CASE REPORT

Subacutely progressed extensive aortic dissection complicated with catheter-induced dissection in left main coronary artery

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Summary A 64-year-old man complaining of resting angina underwent emergent coronary angiogram and significant stenosis in the mid-left anterior descending artery was discovered. Although deployment of the drug-eluting Cypher stent relieved the stenosis, the guiding catheter accidentally induced coronary dissection in the left main coronary artery (LMCA). Then, deployment of another Cypher stent at the lesion successfully managed the complication. 20 days later, although asymptomatic, extensive aortic dissection was detected from the coronary sinus of Valsalva to the femoral artery. 64-Row multidetector computed tomography demonstrated that the dissection originated from the LMCA and retrogradely expanded to the aorta. This type of dissection is a rare complication related to coronary intervention and even in such a clinical setting, asymptomatic delayed progression of retrograde aortic dissection has not previously been reported to our knowledge.

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Introduction

Acute aortic dissection complicated with coronary arterial dissection is a rare event, but is a life-threatening complication. The retrograde aortic dissection is usually reported to occur simultaneously with iatrogenic coronary dissection. A small aortic dissection will be stabilized by stenting across the entry point of the coronary dissection. However, when the lesion extends up the aorta >40 mm from the coronary ostium, surgical treatment is usually required.

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Case report

A 64-year-old man complaining of resting angina was transported to our hospital. He had a history of recent myocardial infarction of the right coronary artery (RCA) and aortic valve replacement (AVR) for aortic valve regurgitation. His blood pressure was 145/92 mmHg, and the pulse rate was 54/min with regular rhythm. Laboratory study did not show any evidence of acute myocardial infarction, but a 12-lead electrocardiogram (ECG) demonstrated a slight depression of ST-segment compared with the previous record. Unstable angina pectoris was suspected and emergent coronary arteriogram was performed. We confirmed 99% stenosis of the mid-left anterior descending artery (LAD) and 50% stenosis of the left main coronary artery (LMCA) (Fig. 1B). Subsequently percutaneous coronary intervention (PCI) was performed for 99% stenosis of LAD via right femoral artery. 6Fr-back-up type guiding catheter was well engaged in the LMCA ostium. After coil wire crossed through the stenotic lesion, drug-eluting Cypher stent (Cordis Corporation, Warren, NJ, USA) was directly deployed at the lesion and the stenosis was well relieved. However, while retrieving the stent-mount balloon, the guiding catheter was accidentally drawn into the LMCA, and coronary flow far from the LMCA portion was suddenly interrupted (Fig. 1C and D). Severe chest pain and ST-segment elevation emerged in the thoracic leads of the ECG. We immediately perceived the occurrence of coronary arterial dissection at the 50% LMCA stenotic lesion-induced by the tip of the guiding catheter. After ballooning at the dissected LMCA, another Cypher stent was deployed in the LMCA crossing over the left circumflex artery (LCX) (Fig. 1E). Coronary flow in both LAD and LCX was improved and accessory symptom promptly vanished (Fig. 1F). The final angiographic results demonstrated that there was no static dye around the coronary sinus of Valsalva. We started intra-aortic balloon pumping (IABP) support for hemodynamic stabilization via right femoral artery and the patient was admitted to the coronary care unit. Clinical progress after PCI was stable and the patient was admitted to the coronary care unit. The patient was afebrile throughout the hospitalization, but as shown in Fig. 2, slight increase in the level of inflammatory markers persisted for a while after admission. On day 20 of hospitalization, we incidentally noticed that there was no pulse in the asymptomatic left lower limb. Vascular echogram identified the mobile flap in the left femoral artery and reduced flow in the distal lumen. To clarify the overall disease status, the main artery was scanned by 64-row multidetector computed tomography (MDCT) (LightSpeed VCT; GE Healthcare, Chalfont St. Giles, United Kingdom). We detected extensive aortic dissection originating from the coronary sinus of Valsalva extending to the femoral artery (Fig. 3). The aortic root had expanded to as large as 7.5 cm in diameter. Three major branches from the aortic arch arose from the true lumen. However, branches in the abdominal area were derived from the pseudo lumen of the dissection. The aortic dissection appeared to start from the LMCA (Fig. 4).

The patient requested surgical therapy and surgical findings showed that the LMCA was originating from the true lumen, and that an intimal crack existed just near the LMCA ostium. Low output syndrome persisted for a protracted postoperative period, and the patient finally passed away.

