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Optical Coherence Tomography Findings of Provoked Coronary Spasms in Patients with Atherosclerotic Vessels

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Abstract

The aim of this case report was to show the dynamics of the wall structure during the spasm. In 2 patients who underwent first-generation drug-eluting stent implantation, optical coherence tomography (OCT) showed detailed cross-sectional structure of the coronary wall. Segments distal to the implanted stents were observed during spasm provocation by acetylcholine. The patient in case 1, an 82-year-old man, underwent Taxus stent implantation (3.0 × 32 mm and 3.0 × 12 mm) for in-stent restenosis and a new lesion 20 months after bare-metal stent implantation in the distal right coronary artery. Five months later, angina pectoris recurred, and follow-up coronary angiography revealed severe coronary spasm distal to the stents. The patient in case 2, a 69-year-old man, underwent Cypher stent (3 × 28 mm) deployment for a new lesion in the mid-left anterior descending artery. The Cypher stent overlapped with a previously implanted bare-metal stent at the proximal site. Nine months later, he again experienced chest oppression; follow-up coronary angiography revealed severe coronary spasm provocation distal to the stents. In both the cases, as observed using OCT, the vessel diameter decreased, the lumen narrowed, and the intimal and medial layers thickened during the spasm, even in segments with atherosclerosis. Furthermore, the lumen border appeared star shaped.

Keywords

Drug-eluting stent; Vasospastic angina; Coronary endothelial dysfunction; Acetylcholine; Optical coherence tomography

Introduction

Optical coherence tomography (OCT) enables evaluation of detailed cross-sectional vascular structure owing to its high resolution, which is approximately 10 times higher than the resolution of intravascular ultrasound [1]. OCT is unique, in that it allows individual assessment of the intimal and medial layers. Coronary spasm may be diagnosed using coronary angiography during occasional attacks

or during an acetylcholine (ACh) provocation test[2]. However, the dynamics of the wall structure during spasm has not been well elucidated. Morikawa et al[3] and Tanaka et al[4] reported OCT findings of provoked coronary spasm in patients with vasospastic angina. However, these studies examined arteries without any remarkable atherosclerotic plaque. In this case study, we obtained OCT findings of provoked spasm in an atherosclerotic artery in which drug-eluting stent implantation was performed at the chronic stage.

Case Presentation: Case 1

An 82-year-old man underwent bare-metal stent (BMS; Liberta 3.0 × 20 mm) implantation for thrombotic occlusion in the distal right coronary artery (RCA). Angina pectoris recurred 20 months later, and coronary angiography revealed in-stent restenosis (ISR) and a new lesion in the mid-RCA. He underwent Taxus stent implantation (3.0 × 32 mm and 3.0 × 12 mm) with overlap for the ISR and the lesion. Five months later, he experienced chest oppression. Follow-up coronary angiography revealed no ISR or a new lesion. However, severe coronary spasm provocation was observed in the RCA distal to the stents (Figure 1a) after intracoronary administration of 20 μg of acetylcholine (ACh) for 30 seconds. OCT was performed using the M2 OCT system (Light Lab Imaging, Westford, MA) and a Helios occlusion balloon catheter (Light Lab Imaging) during an ACh provocation test (Figure 1b), with the OCT system located in the atrioventricular branch. OCT revealed homogeneous neointimal hyperplasia at the proximal site and heterogeneous neointimal hyperplasia at the distal site; in addition, it revealed a peri-strut low-intensity area, but no stent malapposition or delayed healing (Figure 1b, lower column). Spasm could be detected at a site immediately distal to the stents (Figure 1b, upper column). During the spasm, the vessel diameter decreased, the lumen narrowed, and the intimal and medial layers thickened. The maximal thicknesses of the intimal and medial layers were 0.64 and 0.24 mm, respectively, during the spasm, and these values decreased to 0.34 and 0.11 mm, respectively, after isosorbide dinitrate (ISDN) administration (Figure 1b, upper column). The lumen border appeared star shaped.

