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Case Report

Percutaneous coronary intervention strategy for acute coronary syndrome caused by spontaneous coronary artery dissection for relieving ongoing ischemia—Case series and literature review

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ABSTRACT

Although spontaneous coronary artery dissection (SCAD) is one of the causes of acute coronary syndrome (ACS) or sudden cardiac death, its standard management, especially primary percutaneous coronary intervention (PCI) in ACS patients with ongoing ischemia, has not been established. We experienced three ACS patients with SCAD who were treated with a different strategy of primary PCI. Each PCI strategy led to different clinical and procedural results. We describe here such PCI strategies and results, and also discuss the literature regarding primary PCI strategies for SCAD-induced ACS patients with ongoing ischemia.

<**Learning objective:** SCAD is a cause of ACS. However, the treatment strategy of primary PCI for SCAD has not been fully investigated. We used different PCI strategies for three SCAD patients with ongoing ischemia. Our case series suggested that plain old balloon angioplasty is an acceptable option to avoid coronary stenting because the majority of patients were young menstruating women. Coronary vasospasm might be associated with SCAD. Treatment with vasodilators could be a potential pharmacological option for avoiding recurrence of SCAD.>

Introduction

Although spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS), there are many reports on the clinical significance of this disease entity in ACS [1–3]. In terms of the pathophysiology of SCAD, several potential disease conditions (connective tissue disease, pregnancy, and coronary vasospasm) have been proposed [4,5]. Recently, fibromuscular dysplasia was also speculated as being associated with SCAD [6]. However, in contrast to the well-known, above-mentioned pathophysiology, the treatment strategy of SCAD has not been clearly established. Conservative therapy is one of the options for management of SCAD [2]. However, when ongoing ischemia is present, some type of alternative strategy to revascularize the ischemia-inducible dissected coronary artery needs to be formulated [7]. Recently, we experienced four episodes of SCAD in three patients and used different treatment strategies. We report here the different treatment strategies with their clinical and procedural outcomes, and also discuss the relevant literature.

Case series

Case 1

A 38-year-old woman was referred to our institution for severe chest pain, which was suggestive of acute aortic dissection. She was not pregnant and did not have any coronary risk factors. An initial electrocardiogram (ECG) showed ST-segment elevation at

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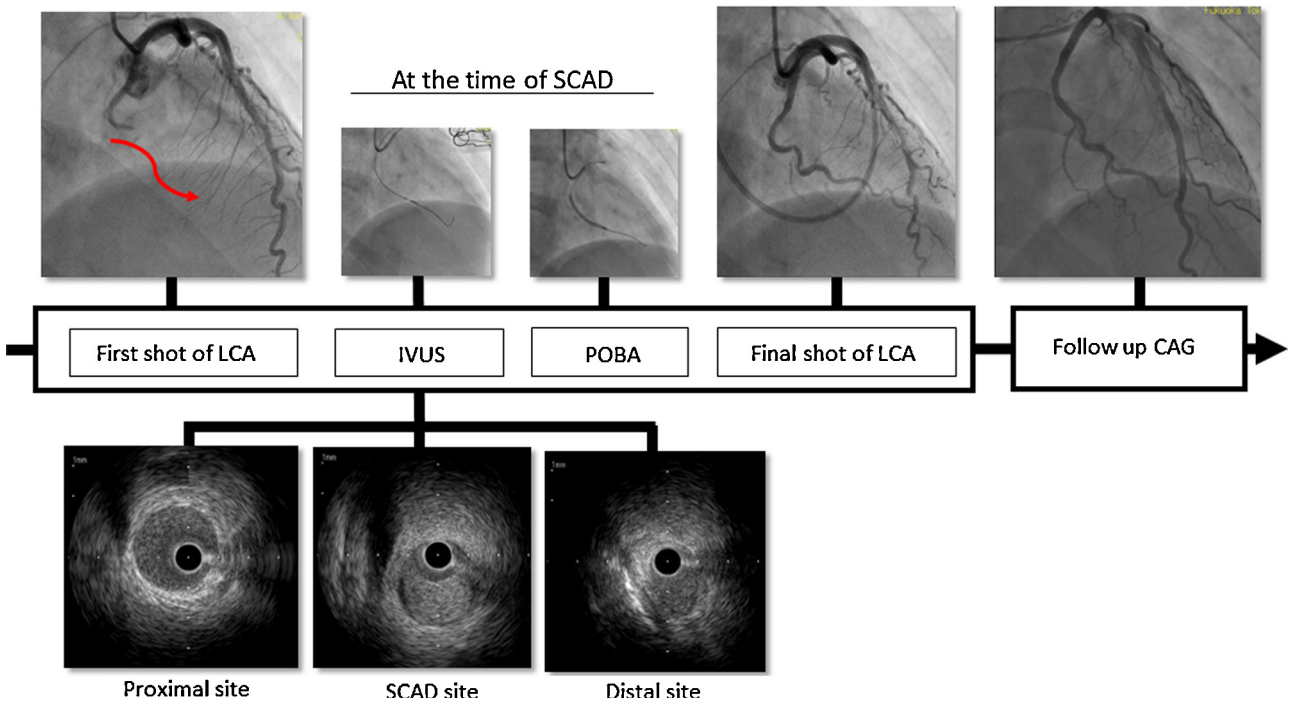

