Background

Spontaneous coronary artery dissection (SCAD) is a unique cause of myocardial infarction, and optimal treatment should be selected according to the ischaemic condition. Patients with ongoing ischaemia or haemodynamic instability may require revascularization. Cutting balloon (CB) angioplasty has been acknowledged as an option for revascularization. However, few observations of the coronary artery conditions after CB angioplasty in SCAD patients have been reported. Here, we demonstrate two cases in which we evaluated the angiographic morphology of targeted coronary arteries in the chronic phase after CB angioplasty.

Case summary

Patient 1 was a 46-year-old woman who presented at our hospital with chest pain. Electrocardiography suggested acute coronary syndrome and coronary angiography was performed. The coronary angiography and intravascular ultrasound (IVUS) examinations revealed SCAD in the left anterior descending artery (LAD). Revascularization with CB angioplasty was successful. Follow-up coronary angiography 15 months after the angioplasty showed no visible stenosis in the LAD. Accordingly, the patient no longer needed antiplatelet therapy. Patient 2 was a 50-year-old woman who was transported to our hospital for ventricular tachycardia. Coronary angiography and IVUS revealed SCAD in the right coronary artery. Coronary flow was restored by CB angioplasty. Follow-up contrast-enhanced computed tomography angiography 36 months after angioplasty showed a healed appearance. Thus, she was able to discontinue antiplatelet therapy.

Discussion

Cutting balloon angioplasty may be a possible method to treat SCAD.

Keywords

Cutting balloon • Revascularization • Spontaneous coronary artery dissection • Case series

Learning points

• Treating spontaneous coronary artery dissection with cutting balloon angioplasty in an acute coronary syndrome may favourably affect healing of the lesion during long-term follow-up.
Introduction

Spontaneous coronary artery dissection (SCAD) is defined as epicardial coronary artery dissection not associated with atherosclerosis. Coronary artery obstruction in SCAD is caused by the formation of intramural haematoma (IMH) or intimal disruption. Spontaneous coronary artery dissection has been described as a common cause of acute coronary syndrome (ACS) in young women.\(^1\)\(^,\)\(^2\) Although SCAD lesions heal spontaneously, when percutaneous coronary intervention (PCI) is indicated for SCAD, the prognosis is often poor due to technical complications and progression of the dissection. Thus, a conservative strategy has been recommended for SCAD patients who have a clinically stable condition.\(^3\)\(^,\)\(^4\) In contrast, patients with ongoing ischaemia and/or haemodynamic instability may require revascularization. However, many complications of PCI have been reported in SCAD patients, and the short- and long-term outcomes are uncertain. Several PCI strategies are available,\(^3\)\(^,\)\(^4\)\(^,\)\(^5\) but they are not optimal for SCAD. Several reports have described the use of a cutting balloon (CB: Flextome, Boston Scientific, MA, USA) to fenestrate the tunica intima and drain the IMH as a novel treatment technique.\(^5\)\(^,\)^\(^6\)\(^,\)^\(^7\)\(^,\)^\(^8\) However, few reports have followed coronary artery appearance after CB angioplasty in SCAD patients. Here, we report an unusual interventional treatment of SCAD without stent implantation.

Timeline

| Time               | Event(s)                                                                 |
|--------------------|--------------------------------------------------------------------------|
| Patient 1 Admission| Hospitalized for chest pain.                                              |
|                    | Positive for high-sensitivity troponin T.                                 |
| 5 days after intervention | Revascularization with a cutting balloon (CB) performed. Coronary dissection evident. |
| 15 months after intervention | Follow-up coronary angiography showed that the coronary dissection healed. |
| Patient 2 Admission| Hospitalized for ventricular tachycardia.                                |
| Day 14 of hospitalization | Electrocardiogram showed ST-segment elevation on the inferior leads. |
| 6 and 12 months after intervention | Revascularization with a CB performed.                                  |
| 36 months after intervention | Discharged without chest pain.                                            |
|                    | Follow-up computed tomography coronary angiography (CTCA) showed coronary artery dissection. |

