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Case Report

Acute coronary syndrome secondary to in-stent plaque rupture occurred at 9 years after deployment of bare metal stent

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ABSTRACT

We report a case with acute coronary syndrome due to in-stent plaque rupture that occurred 9 years after bare metal stent deployment. From the results of intravascular imaging and pathological findings, we diagnosed this case as acute coronary syndrome caused by in-stent plaque rupture. In-stent neoatherosclerosis is an important mechanism of very late stent thrombosis, and the intravascular imaging is helpful to identify this and to decide clinical judgment.

<**Learning objective:** In-stent neoatherosclerosis is an important mechanism of very late stent thrombosis, and the intravascular imaging is helpful to identify this and to decide clinical judgment.>

Introduction

Recent data have shown that in-stent neoatherosclerosis could develop long after stent implantation and cause subsequent plaque rupture leading to acute coronary syndrome [1–3]. We report a case of acute coronary syndrome associated with in-stent plaque rupture, which occurred 9 years after coronary stenting and was visualized clearly using intra-vascular ultrasound (IVUS) and optical coherence tomographic (OCT) imaging. We also compared findings of IVUS and OCT with pathological observations of aspirated materials.

Case report

The case concerns a woman in her 80s with hypertension and dyslipidemia. In February 2002, she had undergone emergent percutaneous coronary intervention (PCI) to the right coronary artery (RCA) due to acute myocardial infarction. Bare metal stents (BMS), Multi-Link Tristar (Guidant, Santa Clara, CA, USA) 4.0 mm × 13 mm and S670 3.5 mm × 18 mm, were deployed in segments #1 to #2. In August 2002, she had undergone elective PCI to the left circumflex artery (LCX) due to effort angina. BMS were deployed in 90% stenosis of segment #13. Successful coronary stenting had been performed in both RCA and LCX. Follow-up coronary arteriography (CAG) was performed in March 2003, and there was no in-stent restenosis in either RCA or LCX. Then, she visited our out-patient clinic regularly without any symptoms for about 8 years.

In January 2011, 9 years after the prior coronary stenting, she had sudden chest pain and was transferred to our hospital. On arrival, she still had severe chest pain (10/10). Her pulse rate was 52 bpm, and blood pressure was 202/86 mmHg. There were no rales and no murmur on chest auscultation. Chest X-ray showed cardiomegaly (cardio-thoracic ratio 64%) and slight pulmonary congestion (Fig. 1). Electrocardiogram (ECG) showed ST elevation in leads II, III, and aVF and ST depression in leads I, aVL, V_2 through V_6. Echocardiographic finding was severe hypokinesis of the inferior wall. Laboratory data on admission showed a slight elevation of only high-sensitive troponin I (0.13 ng/mL) with no changes in levels of cardiac enzymes. Emergent CAG revealed total occlusion at two previous bare metal stent-sites in RCA (in S670 stent) and LCX (Fig. 2). We found the haziness of RCA total occlusion site, suggesting the involvement of fresh thrombus. From these findings of ECG, echocardiography, and CAG, we judged that

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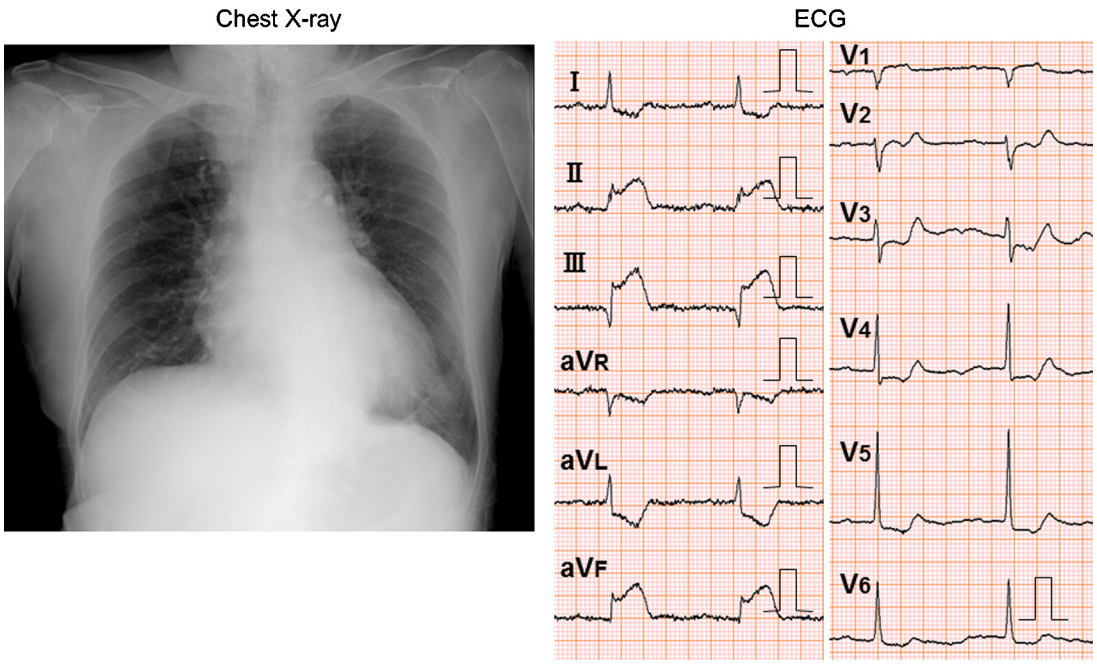

