Usefulness of Cutting Balloon Angioplasty Prior to Stenting with Intravascular Ultrasound Imaging Guidance for Spontaneous Multi-vessel Coronary Artery Dissection Including the Left Main Coronary Artery

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Abstract:
We report a case of percutaneous coronary intervention for spontaneous multi-vessel coronary artery dissection including the left main coronary artery. This case suggests that intracoronary imaging is useful for understanding the complex anatomy and for choosing the appropriate management for effective revascularization. Furthermore, cutting balloon angioplasty prior to stenting is useful for preventing the longitudinal extension of the intramural hematoma and avoiding unnecessary stent implantation.

Key words: spontaneous coronary artery dissection, left main coronary artery, percutaneous coronary intervention, intravascular ultrasound, cutting balloon angioplasty

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Introduction
Spontaneous coronary artery dissection (SCAD) is gaining increasing attention as a cause of myocardial infarction, >90% of cases occur in young women (1, 2). SCAD is quite rare, but the natural history, presentation (clinical and angiographic), and etiology are heterogeneous (3). The diagnosis of SCAD is very important because the initial management of SCAD is different from atherosclerotic coronary artery disease or vasospastic angina. Although intracoronary imaging [intravascular ultrasound (IVUS) and optical coherence tomography (OCT)] is associated with the risk of extending the dissection, it is very useful for making a definitive diagnosis of SCAD. The appropriate initial management of SCAD has remained a matter of debate. Previous studies have reported poor outcomes of percutaneous coronary intervention (PCI) for SCAD (1, 2). However, patients with SCAD should be considered for revascularization with PCI in cases with vessel occlusion, ongoing ischemia, cardiogenic shock, or ventricular arrhythmia. In this study, we describe the case of a 42-year-old man who presented with cardio-pulmonary arrest due to spontaneous multi-vessel coronary artery dissection, which included the left main coronary artery (LMCA). The patient was treated with PCI. Furthermore, we discuss the nearly fatal case of SCAD, focusing on the diagnosis and treatment strategy.

Case Report
A 42-year-old man with a 10 pack-year smoking history walked into our outpatient clinic complaining of intermittent atypical chest pain. He had atypical chest pain after smoking for a week. He had no history of hypertension, diabetes mellitus, or dyslipidemia (hemoglobin A1c, 5.3%; low density lipoprotein cholesterol, 62 mg/dL; high density lipoprotein cholesterol, 41 mg/dL; triglyceride, 110 mg/dL). A physical examination revealed no abnormalities with a blood pressure of 130/80 mmHg, and a regular heart rate (64 bpm). Initial electrocardiography (ECG) was unremarkable. While sitting in the waiting room, he developed cardio-pulmonary arrest. Repeat ECG revealed ST-segment elevation in the anterolateral leads (Fig. 1). He was transferred from the outpatient clinic to the catheter lab with emergent coronary angiography (CAG). Emergent CAG revealed the total occlusion of the left anterior descending artery (LAD) with thrombolysis...
in myocardial infarction (TIMI) flow grade 0 and severe stenosis of the left circumflex artery (LCX) with TIMI flow grade 1 (Fig. 2). The right coronary artery appeared normal. Intracoronary nitrates were administered based on the suspicion of vasospasm; this resulted in the partial improvement of the LAD flow to TIMI flow grade 1. To confirm the cause of severe stenosis, an IVUS examination was performed. IVUS demonstrated intramural hematoma (IMH) from the LMCA to both the LAD and LCX, consistent with SCAD (Fig. 3). No significant atheroma was visible within the vessel on IVUS. Delayed flow, persistent chest pain, and hemodynamic instability did not allow a conservative treatment strategy. Cutting balloon (Flextome, Boston Scientific, Natick, USA) angioplasty was used to fenestrate the dissection flap of both the LAD and LCX. We selected the size of cutting balloon and stent based on the lumen diameter and the external elastic membrane diameter (obtained by IVUS). A 3.0 mm cutting balloon was dilated 8 times at 6 atm in the mid LCX. It was not clear whether the dissection flap could be fenestrated after cutting balloon dilatation by IVUS, the coronary flow in the LCX recovered to TIMI flow grade 3. Subsequently, a 3.5 mm cutting balloon was dilated 10 times at 6 atm in the proximal-to-mid LAD. However, the coronary flow remained TIMI flow grade 1 and dissection newly occurred in the proximal LAD. Then, two drug-eluting stents (4.0 mm) were implanted from the LMCA to the mid LAD. Final CAG showed TIMI flow grade 3 in both the LAD and LCX (Fig. 4). ECG after PCI revealed ST-segment resolution in the antero-lateral leads (Fig. 5). The peak CK, CK-MB, and Troponin I values on day 2 were 3,203 U/L, 310.5 U/L, and 81.7 ng/mL, respectively. The post-procedural course was uneventful without
Figure 3. Angiography after the infusion of nitrates and IVUS images. (A) Caudal projection showing severe stenosis in the LCX after the infusion of nitrates. (B) An IVUS image of the left main coronary artery showing intramural hematoma (asterisk). (C) (D) IVUS images of the LCX showing intramural hematoma (asterisk) compressing the true lumen. (E) Cranial projection showing severe stenosis in the LAD after the infusion of nitrates. (F) (G) IVUS images of the LAD showing intramural hematoma (asterisk) compressing the true lumen. IVUS: intravascular ultrasound, LCX: left circumflex artery, LAD: left anterior descending artery.