Case presentation

Patient 1

A 46-year-old woman presented to our hospital with chest pain. She had no past medical history. Her heart rate was 76 beats/min (regular rhythm); blood pressure was 126/64 mmHg. No heart murmur was heard at presentation. A 12-lead electrocardiogram showed T-wave inversion in the V3–6 leads, and echocardiographic examination revealed motion abnormality in the left ventricular anterior wall. In blood tests, high-sensitivity troponin-T was increased. Finally, she was diagnosed with ACS. Nitroglycerine oral spray did not relieve her chest pain. We performed emergency coronary angiography, which showed diffuse stenotic lesions with thrombolysis in myocardial infarction (TIMI) grade-2 in the middle to distal left anterior descending artery (LAD) (Figure 1A). After the prudent crossing of a 0.014-inch guidewire (SION-blue, ASAHI-Intech, Seto, Japan), intravascular ultrasound (IVUS) (Eagle-Eye Platinum, Philips Volcano, San Diego, CA, USA) confirmed an IMH causing critical stenosis (Figure 1B and C). Therefore, the patient was diagnosed with type 2 SCAD.\(^8\)

As her continuous chest pain, increased troponin-T level, and imaging revealed ongoing ischaemia, we performed PCI with CB angioplasty. A 2.0 × 10 mm CB was delivered easily to the target LAD lesion and carefully inflated to make fenestrations in the IMH. As a result, the compression of the true lumen by the IMH disappeared. Although coronary dissection was seen on coronary angiography, the coronary flow improved from TIMI grade 2 to grade 3 due to the communication between the IMH and true lumen (Figure 2A). Intravascular ultrasound also revealed that the subintimal IMH drained into the true lumen through the opening in the tunica intima (Figure 2B and C). Dual antplatelet therapy (DAPT) was administered immediately before PCI and continued for 3 months, followed by aspirin alone. After PCI, heparin at a dose of 15 000 units/day was administered for 24-h in the coronary care unit (CCU). There was no recurrence of chest pain in the follow-up period. However, due to gastrointestinal bleeding 15 months after PCI, coronary angiography was performed to determine if the aspirin could be discontinued. Coronary angiography showed good coronary flow and the coronary dissection observed after PCI was clearly healed (Figure 3). Based on these findings, the aspirin was discontinued. Thereafter, the gastrointestinal bleeding resolved and there was no recurrence of chest symptoms.

Patient 2

A 50-year-old woman presented to our hospital with ventricular tachycardia and loss of consciousness. She received an electric shock and returned to sinus rhythm. She had no past medical history. Her heart rate was 89 beats/min (regular rhythm); blood pressure was 98/
56 mmHg. No heart murmur was heard at presentation. Based on the ST-elevation in the inferior leads and akinesis in the left ventricular inferior wall on echocardiography, she was diagnosed with acute ST-elevation myocardial infarction. We performed coronary angiography, which revealed an occluded lesion in the distal right coronary artery (Figure 4A). We carefully passed the lesion with a floppy-wire as usual treatment in acute myocardial infarction. After crossing of a 0.014-inch guidewire (SION-blue), IVUS (Eagle-Eye Platinum) showed an extensive IMH. The patient was diagnosed with type-2 SCAD (Figure 4B and C). Because of unstable haemodynamics, we switched to PCI with a 2.0 x 10 mm CB that was gently inflated to generate re-entry on the IMH. The CB angioplasty successfully relieved the compression of the true lumen by the IMH, which promptly improved the coronary flow (Figure 5A). Although the coronary dissection remained, no additional treatment was performed because the dissection did not impair coronary flow and IVUS showed that the IMH was fenestrated and reduced (Figure 5B and C). After PCI, heparin was administered at a dose of 12 000 units/day for 24-h in the CCU. Consciousness was restored on the second day. Dual antiplatelet therapy was given immediately before PCI and continued for 3 months, followed by aspirin alone. She spent days without chest pain and underwent contrast-enhanced computed tomography coronary angiography 6, 12, and 36 months after PCI. The coronary dissection formed after PCI was observed for up to 12 months. However, it healed spontaneously by 36 months (Figure 6A–C). Based on this finding, the aspirin could be discontinued.

In both cases, we did not perform a 're-look' because there was no recurrence of chest pain after PCI, and because repeat coronary angiography may cause dissection progression.