Discussion

The prevalence of established manipulations and sophisticated instruments in the field of interventional cardiology in the past three decades has allowed PCI to be performed more safely. However, we have to keep in mind that procedure-related complications may arise during PCI. Thus, we must be prepared to manage such problems at any time. Although a variety of complications related to PCI have been reported, the development of aortic dissection secondary to coronary artery manipulation has been rarely reported [1]. In such a clinical setting, LMCA retrograde dissection to the coronary sinus of Valsalva is more uncommon than a dissection originating from the RCA [2,3].

Our patient had a history of recent myocardial infarction just 3 months earlier and had an approximately 50% stenosis with eccentric plaque in LMCA. We started intra-aortic balloon pumping (IABP) support for hemodynamic stabilization via right femoral artery and the patient was admitted to the coronary care unit. Clinical progress after PCI was stable and IABP support was discontinued 2 days later. Just after the removal of the sheath, pulsation of the bilateral femoral artery was well confirmed. The patient was afebrile throughout the hospitalization, but as shown in Fig. 2, slight increase in the level of inflammatory markers persisted for a while after admission. On day 20 of hospitalization, we incidentally noticed that there was no pulse in the asymptomatic left lower limb. Vascular echogram identified the mobile flap in the
on angiography. To bail out the LMCA obstruction, we deployed DES, although it is off-label use. For, there have been accumulating reports about safety and efficacy of DES used for unprotected left main trunk [6]. Because the retrograde aortic dissections in past literature were usually noticed at that time of the occurrence of coronary dissections, we never supposed the delayed propagation of aortic dissection but rather were concerned about subacute thrombosis in the stent implanted in LMCA.
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Figure 2 Transition of the inflammatory markers.
Slightly increased markers were observed for a while after admission.

In the case of retrograde acute type aortic dissection, if an aortocoronary dissection is limited to the localized area, stenting on the intra-coronary entry point of the dissection is the most effective method of resolving this catastrophic complication [7,8]. However, when it extends over the aortic sinus of Valsalva, more invasive treatments are usually desired [9,10]. Dunning et al. reported the classification of aortocoronary dissections according to the extent of aortic root involvement, and they proposed a framework for treatment and prognosis in proportion to their classification scheme [11].

Because the retrograde aortic dissection is commonly generated continuously from coronary dissection, intimal tear of the ascending aorta or around the ostium of the coronary artery cannot be observed as Ochi et al. reported [12]. However, our case had an intimal crack although it was unknown when and how it appeared, and the retrograde aortic dissection subacutely progressed. We suppose the pathophysiology in our case was as follows. The aortic dissection did not acutely propagate because driving pressure to expand it retrogradely from the intra-coronary entry point was well diffused through the intimal tear just near the LMCA ostium as well as quickly removed by LMCA stent deployment stabilizing the entry point. Because of the stent deployment, the subintimal connection between the entry point of LMCA and the intimal crack adjacent to the LMCA ostium collapsed, and we could not detect it angiographically just after the procedure.

Next, why the aortic dissection propagated extensively over the aortic bifurcation? First of all, the IABP support could have influenced the deterioration of the dissection through the effect of the augmented diastolic pressure or the direct contact with the aortic wall [1]. Dual antiplatelet therapy

Figure 3 Multidetector computed tomography demonstrated extensive dissection throughout the aorta. Sagittal section of the body is shown in the left panel. Each horizontal section at the level of the dotted line is demonstrated in the panel (A)–(D). (A) The level of the aortic arch. (B) Just above the coronary sinus of Valsalva. (C) The level of the celiac artery. (D) The level of the common femoral artery. Arterial dissection was observed in the every section as indicated by arrows.
Multidetector computed tomography showed the relevance between the left main coronary artery ostium and the aortic dissection. Horizontal section at the level of LMCA ostium indicated that the aortic dissection (A, black arrow) appeared to originate directly from the LMCA ostium (A, white arrow). Volume rendering images of the heart demonstrated the aortic dissection was connected with LMCA ostium (B and C, arrowhead).

It is important to clarify that there was no evidence of aortic dissection before PCI. The patient underwent abdominal CT due to abdominal pain just 60 days before this episode, and the images showed there was no dissection from at least lower thoracic aorta to the bilateral external iliac artery. From that time to this admission, there were no clinical symptoms and findings which were suspicious of progression of aortic dissection. Although there is not enough evidence to conclude the causal relationship between iatrogenic coronary dissection and retrograde aortic dissection, we comprehensively judged the hypothesis based on the circumstantial evidence.

Delayed emergence of the aortic dissection following a catheter manipulation-related coronary dissection is the first report in the world to our knowledge. Because of the asymptomatic progression and our ignorance of the pathological entity, we missed the opportunity to diagnose it as promptly as possible, which finally compelled us to select surgical treatment. Fully understanding the substance of the complications and deciding the best treatment strategy for them are of course important. And more than anything else, physicians must do their best to avoid such complications.
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References


