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Case Report

Stent malapposition occurred 17 days following percutaneous coronary intervention for a severe calcified lesion in acute myocardial infarction

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ABSTRACT

The occurrence of stent malapposition and coronary artery aneurysm (CAA) during the early phase of drug-eluting stent (DES) implantation is rare. This report presents the case of a 55-year-old man who underwent DES implantation to the left circumflex artery with full-arc severe calcified lesion owing to inferior acute myocardial infarction. Coronary angiography and optical coherence tomography (OCT) at 17 days following percutaneous coronary intervention (PCI) revealed stent malapposition and CAA of diameter 6.5 mm in the distal part of the stented vessel. OCT findings at 5 months following PCI revealed a dilated CAA of diameter 7.5 mm and a luminal structure outside the stent. Based on these findings, it was suggested that the cause of CAA in the early phase following DES implantation to the severe calcified lesion was related to coronary sequelae of Kawasaki disease.

<**Learning Objective:** This was a case of coronary artery aneurysm (CAA) in the stented lesion at early phase post percutaneous coronary intervention (PCI). Despite the patient's young age and low coronary risk, there was full-arc severe calcification lesion. Optical coherence tomography after PCI showed luminal structure outside of the stent. It was similar to recanalized CAA of Kawasaki disease (KD). These findings suggested that CAA in early phase following PCI might be related to the sequelae of coronary arteritis, especially KD.>

Introduction

Generally, the mechanisms of stent malapposition are related to (1) positive vascular remodeling, (2) dissolution of the thrombus, (3) chronic stent recoil, and (4) regression of plaque [1]. Coronary artery aneurysm (CAA) following percutaneous coronary intervention (PCI) is associated with vessel wall injury by balloon angioplasty, stent implantation, directional atherectomy, and laser angioplasty. In the drug-eluting stent (DES) era, hypersensitivity reactions to the drug and/or polymer mixture on the DES were associated with CAA [2]. However, stent malapposition and CAA in the early phase following PCI is considered rare.

Case report

A 55-year-old man who survived an out-of-hospital cardiopulmonary arrest with ventricular fibrillation owing to inferior acute myocardial infarction (AMI) visited our hospital. His only coronary risk factor was smoking habit. Emergency cardiac catheterization was performed. Left coronary angiography (LCAG) revealed occlusion of the proximal left circumflex artery (LCX) with continuously severe calcified lesion present distal from the occluded point, and a 5-mm diameter CAA in the left anterior descending artery (LAD; Fig. 1A). The right coronary artery was small and occluded with calcification. The LCX lesion was considered to be reason for AMI, and primary PCI was performed on this lesion. Aspirin (200 mg) and prasugrel (20 mg) were administered as loading dose before he underwent PCI. We attempted to deliver an aspiration catheter to the occluded part of the LCX; however, it was stacked on the proximal part of LCX due to stenosis with severe calcification. After dilation using a non-compliant balloon (NC-TENKU 2.0/15 mm, Abbott Vascular, Santa

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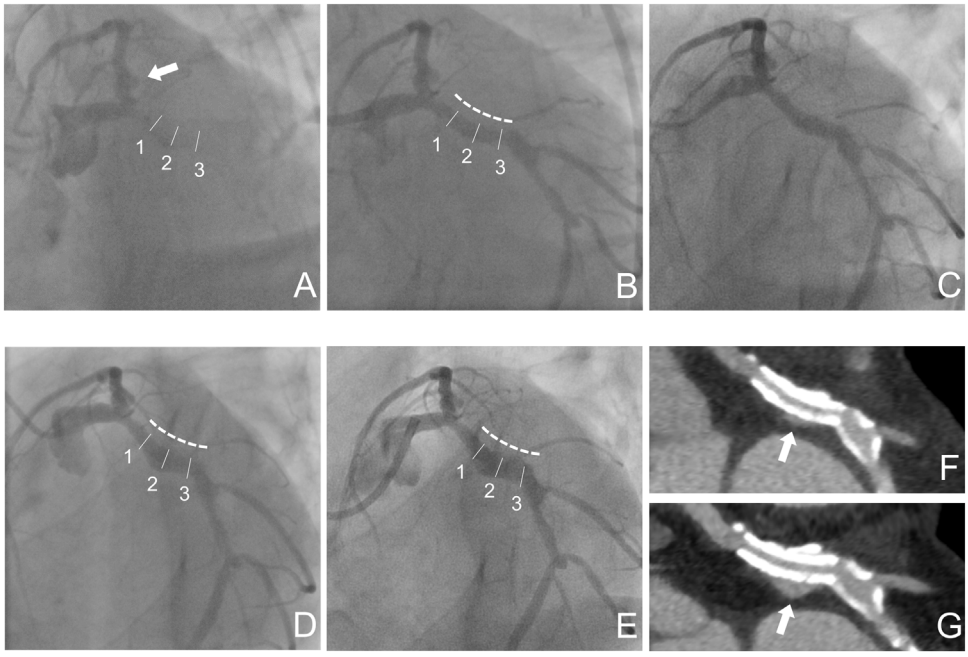