Discussion

In the present report, we have described two SCAD patients who were successfully reperfused with CB angioplasty and had good coronary flow in the chronic phase. In both cases, successful coronary revascularization was achieved by reducing the IMH by fenestration of the tunica intima with a CB. Final coronary angiography on admission showed residual coronary dissection with preserved coronary flow and the procedure completed without stenting. In the chronic phase, these dissections had healed spontaneously and good coronary flow maintained. Finally, the patients could discontinue antiplatelet therapy without recurrence of chest pain. In SCAD patients, if restoring normal coronary flow in the absence of atherosclerotic coronary artery lesion is achieved using a CB, it would be the alternative method to treat this condition without stenting. The absence of atherosclerosis in patients with ACS may bring changes in the peri-procedural medical therapy after CB angioplasty, namely, the choice of single antiplatelet therapy or DAPT as well as the duration of DAPT in cases with DAPT. In patients with coronary vasospastic angina with non-significant atherosclerotic coronary sclerosis, taking aspirin did not affect the major adverse cardiovascular events.9 Conservative therapy is generally recommended for SCAD. However, PCI may be necessary in patients with progressive ischaemia and/or haemodynamic instability. Cutting balloon angioplasty is one of several PCI strategies given in the American Heart Association/American College of Cardiology (AHA/ACC) scientific statement for SCAD.10,11 However, CB angioplasty is often avoided due to the risk of coronary artery perforation and poor delivery of a stiff CB. However, in SCAD patients, a CB may be useful for creating...
a communication between the true and false lumens, decompress the IMH, and achieve reperfusion. To create a communication between the false and true lumens, conventional PCI balloons may not be useful. Stenting may cause IMH progression at both edges of the stent, often requiring the deployment of multiple stents and increasing the risk of in-stent restenosis. Although there is a risk of coronary artery perforation with CB angioplasty, several studies have reported that CBs can be used safely if the balloon size is appropriately selected to match the vessel diameter and dilated slowly to the indicated normal pressure. Our two cases had coronary dissections after CB angioplasty, but they did not impair the coronary flow throughout the clinical course. Similarly, previous reports have described maintained coronary flow, relieving myocardial ischaemia, even though PCI with CB resulted in a double-barrelled residual coronary dissection.

The novel findings of the current study were that coronary dissections caused by CB angioplasty did not reduce coronary flow, but they were spontaneously healed and the IMH absorbed more than 12 months after angioplasty. There have been several reports of healed coronary arteries after CB angioplasty in SCAD. In patients in whom conservative therapy was chosen, there have been reports that the IMH was spontaneously absorbed and the coronary dissection spontaneously repaired in several months.

The mechanism of SCAD development is considered to be bleeding within the vessel wall caused by arterial fragility and the formation of a tear in the intima of the coronary artery. We thought that the IMH may be more easily absorbed by the CB-induced communication between the true and false lumens. In addition, the maintenance of coronary flow by reducing the IMH may contribute to spontaneous healing of the coronary dissection.
Figure 4 Coronary angiography and intravascular ultrasound before intervention in Patient 2. (A) Urgent coronary angiography showed total occlusion in the distal of the right coronary artery (black arrows). (B) The proximal right coronary artery was normal on intravascular ultrasound. (C) An intramural haematoma (white arrow) was present within the vessel.

Figure 5 Coronary angiography and intravascular ultrasound after cutting balloon angioplasty in Patient 2. (A) Final coronary angiography revealed the presence of coronary dissection, but coronary flow was improved (black arrows). (B) Intravascular ultrasound showed a tear (white arrow) and the cutting balloon communicated between the true and false lumens. (C) The intravascular ultrasound showed a tear (white arrow) and the reduced haematoma.
We used DAPT in these patients in case coronary stenting may be needed, as they were diagnosed with ACS. Based on the guideline to prevent stent-thrombosis, we decided at that time to continued DAPT for 3 months for avoiding coronary re-occlusion due to the IMH and possible intraluminal haematoma, because after CB angioplasty, endothelium at the lesion should have been heavily damaged. Three months were a standard length using DAPT after stenting in Japan at that time. However, there is no consensus on whether antiplatelet therapy is necessary for SCAD patients, especially in the chronic phase. We discontinued antiplatelet therapy after confirming that the coronary lesions had healed. Because of the serious complications of antiplatelets, such as gastrointestinal bleeding and intracranial bleeding, stopping antiplatelet therapy should be beneficial for SCAD patients after the coronary lesion has healed in the follow-up process. A previous report recommended long-term use of aspirin because no definitive evidences and guidelines are available for aspirin use in the chronic phase, and the decision to continue or discontinue antiplatelet therapy should be made based not only on the coronary appearance at the SCAD lesion but also the patient’s bleeding risks. Future studies need to establish the necessity of antiplatelets for the chronic phase in SCAD patients.