Fig. 1. CAG and IVUS images in Case 1. SCAD occurred in the distal left circumflex artery (red line). CAG, coronary angiography; IVUS, intravascular ultrasound; SCAD, spontaneous coronary artery dissection; LCA, left coronary artery; POBA, plain old balloon angioplasty.

the inferior leads, and her cardiac enzymes were not elevated. We performed emergency coronary angiography (CAG), and the distal site of the large left circumflex coronary artery (LCX) was occluded without thrombus (Fig. 1). We carefully crossed the lesion with Xtreme XT-A guidewire (Asahi Intecc Co., Nagoya, Japan) using a microcatheter (FineCross MG, Terumo Corp., Tokyo, Japan). After successful guidewire crossing, intravascular ultrasound (IVUS) provided a definitive diagnosis of SCAD, visualizing an intramural hematoma and its compression of the true lumen. Therefore, we used balloon angioplasty with a long-length, semi-compliant, small-sized balloon (Sprinter Legend 2.0 mm × 30 mm, Medtronic Japan Co., Tokyo, Japan) to prevent longitudinal shift of the hematoma. We also inserted an intra-aortic balloon pump (IABP) to maintain sufficient coronary blood flow of the true lumen. Waiting 30 min after ballooning, final CAG confirmed maintenance of Thrombolysis in Myocardial Infarction (TIMI) grade 3 antegrade flow and the absence of progression of the SCAD segment. Cardiac enzymes peaked, with creatine kinase (CK)/CK-MB isoenzyme (CK-MB) levels of 1910/178 IU/L. No SCAD-prone disease conditions (e.g. connective tissue disease) were detected by subsequent systemic work-up. She was discharged 13 days after urgent hospitalization without any major complications. A routinely performed 6-month follow-up CAG showed a slightly persisting dissected segment at the distal LCX. She was conservatively managed without any additional intervention because of the absence of ischemia as shown by stress myocardial perfusion imaging.

Case 2

A 33-year-old woman, who had no coronary risk factors, suddenly felt chest pain and was transferred to our institution. An initial ECG showed anterior ST-segment elevation and an ultrasonic cardiogram showed akinesis in the anterior wall. She was diagnosed with anterior acute myocardial infarction and underwent emergency CAG. Before CAG, ventricular fibrillation occurred and she was defibrillated by direct-current cardioversion. CAG showed 99% narrowing at the middle segment of the left anterior descending coronary artery (LAD). We performed percutaneous coronary intervention (PCI) under mechanical support of an IABP (Fig. 2). We carefully crossed Sion Blue guidewire (Asahi Intecc Co.) into the true lumen and performed IVUS imaging. IVUS clearly showed the absence of plaques and the presence of an intramural hematoma at the SCAD segment and its compression of the true lumen (Fig. 2). After ballooning (Ryujin 2.5 mm × 30 mm, Terumo Corp.), a bare metal stent (Integrity 3.0 mm × 30 mm, Medtronic Japan Co.) was implanted in the mid-LAD because of difficulty in maintaining TIMI grade 3 flow only by balloon angioplasty. Post-stent CAG and IVUS showed that the hematoma was extended proximally across the proximal edge of the stent. An additional short, bare metal stent (Driver 3.0 mm × 15 mm, Medtronic Japan Co.) was then implanted from just the proximal LAD to the distal stent. After post-dilatation of the proximal stent, we confirmed that the hematoma was not extended, and finished the PCI procedure. Peak CK/CK-MB levels were 2333/185 IU/L. Three months after discharge, a follow-up CAG showed the absence of in-stent restenosis, and an ergonovine provocation test revealed vasoconstriction of the distal LAD. Therefore, we added nitrate to prevent coronary spasms.

Case 3

A 56-year-old woman, who had no coronary risk factors, was referred to our institution for evaluation of chest pain. An initial ECG showed anterior ST-segment elevation and T-wave inversion. We diagnosed her with anterior ST-elevation myocardial infarction and performed emergency CAG. The middle segment of the LAD had diffuse narrowing and the distal part was occluded (Fig. 3). We attempted crossing the lesion with the Xtreme XT-A guidewire (Asahi Intecc Co.) and Corsair (Kaneka Corp., Osaka, Japan) microcatheter. Despite the strong support system, we could not cross the lesion. Therefore, we used the IVUS system to evaluate the