Fig. 1. (A) Initial left coronary angiography (LCAG) of spider view revealed occlusion in the proximal left circumflex artery (LCX) with severe calcified lesion and 5-mm diameter coronary artery aneurysm in the proximal left anterior descending artery (*white arrow*). (B) LCAG after stent implantation revealed an in-stent thrombus. The dashed line indicates the stented segment. (C) LCAG at 3 days following percutaneous coronary intervention (PCI) revealed disappearance of the thrombus and there appeared to be no stent malapposition. (D) LCAG at 17 days following PCI revealed peri-stent contrast staining (PSS) in the distal part of the stent. The dashed line indicates the stented segment. (E) LCAG at 5 months following PCI revealed that PSS expanded to the distal part. The dashed line indicates the stented segment. (F) Computed tomography (CT) at 17 days following PCI revealed the dilated vessel wall of the stented lesion with a maximum diameter of 6 mm (*white arrow*). (G) CT at 3 months following PCI revealed dilation of the stented vessel wall to 7 mm (*white arrow*).

Clara, CA, USA), an aspiration catheter could pass. However, no thrombus could be aspirated. Intravascular ultrasound (IVUS) revealed a continuous severe stenotic lesion with full-arc calcification (Fig. 2A1–3). After repeat dilation using a non-compliant balloon (NC-TENKU 3.0/15 mm), we deployed a DES (Promus Premier 3.0/16 mm, Boston Scientific, Natick, MA, USA) because of lesion recoil (Fig. 1B). Because neo-thrombus continuously appeared in the culprit lesion, prolonged dilation with a perfusion balloon (Ryusei 3.0/20 mm, Kaneka Medix, Tokyo, Japan) was essential. Heparin and argatroban were administered both during and after PCI. However, the thrombus in the stent did not completely disappear, and eventually we had to terminate PCI, leaving the thrombus in the stent. The patient was admitted to the intensive care unit and received intra-aortic balloon pumping (IABP) following PCI. Administration of aspirin (100 mg) and prasugrel (3.75 mg) were continued as dual anti-platelet therapy (DAPT) after PCI. Maximum elevated levels of serum creatine kinase (CK) and CK-MB isozyme at 6 h following admission were 4532 and 685 IU/L, respectively. We performed LCAG 3 days after PCI; it revealed that the thrombus had disappeared and there appeared to be no stent malapposition (Fig. 1C). Continuous administration of heparin and argatroban was stopped and IABP was removed following LCAG. The patient had no heparin-induced thrombocytopenia antibodies, and his platelet count did not decrease during the clinical course. We examined the coronary artery before discharge, and LCAG at 17 days post PCI revealed peri-stent contrast staining (PSS) in the distal part of the stent (Fig. 1D). Optical coherence tomography (OCT) revealed dilation of the vessel in the stenting lesion as an aneurysm, with a maximum diameter of 6.5 mm and cracks of calcification (Fig. 2C1–3). He did not remember whether he was affected with Kawasaki disease (KD) in his childhood. Therefore, the history of KD remained unknown. His meloperoxidase (MPO) antineutrophil cytoplasmic antibody (ANCA) and proteinase 3 ANCA were negative. C-reactive protein and erythrocyte sedimentation rate were in the normal range. To prevent the formation of thrombus in the stent, administration of warfarin with DAPT was initiated. He was discharged 21 days after admission. Computed tomography (CT) at 3 months following PCI revealed dilation of the vessel in the stenting lesion up to 7 mm (Fig. 1G). At 5 months following PCI, we performed cardiac catheterization. LCAG revealed that PSS had expanded to the distal region (Fig. 1E). OCT revealed dilation of the aneurysm in the stenting lesion, with a maximum diameter of 7.5 mm and luminal structure outside the stent (Fig. 2D1–3, Fig. 3D–F). Prasugrel was discontinued at 6 months following PCI. The patient has been followed-up with an annual CT examination once a year. In the 2 years of clinical course, the CAA in the stented vessel did not change, and there were no other symptoms.