Figure 6 Sequential contrast-enhanced computed tomography coronary angiography of the right coronary artery 6-, 12-, and 36 months after percutaneous coronary intervention in Patient 2. (A) Six months after coronary interventions, the coronary dissection remained at the distal right coronary artery (red arrows). It is particularly clear in the cross-sectional view (white arrow). (B) At 12 months, the coronary dissection remained at the distal right coronary artery (red arrows), which is apparent in the cross-sectional view (white arrow). (C) At 36 months, the coronary dissection was healed in the distal right coronary artery in the cross-sectional view (white arrow).
Conclusion

Our report revealed no visible coronary stenosis with the healing of SCAD in the chronic phase after CB angioplasty. Although SCAD is still a poorly understood disease, CB angioplasty may be useful in the coronary revascularization strategies for SCAD. In addition, our findings suggest that the CB angioplasty has the benefit of leading to discontinuation of antiplatelets in the chronic phase because no stent is implanted.

Lead author biography

Hiroshi Fujita is an interventional cardiologist and an Assistant Professor of Department of Cardiology Graduate School of Medical Sciences, Nagoya City University. He had interventional training in Toyohashi Heart Center, Toyohashi, Japan from 2005 to 2009.

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patients in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.

References

1. Mortensen KH, Thuesen L, Kristensen IB, Christensen EH. Spontaneous coronary artery dissection: a Western Denmark Heart Registry study. Catheter Cardiovasc Interv 2009;74:710–717.
2. Nakashima T, Naguchi 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;207:341–348.
3. Amsterdam EA, Wenger NK, Brindis RG, Casey DE Jr, Ganiats TG, Holmes DR Jr et al.; Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;130:2354–2394.
4. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F et al.; ESC Scientific Document Group. 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2016;37:267–315.
5. Ito T, Shintani Y, Ichihashi T, Fujita H, Ohse N. Non-atherosclerotic spontaneous coronary artery dissection revascularized by intravascular ultrasonography-guided fenestration with cutting balloon angioplasty. Cardiovasc Interv Ther 2017;32:241–243.
6. Yumoto K, Sasaki H, Aoki H, Kato K. Successful treatment of spontaneous coronary artery dissection with cutting balloon angioplasty as evaluated with optical coherence tomography. JACC Cardiovasc Interv 2014;7:817–819.
7. Sharma H, Vetrugno V, Khan SQ. Successful treatment of a spontaneous right coronary artery dissection with a 4-mm diameter cutting balloon: a case report. Eur Heart J Case Rep 2019;3:1–6.
8. Kaya E, Iwata H, Miyazaki S, Mattson PC, Takamura K, Nishiyama H et al. Successful coronary flow restoration by sent-free strategy using the pull-back method of cutting balloon in spontaneous coronary artery dissection. JQ Open 2019;4:213–215.
9. Ishii M, Kaikita K, Sato K, Yamanga K, Miyazaki T, Akasaka T et al. Impact of aspirin on the prognosis in patients with coronary spasm without significant atherosclerotic stenosis. Int J Cardiol 2016;220:328–332.
10. Hayes SH, Kim ESH, Saw J, Adlam D, Arslanian-Engoren C, Economy KE et al.; American Heart Association Council on Peripheral Vascular Disease; Council on Clinical Cardiology, Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; and Stroke Council. Spontaneous coronary artery dissection: current state of the science. A scientific statement from the American Heart Association. Circulation 2018;137:e523–e557.
11. Tweet MS, Eledj MF, Best Pj, Lennon Rj, Lerman A, Rihal CS et al. Spontaneous coronary artery dissection: revascularization versus conservative therapy. Circ Cardiovasc Interv 2014;7:777–786.
12. Huber MS, Mooney JF, Madison J, Mooney MR. Use of a morphologic classification to predict clinical outcome after dissection from coronary angioplasty. Am J Cardiol 1999;83:467–471.
13. Yip A, Saw J. Spontaneous coronary artery dissection: a review. Cardiovasc Diagn Ther 2015;5:37–48.