A 51-year-old man was admitted to a local hospital with sudden onset of severe back pain diagnosed as acute type A aortic dissection on contrast computed tomography (CT). The patient was transferred to Kobe University Hospital and underwent total arch replacement. He had no cardiac risk factors except for obesity, and no connective tissue disorders or clinical features of Marfan syndrome. From the intraoperative findings, the entry of the aortic dissection was confirmed to be in the aortic arch without coronary artery ostium involvement. On the fourth day after the
surgery, he developed chest discomfort associated with ST-segment elevation in the precordial leads. Emergent angiography showed Thrombolysis In Myocardial Infarction (TIMI) 2 flow with a diffuse narrowing throughout the entire left anterior descending coronary artery (LAD), which was refractory to intra-coronary nitroglycerin infusion (Figure 1). No obvious flap or dissection was observed on coronary angiography. Intravascular ultrasound (IVUS) showed a disruption of the intima–media complex with a compressed true lumen by an extensive false lumen in the entire epicardial LAD (Figure 1). Because the entry site of the dissection was not able to be visualized on IVUS, we used frequency-domain optical coherence tomography (FD-OCT). On longitudinal FD-OCT the entry port of the dissection was seen at 7 mm distal to the ostium of the circumflex (Figure 1). Also, we confirmed that the guidewire was located in the true lumen throughout the entire LAD and there was no atherosclerotic plaque. Therefore, we implanted a single bare metal stent (BMS; 3.5×18 mm; Duraflex™) in the initiation port of the dissection in order to seal the entry of the dissection (Figure 2). After the stent implantation, we obtained angiographic TIMI 3 flow with acceptable OCT demonstrating well-expanded and apposed stents with no residual dissection. Eight months after stenting lack of in-stent re-stenosis was confirmed on 64-slice multidetector CT.

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome. The etiology and pathogenesis of SCAD remain unknown, but it has been reported to be associated with connective tissue disorders (eg, Marfan syndrome) and/or acute elevation of blood pressure. According to previous reports, a relatively high comorbidity rate of coronary artery dissection and aortic dissection has been reported (3.2%). In most of such cases, however, continuous arterial dissection through coronary artery ostium could be observed. In the present case, the operative findings as well as OCT showed no evidence of left main ostium involvement. Thus, we diagnosed this case as SCAD probably triggered by acute elevation of blood pressure.

The acute prognosis of SCAD has been reported to be unfavorable, with mortality rates of 43%. Therefore, early diagnosis has been considered important for better prognosis, probably obtained with prompt initiation of optimal treatment. Diagnosis of SCAD is, however, often difficult in part due to the lack of a simple, quick, and reliable imaging modality available in the current clinical setting. In addition, precise detection of the entry port of SCAD is generally challenging on angiography alone, as well as on the current gold standard intracoronary imaging modality, IVUS. There have been few reports showing the value of OCT in the diagnosis of SCAD. In the present case, however, OCT played an important role not only as a diagnostic tool but also in the development of the treatment strategy. Different stenting strategies for the treatment of SCAD may be followed: (1) to perform multiple stenting from distal to proximal starting in the non-dissected distal part of the vessel; or (2) to stent the suspected dissection entry site primarily. Of these 2 strategies, the first may be superior in terms of complete control of the dissected artery and might be favorable in cases in which the exact location of the entry site is ambiguous. Longer stenting in non-atherosclerotic lesion, however, might increase the like-
likelihood of in-stent re-stenosis during follow-up. In the present case, because OCT provided detailed information about the exact location of the entry site, we successfully used a single stent to seal the entry site, thereby avoiding mid-term in-stent re-stenosis possibly induced by multiple long BMS implantation. The ability of OCT to visualize micro-structures in real time enabled the procedure to be carried out precisely. In contrast, OCT has a well-known disadvantage in its relatively poor penetration depth as compared with IVUS. In fact, on the cross-sectional OCT image high-backscattering was seen in the false lumen, probably due to residual red blood cells, which might be misinterpreted as lipid-rich atherosclerotic plaque. A comprehensive diagnostic approach might be required for the diagnosis of SCAD.

Unlike conventional time-domain OCT, which requires coronary artery occlusion during imaging, FD-OCT offers faster image acquisition, allowing for OCT imaging without coronary artery occlusion. This ability of FD-OCT to scan long lesions within a few seconds was very useful, especially in the unstable acute phase. Previous reports have consistently demonstrated the utility of OCT as a research tool in the field of cardiovascular intervention. The present case, however, shows that FD-OCT has additional value extending beyond that of a simple research tool, and can possibly impact on clinical interventional strategy.

Disclosure

The authors report no financial relationships or conflicts of interest regarding the content herein.

References