Discussion

The present case demonstrated a stent malapposition in an early phase following PCI, with a severe calcified lesion. Coronary aneurysms with severe calcification are associated with coronary artery vasculitis such as KD and systematic vasculitis. The present case had no laboratory evidence of systematic vasculitis. OCT images of vessel wall in CAA showed intimal thickening which is a pathological characteristic of KD (Fig. 2C3,D3). Therefore, cardiac sequelae of KD were considered to be involved with this phenomenon. Aneurysms linked with KD can be categorized into two pathological types. The first is

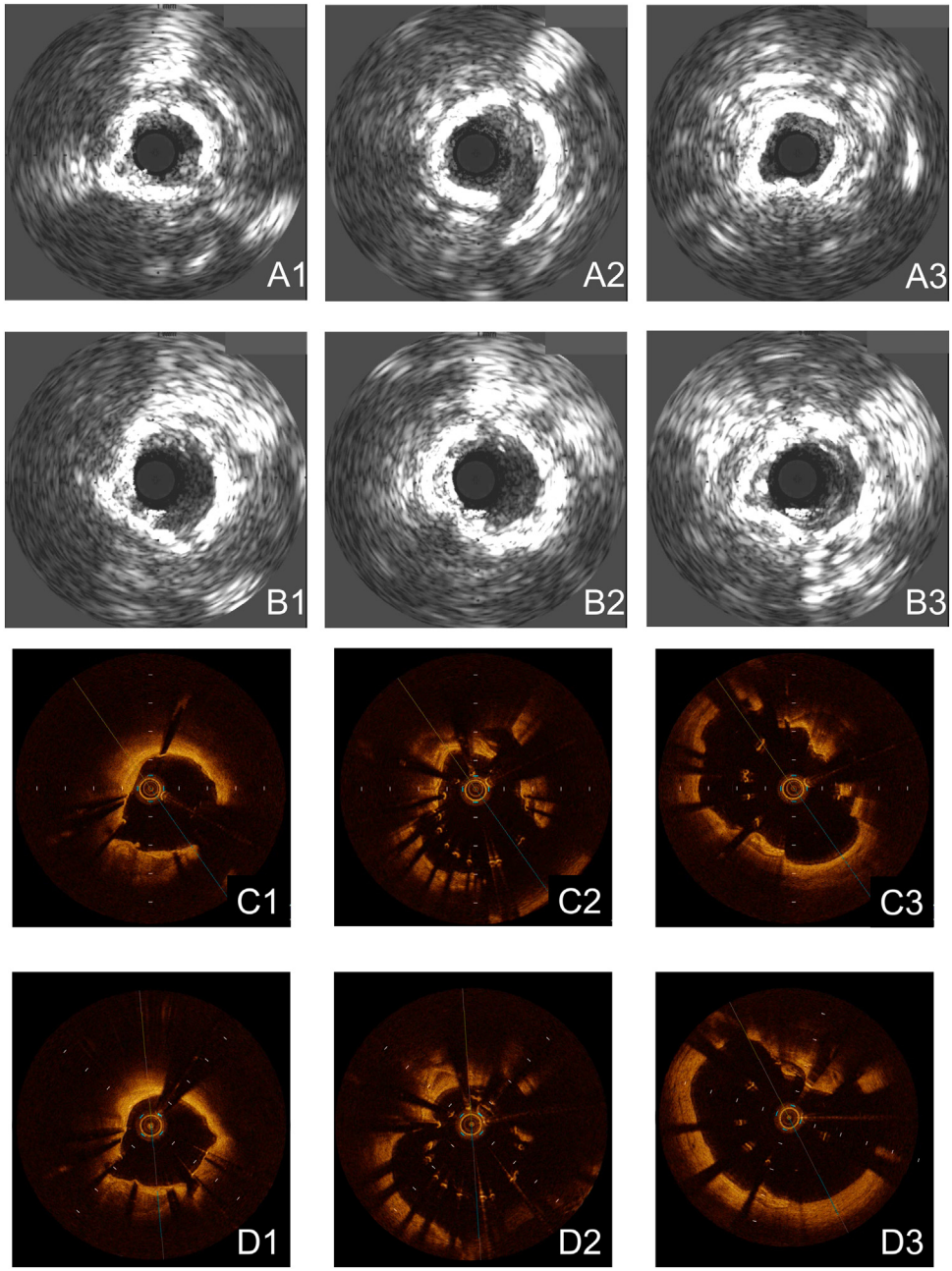

Fig. 2. (A1–3) Initial intravascular ultrasound (IVUS) revealed stenosis with continuous severe calcification and some cracks by balloon angioplasty. (B1–3) IVUS findings after stent implantation revealed stent apposition. (C1–3) Optical coherence tomography (OCT) at 17 days following PCI showed dilation from the middle to the distal part of the stenting lesion acquired with cracking of calcification. (D1–3) OCT at 5 months following PCI revealed dilation of the vessel wall in the stenting lesion with a maximum diameter of 7.5 mm.

remaining aneurysm with calcified vessel walls and the second is recanalized aneurysm following thrombotic occlusion. The latter type of aneurysm shows a lotus root-like shape [3]. LCAG revealed CAA in LAD, and IVUS showed full-arc