Figure 4. The final angiogram and IVUS images of the LCX after cutting balloon angioplasty and the LAD after stent implantation. (A) The final angiogram showing TIMI flow grade 3 in the LCX. (B) An IVUS image of the left main coronary artery showing the well-expanded stent without evidence of malapposition. (C) (D) IVUS images of the LCX showed that the intramural hematoma (asterisk) remained, but that it did not compress the true lumen. (E) The final angiogram showing TIMI flow grade 3 in the LAD. (F) (G) An IVUS image of the LAD showing a well-expanded stent without evidence of malapposition. IVUS: Intravascular ultrasound, LCX: left circumflex artery, LAD: left anterior descending artery, TIMI: thrombolysis in myocardial infarction.
Figure 5. An electrocardiogram obtained after percutaneous coronary intervention revealed ST-segment resolution in the I, II, aVL, and V3-6 leads.

recurrent ischemia. Follow-up CAG at 6 months revealed the good patency of both the LAD and LCX.

Discussion

SCAD is gaining increasing attention as a cause of myocardial infarction. The etiology of SCAD appears to be multifactorial. Previous studies have reported many potential predisposing [e.g., fibromuscular dysplasia (FMD), pregnancy-related, connective tissue disorder, systemic inflammatory disease, hormonal therapy, coronary artery spasm or idiopathic] and precipitating factors [e.g., intense exercises (isometric or aerobic activities) or intense emotional stress] for SCAD (3). In this case, however, there were no predisposing or precipitating factors for SCAD.

Although SCAD is an uncommon but important cause of acute coronary syndrome, there are no established guidelines for the diagnosis and management of SCAD. In the present case, we learned two important lessons. First, we should not hesitate in performing intracoronary imaging if the diagnosis of SCAD is unclear on CAG. A recent report suggested three distinct angiographic appearances and patterns of SCAD: type 1 (evident arterial wall stain), type 2 (diffuse stenosis of varying severity) and type 3 (mimic atherosclerosis) (4). In the absence of a type 1 pattern on CAG, the diagnosis of SCAD could be very difficult and we should also be aware that SCAD can easily be mistaken for atherosclerotic changes, which enabled us to diagnose SCAD. With regard to SCAD imaging, OCT is superior to IVUS for visualizing intimal tears, false lumens, and IMH (3). However, because OCT imaging requires the intracoronary injection of contrast agent, which might expand the dissection, we believe that IVUS is more useful for imaging in cases of SCAD.

Secondly, if rescue PCI must be performed for SCAD, the performance of cutting balloon angioplasty to fenestrate the dissection flap might be useful for preventing the longitudinal extension of the IMH and for avoiding unnecessary stent implantation. Previous studies have reported that rescue PCI for SCAD is associated with poor outcomes and that it is still challenging to perform (1, 2, 5). The longitudinal extension of the IMH with stenting is the main limitation of stenting in SCAD. There have been recent reports of the successful performance of cutting/scoring balloon angioplasty to decompress the hematoma in order to avoid longitudinal extension (6, 7). In our case, IMH was observed extensively from the LMCA to both the LAD and LCX. We initially tried to fenestrate the dissection flap with a cutting balloon. As a result, we were able to avoid complex stenting in the LMCA bifurcation.

In conclusion, SCAD is an uncommon but important cause of acute coronary syndrome. Emergent multi-vessel PCI due to SCAD of LMCA is still challenging because of its unusual anatomy and because the response to conventional PCI techniques is difficult to predict. Our case illustrated that intracoronary imaging is useful for understanding the complex anatomy and for choosing appropriate management with effective revascularization. Furthermore, cutting balloon angioplasty prior to stenting is useful for preventing the longitudinal extension of the IMH and avoiding unnecessary stent implantation.
The authors state that they have no Conflict of Interest (COI).

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