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Extrinsic Compression of the Left Main Coronary Artery: A Case of Atrial Septal Defect with Enlarged Pulmonary Artery

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ABSTRACT

We report the case of an adult referred to our center with an initial diagnosis of stenosis of the left main coronary artery (LMCA). A preoperative investigation disclosed an atrial septal defect (ASD) with pulmonary artery hypertension. The angiographic studies confirmed the diagnosis and showed external compression of the LMCA by an enlarged pulmonary artery. Surgical closure of the ASD and tricuspid valve ring annuloplasty with coronary artery bypass surgery (left internal mammary artery to left anterior descending artery) were undertaken. Six months after the surgery, the patient is doing well.

INTRODUCTION

Extrinsic compression of the left main coronary artery (LMCA) by a dilated pulmonary artery is seldom reported in the literature. In 1957, Corday et al. first reported LMCA stenosis due to extrinsic compression by dilation of a pulmonary artery caused by pulmonary hypertension [Corday 1957]; however, several diseases that trigger pulmonary hypertension have been shown to cause this uncommon entity. In this article, we report an example of LMCA compression caused by secondary pulmonary hypertension related to an atrial septal defect (ASD).

CASE REPORT

A 51-year-old man was referred to our hospital with a diagnosis of exertional dyspnea and chest pain. A cardiac 256-slice multidetector computed tomography (MDCT) evaluation performed in the previous hospital had revealed ostial stenosis of the LMCA with marked dilatation of the main trunk of the pulmonary artery. In addition, the patient was noted to have a heart murmur since childhood that had not been investigated further. A physical examination revealed a heart rate of 90 beats/min and a normal blood pressure of 120/75 mm Hg. An examination of the cardiovascular system was positive for a prominent precordial beat, a grade III/VI systolic ejection murmur and fixed splitting of the S_2 heart sound. Bilateral breath sounds were clear, and fine crackles were heard in the lower zones of the lungs. No peripheral edema was found. A 12-lead electrocardiography examination revealed sinus rhythm with a right bundle branch block and an ST-segment depression involving the anterior leads. The results of the patient's laboratory blood tests were normal. A chest radiograph showed mild cardiomegaly and a prominent dilated pulmonary artery (Figure 1A). A transthoracic echocardiography evaluation revealed a large ostium secundum–type ASD (diameter, 32 mm) with left-to-right shunt flow, moderate to severe tricuspid valve regurgitation, significant dilatation of the right ventricle and the main pulmonary artery (33 mm), severe pulmonary hypertension (systolic pulmonary artery pressure, 90 mm Hg), normal left ventricle size, and normal global systolic function. Because the patient was experiencing typical angina upon mild exertion, a coronary angiography examination with right heart catheterization was performed. Oxymetric studies revealed the absence of a right-to-left shunt with a pulmonary-to-systemic blood flow ratio (Qp/Qs) of 1.8. Angiographic evaluation of the right heart showed a secundum ASD with a left-to-right shunt and a dilated main pulmonary artery. Angiographic hemodynamic study revealed severe pulmonary artery hypertension (systolic pulmonary artery pressure, 80 mm Hg). The coronary angiography evaluation revealed substantial ostial stenosis (90%)

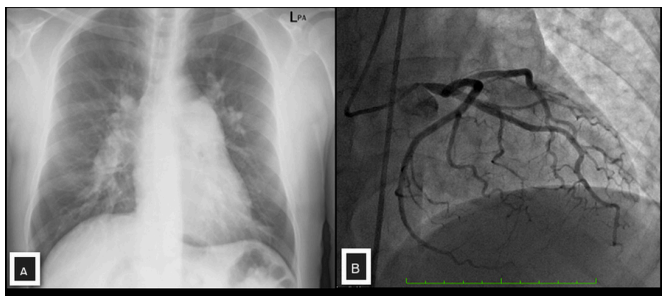

Figure 1. A, Chest radiograph of the patient on admission. Mild cardiomegaly and pulmonary artery dilatation are prominent. B, Coronary angiography in the left anterior oblique cranial projection demonstrates ostial narrowing (90%) of the proximal left main coronary artery with smooth tapering in distally.

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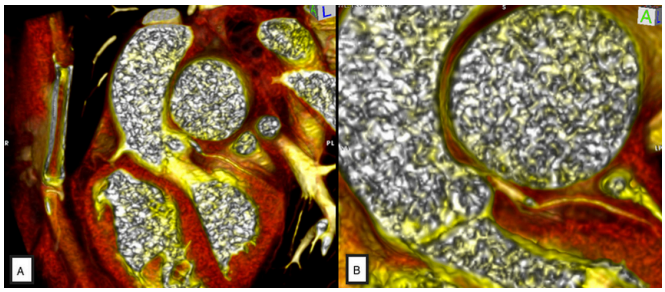

Figure 2. Coronal reconstruction of 256-slice MDCT angiography image demonstrates a severe dilatation of the pulmonary artery causing extrinsic compression with inferior displacement of the left main coronary artery at the ostium.

of the proximal part of LMCA and otherwise normal vessels (Figure 1B). The aspect of the LMCA remained unchanged after intracoronary injection of nitroglycerin, which eliminates catheter-induced coronary artery spasm.