severe calcification in LCX, despite the patient's young age and low coronary risk. These findings indicated sequelae of coronary arteritis. Stent malapposition in the early phase after PCI might have been due to recanalized CAA which existed, and was occluded prior to PCI, following anticoagulant therapy and the destruction of calcification. The luminal structure outside the stent, which was found by OCT at 5 months following PCI, suggested recanalization of occulted CAA and was similar to CAA associated with KD. Neo-aneurysms have been previously observed at short term follow-up in KD patients who have received balloon angioplasty or stent implantation [4]. Although this patient did not have a history of KD, the mechanism of his condition seemed to be related to sequelae of KD. However, layer structure of blood vessel wall in CAA could not be confirmed (Fig. 2C2,D2). This finding suggested involvement of pseudoaneurysm due to aggressive balloon dilation could not be denied.

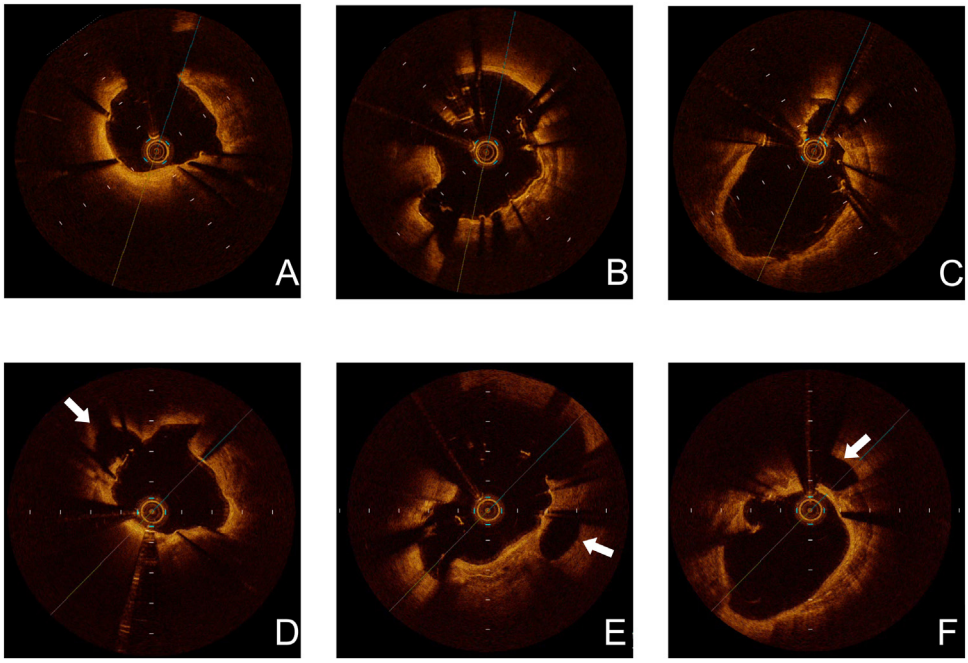

Fig. 3. (A–C) OCT at 17 days following PCI did not observe a luminal structure outside the stent clearly. (D–F) A luminal structure outside the stent was observed from the proximal to the distal part of the stenting lesion at 5 months following PCI (*white arrow*).

Conflict of interest

The authors declare that there is no conflict of interest.

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