Fig. 1. Chest X-ray and electrocardiogram (ECG) on arrival.

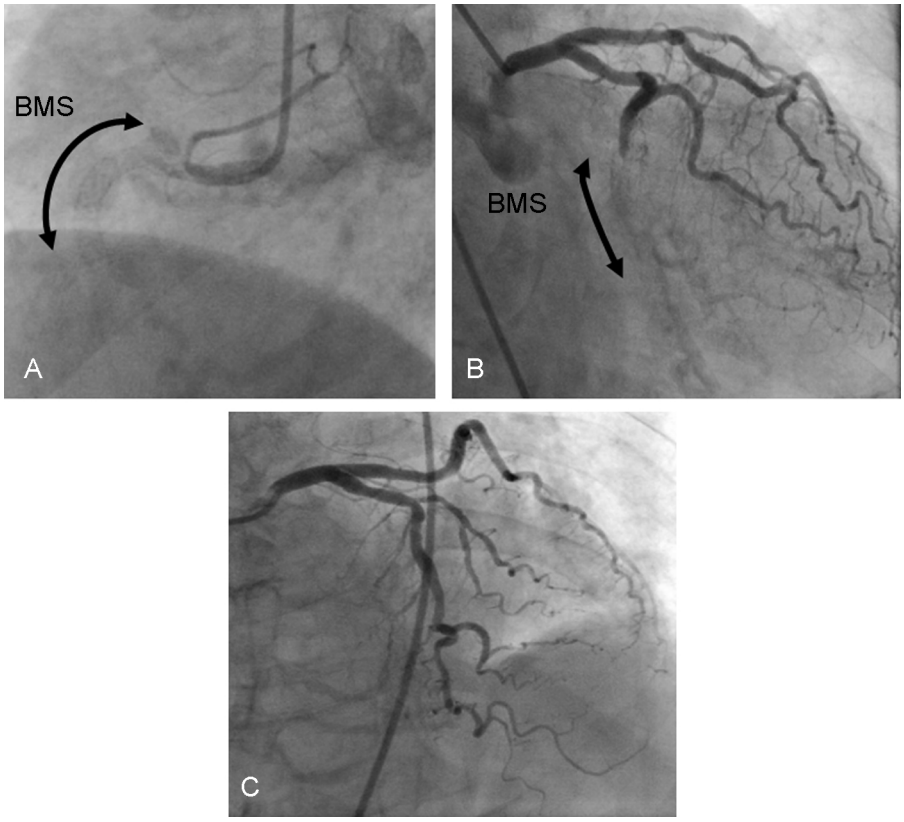

Fig. 2. Emergent coronary arteriography on arrival. Left anterior oblique view of right coronary artery (RCA) (A), right anterior oblique (RAO)-caudal view (B), and RAO-cranial view (C) of the left coronary artery. Previous stenting sites with bare metal stents (BMS) at RCA and left circumflex artery were occluded.

the culprit lesion was RCA and decided to perform emergent PCI to RCA. LCX was thought to be chronic total occlusion lesion.

Prior to PCI to RCA, a temporary pacing lead was inserted into the right ventricle as shown in Fig. 3. Because thrombolysis in myocardial infarction (TIMI) II flow was achieved after the guide wire crossed to RCA, examinations with IVUS and OCT were performed. After thrombus aspiration, drug-eluting stent was deployed directly in the previous BMS site. Finally TIMI III flow was