and preserved coronary flow, conservative treatment is suggested,[2] not only because of a high procedural failure rate but also because aggressive treatment does not reduce the future target vessel revascularization rate or recurrent SCAD.[11] Rogowski et al[12] reported on 64 patients with SCAD, of whom 56 received medical therapy; 31 of them received scheduled CAG 6 months after the initial event, and the results showed completely healed dissections. A lack of randomized clinical studies makes it unclear whether traditional drugs for ACS would benefit patients with SCAD. Low-dose aspirin is recommended, but dual antiplatelet therapy is also reasonable for patients with ACS.[1,2] In our patient, both dual antiplatelet and anticoagulant therapies were discontinued once a definite diagnosis of SCAD was obtained. After 7 days of watchful waiting, a follow-up angiography still revealed the extension of the dissection and progression of the intramural hematoma. This indicates that the risks of progressive worsened dissection and the benefits of resolving an overlying thrombus when using antiplatelet and anticoagulation agents should be carefully considered according to the needs of specific patients. Under the most conservative treatment, the prognosis for SCAD is generally good, but the reported risk of recurrent episodes of SCAD ranges from 13.1% to 17%.[2,3]

In conclusion, SCAD is an unusual cause of ACS. Diagnostic delay can result in unfavorable outcomes, rendering it crucial that cardiologists be aware of SCAD symptoms, especially when the initial CAG shows an insignificant result. Endovascular imaging tools enable accurately diagnosing the severity of the disease. The earlier the diagnosis, the sooner treatment can be initiated, yet the management strategy depend on the types of clinical manifestations. Conservative treatment can be applied to patients in a stable condition, but revascularization therapy should be

considered for patients with angina symptoms and potentially progressing intramural hematomas.

Author contributions

Conceptualization: ChunChao Chen, JuChi Liu, LiChin Sung.
Data curation: JuChi Liu, LiChin Sung.
Supervision: JuChi Liu.
Writing – original draft: ChunChao Chen.
Writing – review & editing: LiChin Sung.
Li-Chin Sung orcid: 0000-0003-1564-5592.

References

[1] Tweet MS, Gulati R, Hayes SN. What clinicians should know about spontaneous coronary artery dissection. Mayo Clin Proc 2015;90:1125–30.
[2] Yip A, Saw J. Spontaneous coronary artery dissection-A review. Cardiovasc Diagn Ther 2015;5:37–48.
[3] Tweet MS, Hayes SN, Pitta SR, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012;126:579–88.
[4] Alfonso F, Paulo M, Dutary J. Endovascular imaging of angiographically invisible spontaneous coronary artery dissection. JACC Cardiovasc Interv 2012;5:452–3.
[5] Daoulah A, Al Qahtani A, Mazen Malak M, et al. Role of IVUS in assessing spontaneous coronary dissection: a case report. J Tehran Heart Cent 2012;7:78–81.
[6] Maehara A, Mintz GS, Castagna MT, et al. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol 2002;89:466–8.
[7] Paulo M, Sandoval J, Lennie V, et al. Combined use of OCT and IVUS in spontaneous coronary artery dissection. JACC Cardiovasc Imaging 2013;6:830–2.
[8] Tweet MS, Gulati R, Williamson EE, et al. Multimodality imaging for spontaneous coronary artery dissection in women. JACC Cardiovasc Imaging 2016;9:436–50.
[9] Saw J, Mancini GB, Humphries K, et al. Angiographic appearance of spontaneous coronary artery dissection with intramural hematoma proven on intracoronary imaging. Catheter Cardiovasc Interv 2017;89:507.
[10] Yumoto K, Sasaki H, Aoki H, et al. Successful treatment of spontaneous coronary artery dissection with cutting balloon angioplasty as evaluated with optical coherence tomography. JACC Cardiovasc Interv 2014;7:817–9.
[11] Tweet MS, Eleid MF, Best PJ, et al. Spontaneous coronary artery dissection: revascularization versus conservative therapy. Circ Cardiovasc Interv 2014;7:777–86.
[12] Rogowski S, Maeder MT, Weilenmann D, et al. Spontaneous coronary artery dissection: angiographic follow-up and long-term clinical outcome in a predominantly medically treated population. Catheter Cardiovasc Interv 2017;89:59–68.