Case 2

A 69-year-old man with diabetes mellitus and dyslipidemia underwent BMS implantation in the proximal left anterior descending artery (LAD) for acute myocardial infarction. A Cypher stent (3 × 28 mm) was deployed for a new lesion in the mid-LAD 6 years later. Nine months later, he again experienced chest oppression; follow-up coronary angiography did not reveal ISR or a new lesion. However, severe coronary spasm provocation was observed in the LAD distal to the stents, after intracoronary administration of 50 μg of ACh for 30 seconds (Figure 2a). OCT (Figure 2b lower column) revealed thin and homogeneous neointimal hyperplasia on the stent struts with peri-strut low-intensity areas at both the proximal and distal sites. Moreover, the distal reference segment showed eccentric, homogeneous, and high-intensity hyperplasia. The coronary artery was found to be narrowed, with intimal and medial thickening (0.9 and 0.13 mm, respectively) during spasm provocation; the intimal and medial thickening decreased to 0.78 and 0.08 mm, respectively, after ISDN administration (Figure 2b, upper column). The lumen border appeared star shaped. A calcium-channel blocker was prescribed for the treatment of the coronary spasm [5]. The symptoms eventually subsided in both the cases.

Discussion

In both the cases, OCT showed detailed cross-sectional structure of the intimal and medial layers

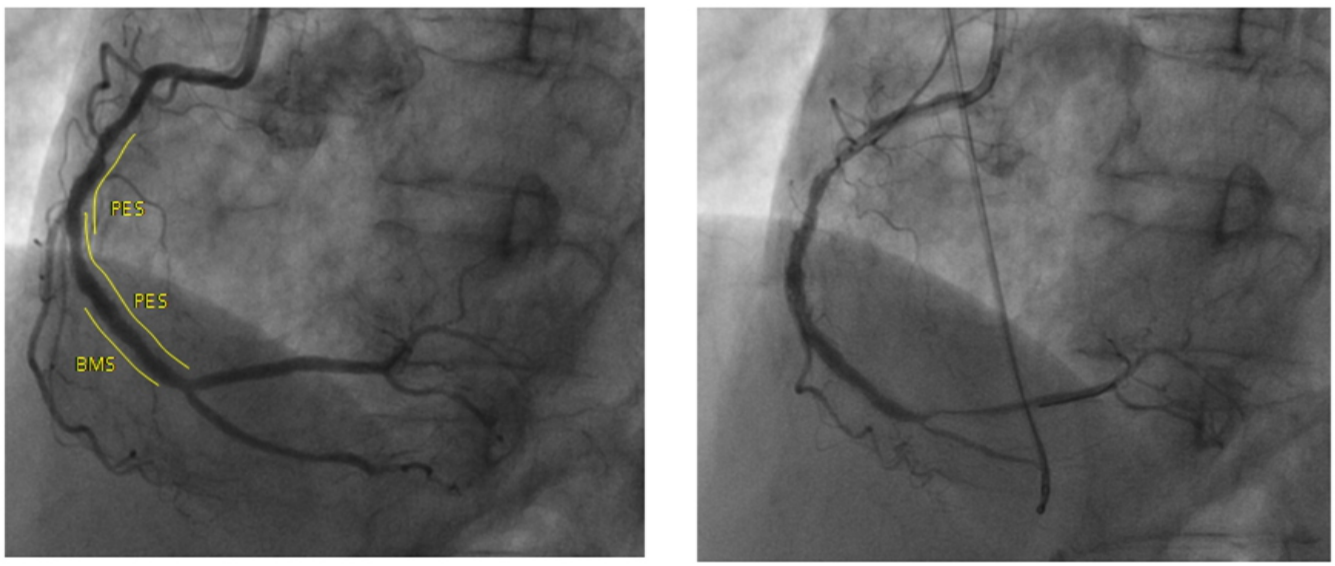
of the atheromatous coronary artery, distal to the implanted drug-eluting stents during spasm provocation. To the best of our knowledge, ours is the first case study wherein OCT findings of the spasm in vessels with an atherosclerotic plaque were obtained.

Drug-eluting stent (DES) has been reported to cause endothelial dysfunction[2,6-8], which might be related to stent thrombosis [9]. Delayed healing or poor neointimal coverage with DES has been reported to cause coronary endothelial dysfunction [10-12]. However, in our cases, neointimal hyperplasia was observed on the struts without delayed healing or malapposition. Thus, neointimal hyperplasia after DES implantation might not always guarantee preservation of the endothelial function. Further studies are required to elucidate this topic. Because we could not perform an ACh provocation test before stent implantation, we did not know whether spasm provocation was related to DES in our cases. The spasm in case 1 might be caused by the infarct-related artery [13,14]. The remarkable narrowing of the atheromatous vessel during the spasm in our cases confirmed that the spasm may cause plaque rupture [15] through compression of the vulnerable plaque.

