Acute Posterior-inferior Myocardial Infarction Caused by Total Occlusion Distal to the Apex of the Hyperdominant Left Anterior Descending Artery

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Abstract:
A 77-year-old woman was admitted to our hospital with severe ongoing chest pain. Electrocardiography showed ST-segment elevation in the inferior leads and tall R waves in leads V1-2. Posterior-inferior myocardial infarction was diagnosed. Emergent coronary angiography (CAG) revealed the wrap-around left anterior descending artery (LAD) with total occlusion distal to the cardiac apex. She underwent percutaneous coronary intervention (PCI). Despite difficulty navigating the long and tortuous LAD, we successfully performed reperfusion of the wrap-around LAD. CAG post-PCI showed the posterior descending artery arising from the LAD, described as hyperdominant LAD.

Key words: acute myocardial infarction, hyperdominant left anterior descending artery, total occlusion distal to the apex, percutaneous coronary intervention

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Introduction
Acute posterior-inferior myocardial infarction (MI) is usually due to the occlusion of the right coronary artery (RCA) or left circumflex artery (LCX). Simultaneous anterior and inferior MI have been reported due to the occlusion of the wrap-around left anterior descending artery (LAD) (1-3). However, posterior-inferior MI due to the occlusion of the wrap-around LAD has not been previously reported.

We herein report a patient who underwent successful primary percutaneous coronary intervention (PCI) for posterior-inferior MI caused by acute total occlusion distal to the apex of the wrap-around LAD.

Case Report
A 77-year-old woman was admitted to our hospital complaining of severe chest pain that she had been experiencing for 6 hours. She had no remarkable medical history. On admission, her blood pressure was 167/77 mmHg, and her heart rate was 50 bpm. Electrocardiography showed ST-segment elevation in leads II, III, and aVF and tall R waves in leads V1-2 (Fig. 1). Transthoracic echocardiography revealed mild hypokinesis of the basal posterior-inferior wall and normal motion of the mid wall to apex. Her serum creatinine kinase (CK) level was elevated to 1,128 IU/L and troponin T was positive. Emergent coronary angiography (CAG) by radial access showed total occlusion distal to the cardiac apex of the wrap-around LAD (Fig. 2A). The RCA was short and non-dominant (Fig. 2B), and the LCX was almost normal. We diagnosed her with posterior-inferior ST-segment elevation MI (STEMI) and performed primary PCI for the LAD.

We delivered a 7-Fr EBU 3.75 guiding catheter (Medtronic, Minneapolis, USA) to the left coronary artery ostium. The culprit lesion was crossed with an Amati guidewire (Japan Lifeline, Tokyo, Japan) supported by a Caravel microcatheter (Asahi Intecc, Seto, Japan). A 1.0×6-mm Ikazuchi balloon (Kaneka, Tokyo, Japan) was delivered with a Guideplus guide extension catheter (Nipro, Osaka, Japan) and dilated for total occlusion (Fig. 3A). As a result, the door-to-device time was 83 minutes.

However, we were unable to obtain coronary flow restora-
Figure 1. Twelve-lead ECG on admission. ECG showed ST-segment elevation in leads II, III, and aVF and tall R waves in leads V1-2 as well as ST-segment depression in leads I, aVL, and V3-6. ECG: electrocardiography

Figure 2. Emergent CAG on admission. (A) Left CAG showed total occlusion (white arrow) distal to the apex of the wrap-around LAD. (B) The RCA was short and non-dominant. CAG: coronary angiography, LAD: left anterior descending artery, RCA: right coronary artery

Several cases of proximal total occlusion of the wrap-around LAD have been reported (1-3). We herein report a rare case of posterior-inferior STEMI with total occlusion distal to the apex of the wrap-around LAD. Hyperdominant LAD is a rare coronary artery anomaly that wraps around the left ventricular apex and continues as the PDA (4).

Discussion

Several cases of proximal total occlusion of the wrap-around LAD have been reported (1-3). We herein report a rare case of posterior-inferior STEMI with total occlusion distal to the apex of the wrap-around LAD. Hyperdominant LAD is a rare coronary artery anomaly that wraps around the left ventricular apex and continues as the PDA (4).

Despite the difficulty of PCI due to the long and tortuous LAD and the culprit lesion distal to the apex, we successfully performed PCI and prevented the expansion of the myocardial infarct area at the posterior-inferior wall. We selected a guiding catheter with strong backup support to
Figure 3. Primary PCI and CAG during PCI. (A) A 1.0×6-mm balloon (white arrow) was delivered using a Guideplus guide extension catheter (yellow arrow) and dilated at the culprit lesion. (B) CAG after balloon angioplasty showed TIMI grade 1 flow. (C) TA was performed after delivering an aspiration catheter (red arrow) to the distal lesion. (D) Coronary flow restoration with TIMI grade 2 flow was achieved. (E) CAG after successful PCI showed that the distal LAD was long enough to continue as the posterior descending artery with TIMI grade 3 flow. CAG: coronary angiography, LAD: left anterior descending artery, PCI: percutaneous coronary intervention, TA: thrombus aspiration, TIMI: thrombus in myocardial infarction.

Figure 4. Coronary CT angiography showing hyperdominant LAD without significant stenosis or plaque. (A) Maximum intensity projection image. (B) Curved multiplanar reconstruction image. CT: computed tomography, LAD: left anterior descending artery.
cross the far-distal occlusion. To prevent coronary perforation at the distal position of the wrap-around LAD, we used a guidewire with a low tip weight and good steerability with a knuckled shape, supported by a microcatheter. Furthermore, a guide extension catheter was used to facilitate device delivery to the distal position, which had excellent vascular tractability because of its flexible tip and hydrophilic coating.

The histopathological findings of the aspirated samples showed non-atherosclerotic components with a high proportion of fibrin. Based on the universal definition (5), our case was classified a type 2 MI. Shibata et al. have reported that the prevalence of coronary artery embolism (CE) in patients with de novo MI is 2.9%. The underlying cause of CE is multifactorial, and atrial fibrillation (AF) is the most frequent cause (6). Our patient had no history of AF, and AF was not detected on the electrocardiographic monitoring or Holter electrocardiography after admission. Although there was no evidence of an embolic source in our patient, according to the National Cerebral and Cardiovascular Center criteria (6), she was diagnosed with probable CE (one major criterion: angiographic evidence of coronary artery embolism and thrombosis without atherosclerotic components, one minor criterion: <25% stenosis on angiography, except for the culprit lesion). Our case of posterior-inferior STEMI involved a rare mechanism where the distal position to the apex of hyperdominant LAD was occluded by CE.

Randomized trials have shown that routine TA does not improve the clinical outcome in STEMI (7, 8). According to the European Society of Cardiology guidelines (9), TA is a class III recommendation with level A evidence. Our patient was treated with balloon angioplasty, showing TIMI grade 1 flow. We were able to deliver the aspiration device to the far-distal occlusion, so TA was useful for achieving TIMI grade 3 flow in our case. TA may therefore be an important strategy for select patients with suspected CE.

In conclusion, posterior-inferior STEMI can also occur in patients with occlusion of the wrap-around LAD. Interventional cardiologists should be aware of the efficacy and safety of primary PCI for total occlusion distal to the apex of hyperdominant LAD due to CE, as in our case. Early primary PCI with TA is likely a lifesaving procedure for these patients.

The authors state that they have no Conflict of Interest (COI).

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