Extrinsic Compression of the Left Anterior Descending Coronary Artery by Rib in a Patient With Progressive Left Ventricular Remodeling

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A 78-year-old man with a history of dilated cardiomyopathy diagnosed 5 years previously was admitted to our clinic with accelerating chest pain and dyspnea. The initial ECG showed sinus tachycardia and left bundle-branch block. Laboratory tests revealed elevated levels of troponin T (0.409 ng/mL), creatinine kinase (218 U/L), and creatinine kinase-MB isoenzyme (9.8 μg/L). The peak level of creatinine kinase-MB isoenzyme was 41.9 μg/L. Transthoracic echocardiography revealed a more progressed left ventricular remodeling in comparison with an echocardiography conducted 2 years previously. Compared with the previous study, the end-diastolic dimension of the left ventricle (LV) was increased from 68 mm to 76 mm and the LV end-diastolic volume was increased from 199 mL to 331 mL. Previously, the LV showed global hypokinesia with decreased global LV systolic function (ejection fraction < 30%). The present study showed worsened regional wall motion abnormality at the territories of the left anterior descending coronary artery (LAD) with aggravated LV dysfunction (ejection fraction < 23%; online-only Data Supplement Movies I and II).

Coronary angiography revealed an unusual obstruction of the distal LAD (Figure 1). In the course of distal LAD, a filling defect, evident as a seeming loss of contrast, abruptly began and ended with sharp edges. The proximal and distal reference segments appeared normal. Intravascular ultrasound (IVUS) and optical coherence tomography revealed that the distal LAD was deformed to a slitlike appearance, suggesting an extrinsic compression (Figure 2). To evaluate the cause of extrinsic compression of the distal LAD, cardiac multidetector computed tomography was performed. Cardiac multidetector computed tomography showed no evidence of mediastinal or intrathoracic masses, which could cause the extrinsic compression of LAD. Because the severely dilated LV contacted with the chest wall, the distal LAD was trapped between dilated LV and costochondral cartilage and was narrowed because of extrinsic compression by the costochondral cartilage (Figures 3 and 4).

A diagnosis was made of extrinsic compression of the distal LAD secondary to LV remodeling resulting from dilated cardiomyopathy.

Figure 1. Appearance of coronary angiography conducted 2 years previously (A) and presently (B). In A, no significant stenosis was evident, but, in B, that distal segment of left anterior descending coronary artery was cut off.
Because of refractory angina and hemodynamic instability, we decided to proceed with percutaneous coronary intervention. The distal LAD was directly stented with a 3.0\texttimes38 mm Endeavor stent (Medtronic, Minneapolis, MN) deployed to cover the compressed lesion (Figure 5). No postprocedural complications occurred, and the angina-free patient was discharged 1 week later. Successful stenting of LAD led to subsequent relief of symptoms, achievement of hemody-
namic stability, and decrease of cardiac enzymes. However, there was no improvement in the LV remodeling and systolic dysfunction (online-only Data Supplement Movies III and IV).

Extrinsic compression of coronary artery is a rare disease entity. There are 2 main subgroups of extrinsic coronary arterial compression. One is an anomalous origin of either the right or left coronary artery with subsequent coursing between the ascending thoracic aorta and main pulmonary artery. The other is the left main coronary artery compression syndrome that is due to the dilated pulmonary artery in patients with pulmonary hypertension. However, extrinsic compression of the coronary artery because of the LV dilatation is extremely rare, with only 1 case report. In the present case, we demonstrated that LV dilatation resulted in extrinsic compression of coronary artery causing myocardial ischemia, which was confirmed by intravascular imaging modalities and cardiac multidetector computed tomography. The progressive LV remodeling caused the LAD to be trapped and compressed between dilated LV and rib. The extrinsic coronary arterial compression led to myocardial ischemia. Although the myocardial ischemia induced by coronary compression also aggravated LV dilatation at a time, the initial pathophysiology of coronary compression was progression of dilated cardiomyopathy.

There have been no data about the optimal treatment of extrinsic compression of coronary artery owing to dilated LV. In the present case, the compression led to significant myocardial ischemia, necessitating intervention. We chose percutaneous coronary intervention because of the refractory angina and hemodynamic instability. Successful stenting of LAD led to subsequent relief of symptoms. To our knowledge, progressive LV remodeling resulting in LAD compression by costochondral cartilage presenting with myocardial infarction, which was treated with successful percutaneous coronary intervention, is the first in the literature.

Disclosures
None.

References
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