Because of the lack of an appropriate imaging method for the individual evaluation of the intimal and medial layers, the mechanism by which each arterial layer is altered in accordance with the drastic luminal narrowing that occurs at the vasospasm lesion has not been well elucidated. During the ACh-induced spasm, the size of the lumen and total vascular areas remarkably decreased, whereas the intimal area did not alter in comparison with the area during complete vasodilatation. The luminal surface of the intima formed a markedly wavy configuration during the spasm[3]. Tanaka et al [4] showed that the medial thickness increased because of medial contraction and facilitated intimal gathering without alteration of the intimal area during vasospasm in the coronary artery. Intimal gathering was defined as folding or gathering of the intima, resulting in multiple kinks in the luminal contour that resolved after nitroglycerine administration. These studies examined patients without a remarkable atheroscrelotic plaque. In our study, even in the case of the atheromatous arteries, the vessel diameter decreased, the lumen narrowed, and the intimal and medial layers thickened during the spasm. The lumen appeared star shaped, but intimal folding or gathering was not observed. Although spasm might be related to coronary endothelial dysfunction, morphological endothelial abnormalities could not be detected, mainly because of the associated atherosclerotic alteration.

Figures

a



baseline after ACh administration

b

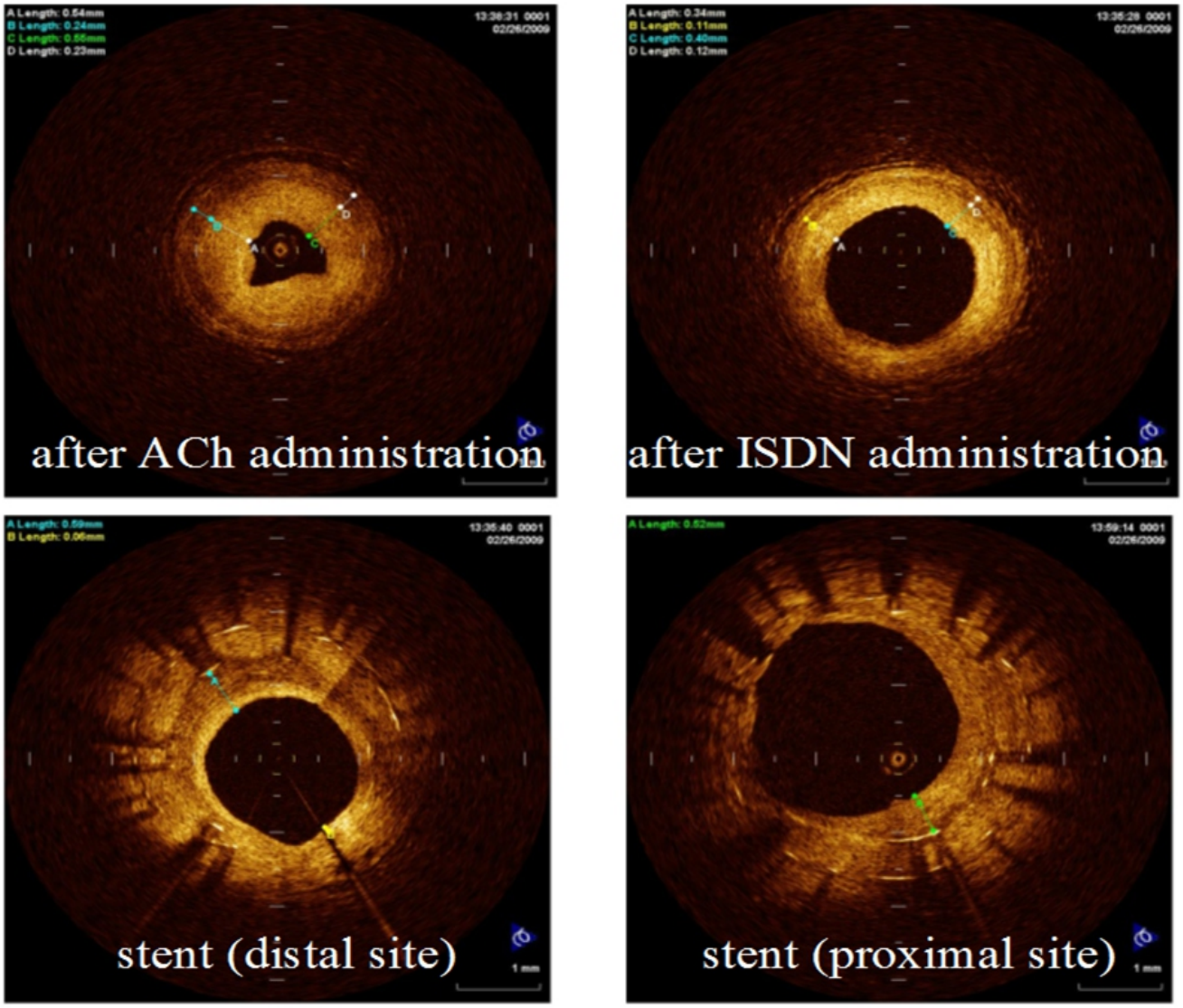


Figure 1: Follow-up catheterization in case 1

a: Angiographic findings at baseline and after ACh provocation

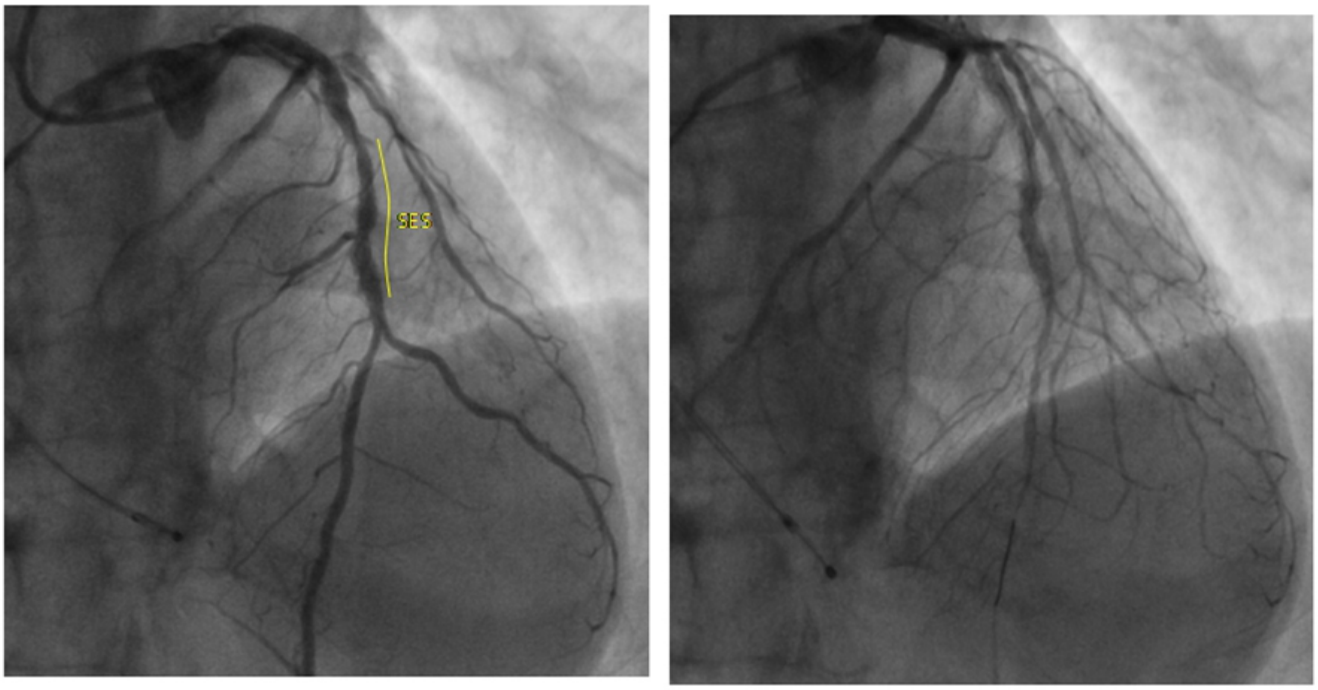
Angiography showing no ISR or a new lesion; however, a severe coronary spasm is provoked in the RCA distal to the stents.

b: OCT findings after ACh provocation and after ISDN administration

Homogeneous or heterogeneous neointimal hyperplasia and peri-strut low-intensity area are observed (lower column). A spasm detected in the ostial segment distal to the stents, where concentric, homogeneous, and high-intensity intimal hyperplasia exist (upper column). During the spasm, a decrease in the vessel diameter and narrowing of the lumen and thickening of the intimal and medial layers is noted. The maximal thicknesses of the intimal and medial layers during the spasm were 0.64 and 0.24 mm, these values decreased to 0.34 and 0.11 mm after ISDN administration, respectively. The lumen border appears star shaped.

