Severe Compression of the Left Main Coronary Artery in a Patient with Chronic Thromboembolic Pulmonary Hypertension

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Extrinsic compression of the left main coronary artery (LMCA) can occur in patients with an enlarged pulmonary artery trunk secondary to severe pulmonary hypertension (PH). This phenomenon rarely occurs in PH; moreover, few reports have shown that chronic thromboembolic PH can be a triggering factor for this syndrome. Herein, we describe a patient with extrinsic compression of the LMCA with chronic thromboembolic PH who underwent pulmonary endarterectomy and coronary artery bypass grafting successfully. Intravenous ultrasonography (IVUS) was effective for detecting and assessing the compression.

Keywords: chronic thromboembolic pulmonary hypertension, left main coronary artery, pulmonary endarterectomy, coronary artery bypass grafting, intravenous ultrasonography

Introduction

There have been some reports regarding coronary artery compression by a dilated main pulmonary artery trunk secondary to primary pulmonary hypertension (PH) and congenital heart diseases since extrinsic compression of the left main coronary artery (LMCA) was first described in 1957. However, there have been no reports of LMCA compression due to chronic thromboembolic pulmonary hypertension (CTEPH). Although the optimal management of LMCA compression is unknown, there are two choices for treating this condition: catheter intervention and coronary artery bypass grafting (CABG).

Case Report

A 57-year-old woman who had been receiving medical treatment for CTEPH for 3 years had undergone balloon pulmonary angioplasty twice. However, her PH did not improve and she was referred to our hospital. The patient’s preoperative laboratory data were normal: white blood cell 5.0 × 10^3/μl, hemoglobin 12 g/dL, D-dimer 0.5 μg/mL, and C-reactive protein 0.05 mg/dL. The chest radiograph and chest computed tomography scan showed cardiomegaly involving the right cavities with notable enlargement of the pulmonary arch (Figs. 1a and 1b). The orifice of LMCA was compressed by enlarged pulmonary arch in the chest computed tomography scan (Fig. 1b). The electrocardiogram showed right heart strain (Fig. 1c). The lung perfusion scintigraphy demonstrated multiple perfusion defects in both lungs (Fig. 1d), and the pulmonary angiogram showed intimal irregularities and abrupt narrowing of both pulmonary arteries (Fig. 1e). The right heart catheter examination showed severe PH, with the mean pulmonary artery pressure of 49 mmHg and the pulmonary vascular resistance of 921 dynes/sec/cm⁻⁵. The coronary angiogram
In 1957, Corday et al. first described LMCA compression by a dilated pulmonary artery as the cause of coronary insufficiency in patients with PH. There have been some reports regarding coronary artery compression by a dilated main pulmonary artery secondary to primary PH and congenital heart diseases, which may contribute to a higher incidence of sudden death due to malignant arrhythmia and left ventricular dysfunction caused by LMCA compression. The optimal management of LMCA compression in these patients is unknown because treatment of this condition is based on a limited number of case reports.

In 2001, Rich et al. reported the first two patients with primary PH whose extrinsic LMCA compression was treated successfully by percutaneous insertion of an intracoronary stent. Since then, successful intervention cases with short term, but acceptable clinical outcomes have been reported. Extrinsc LMCA compression due to PH manifests as an unusual narrowing of the coronary ostium with progressive recovery in the distal diameter, whereas the remaining coronary circulation remains demonstrated severe stenosis of the LMCA orifice (Fig. 2a). The rest of the coronary branches showed no significant atherosclerotic changes. The intravenous ultrasonogram revealed extrinsic compression of the LMCA by the dilated pulmonary arterial trunk without intraluminal stenosis (Figs. 2c–2f).

Standard pulmonary endarterectomy for CTEPH using intermittent circulatory arrest under deep hypothermia and concomitant CABG to the left anterior descending artery using a great saphenous vein graft were performed (Fig. 3a). The weaning from cardiopulmonary bypass was uneventful, and PH improved to almost within normal range. The PH had improved dramatically, the mean pulmonary artery pressure of 16 mmHg and the pulmonary vascular resistance of 123 dynes/sec/cm−5. The lung perfusion scintigraphy showed improvement of perfusion defects in both lungs (Fig. 3b), and the pulmonary angiogram showed improvement of intimal irregularities and abrupt narrowing of both pulmonary arteries (Fig. 3c). The postoperative coronary angiogram showed release of the LMCA compression (Fig. 2b) and a patent bypass graft.

Comment

In 1957, Corday et al. first described LMCA compression by a dilated pulmonary artery as the cause of coronary insufficiency in patients with PH. There have been some reports regarding coronary artery compression by a dilated main pulmonary artery secondary to primary PH and congenital heart diseases, which may contribute to a higher incidence of sudden death due to malignant arrhythmia and left ventricular dysfunction caused by LMCA compression. The optimal management of LMCA compression in these patients is unknown because treatment of this condition is based on a limited number of case reports.

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normal, similar to the present patient. Stent implantation is not intended primarily in the LMCA lesion; however, catheter intervention can be a great boon to some frail patients. Moreover, LMCA compression due to PH, as in the present patient, is transient and shows no significant atherosclerotic changes.

However, concomitant CABG is an available revascularization strategy. Although few reports have described successful CABG during cardiac operation, CABG should be considered for the following patients: those with a heart disease that is indicated for surgical treatment and those with a giant pulmonary artery aneurysm. Herein, our
approach is based on the concept that CABG is preferable, with percutaneous coronary intervention as a possible alternative. Using a saphenous vein graft as the revascularization method is preferable because the saphenous vein is more suitable than other artery grafts for gaining quick, sufficient blood flow in ischemic coronary arteries.

LMCA compression in patients with CTEPH has not been reported, and this is the first case with stenosis of the LMCA among more than 200 surgical cases with CTEPH in our experience. More similar reports with clear evidence are needed. Rich et al. believed that coronary angiography should be performed in all patients with PH presenting with effort angina or left ventricular dysfunction. At our hospital, we have been performing coronary angiography in every patient with CTEPH since the present case, and if necessary, intravenous ultrasonography (IVUS) should be considered.

IVUS has been advocated by some authors as effective for assessing the degree and type of stenosis. In the present case, IVUS was effective for detecting the extrinsic LMCA compression, and it revealed the absence of intraluminal atherosclerotic changes.

**Conclusion**

We described the case of a CTEPH patient with severe LMCA compression. Pulmonary endarterectomy subsequently improved her PH and LMCA compression. Coronary angiography should be performed to detect LMCA compression in every patient with CTEPH. IVUS is effective for estimating extrinsic compression and intraluminal lesions.

**Disclosure Statement**

The authors declare no conflict of interest.

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**References**