Abbreviations: ACh: acetylcholine; ISR: in-stent restenosis; RCA: right coronary artery; OCT: optical coherence tomography; ISDN: isosorbide dinitrate

a



baseline

after ACh administration

b

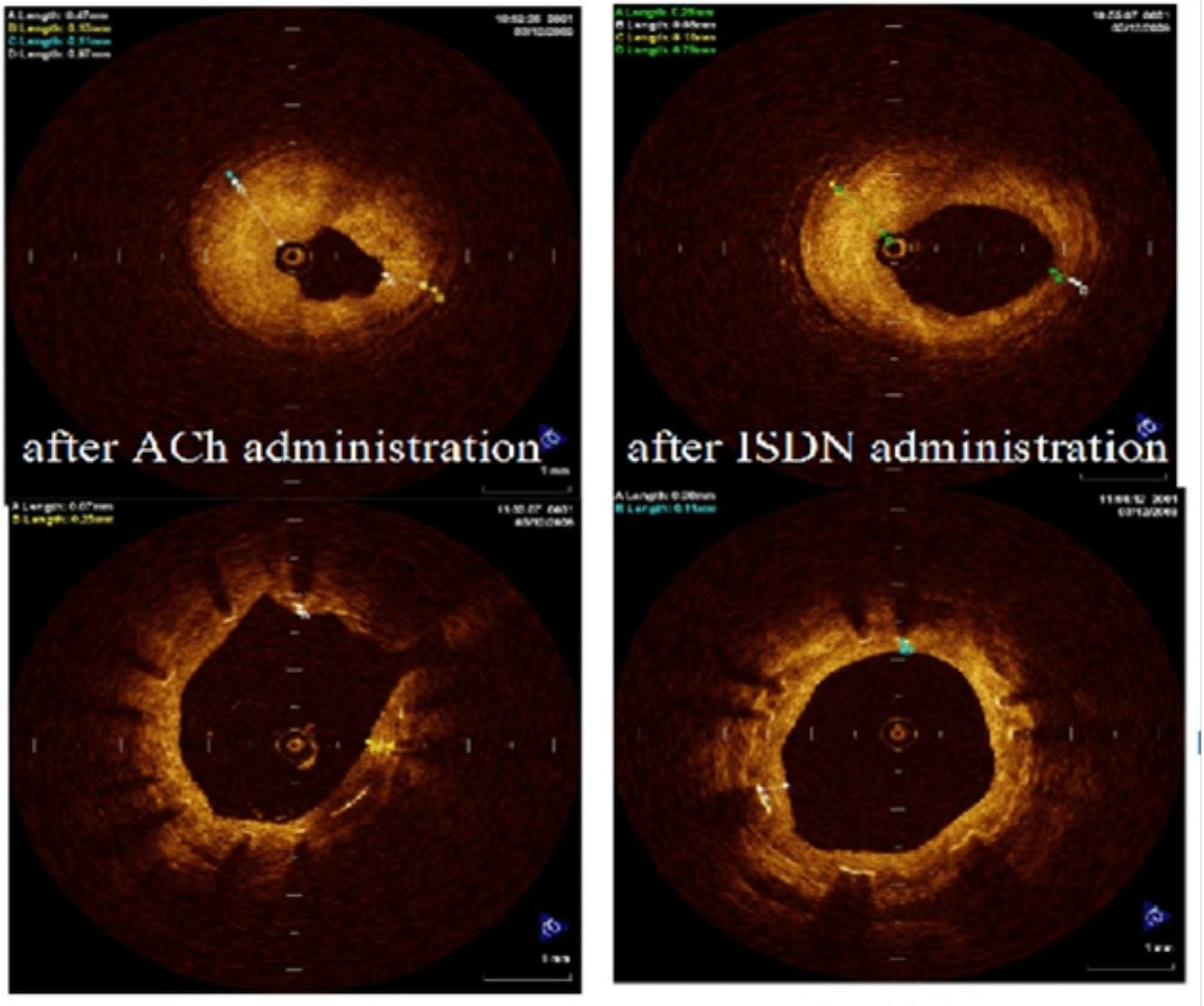


Figure 2: Follow-up catheterization in case 2

a: Angiographic findings at baseline and after ACh provocation

Angiography showing no ISR or new lesion; however, severe coronary spasm is provoked in the LAD distal to the stents

b: OCT findings after ACh provocation and after ISDN administration

Thin, homogeneous neointimal hyperplasia on the stent struts with peri-strut low-intensity area is noted. The distal reference segment also shows eccentric, homogeneous, and high-intensity hyperplasia. Intimal and medial thickening observed at 0.97 and 0.13 mm, respectively, during spasm provocation that decreased to 0.78 and 0.08 mm after ISDN administration, respectively. The lumen border appears star shaped.

Abbreviations: ACh: acetylcholine; ISR: in-stent restenosis; LAD: left anterior descending artery; ISDN: isosorbide dinitrate

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