MDCT angiography images in the axial and coronal planes confirmed that the stenosis in the LMCA was due to extrinsic compression by a significantly enlarged pulmonary artery (Figure 2). From these angiographic findings, this case was diagnosed as LMCA stenosis due to extrinsic compression caused by dilatation of the main pulmonary artery trunk associated with pulmonary hypertension secondary to the ASD. From the treatment alternatives, we considered and selected primary closure of the ASD, tricuspid valve ring annuloplasty (31-mm Duran AnCore; Medtronic, Minneapolis, MN, USA) and coronary artery bypass surgery (left internal mammary artery [LIMA] to left anterior descending coronary artery [LAD]) with cardiopulmonary bypass. At 6 months after the surgery, the patient remained clinically stable with no further episodes of angina.

DISCUSSION

Extrinsic compression of the LMCA due to a dilated pulmonary artery has rarely been reported [Mitsudo 1989; Kothari 1994]. If left untreated, however, these patients have a high risk of sudden death due to malignant arrhythmia and left ventricular dysfunction. This rare finding (5% to 44%) has been described in adult patients with ASD, ventricular septal defect, Eisenmenger ductus arteriosus, and primary pulmonary hypertension [Fujiwara 1992; Higgins 1993; Patrat 1997; Kawut 1999].

Because of a particular anatomic feature, the proximal part of LMCA can easily be compressed by the fascial sheath surrounding the great vessels [Corday 1957; Fujiwara 1992]. The degree of compression of the LMCA is related to the anatomic relationship between the main pulmonary artery trunk and the origin of the LMCA. In patients with an ASD, the incidence of LMCA compression ranges from 4.8% to 44% [Mitsudo 1989; Kothari 1994]. The mechanism of LMCA compression is not clearly understood. Dilatation of the main pulmonary artery appears to be the main mechanism; however, it is not the only factor leading to the compression. There are several associated factors, such as the size of the main pulmonary artery, a young age of onset, the ratio of pulmonary artery diameter to the aorta diameter (the risk is increased when the main pulmonary artery diameter is 40 mm and the ratio of the main pulmonary artery diameter to the aorta diameter is 1.21), and the anatomic relationship between the main pulmonary artery and the origin of the LMCA in the sinus of Valsalva [Mesquita 2004].

The definitive diagnosis of LMCA compression has been based largely on angiographic studies of the LMCA. Several techniques, including transesophageal echocardiography, magnetic resonance imaging, and intravascular ultrasonography, have been used. Nowadays, however, advanced noninvasive imaging techniques such as MDCT and magnetic resonance imaging provide valuable information and might be the preferred initial tests [Kawut 1999; de Jesus Perez 2009]. Selecting the proper diagnostic modality requires a strong suspicion of LMCA compression, as was the case with our patient. In our case, MDCT helped to diagnose the LMCA compression, and we believe that MDCT can be considered the initial diagnostic test for this group of patients. However, another noninvasive test, the myocardial-perfusion imaging technique, cannot play a role in diagnosis because of its low sensitivity for detecting LMCA compression.

The ideal management of LMCA compression due to an enlarged pulmonary artery caused by pulmonary hypertension is still a subject of debate. Several approaches, including pulmonary artery thromboendarterectomy, coronary artery bypass surgery, stenting, unroofing (marsupialization) of the coronary artery, and heart-lung transplantation, have been proposed to manage LMCA compression in patients with severe pulmonary hypertension [Fujiwara 1992; Patrat 1997; Rich 2001; Bonderman 2002]. Treating the underlying cause, however, might be the preferred initial approach to treating LMCA compression [Fujiwara 1992]. Among noninvasive procedures, percutaneous revascularization is an alternative treatment modality for LMCA compression. With the drug-eluting stents that are available today, satisfactory results are achievable [Rich 2001; Dubois 2007]. Pulmonary hypertension is the main reason for making coronary artery bypass surgery a less desirable choice for this group of patients, and it is less frequently performed because of the high surgical risk [Kuralay 2002]. Its use is mostly limited in conjunction with other surgical entities, such as pulmonary thromboendarterectomy, correction of underlying congenital cardiac anomalies, or reconstruction of the pulmonary artery [Fujiwara 1992; Ngaage 2005; Jo 2008; Jodocy 2009]. We did not want to subject our patient to stenting of an unprotected LMCA, however, because of the morbidity associated with the procedure [Park 1998; Kosuga 1999; Rich 2001; Park 2009]. Similarly, we also decided to perform a LIMA-to-LAD bypass and a tricuspid ring annuloplasty instead of just closing the ASD. It was not certain that the proximal LMCA compression would disappear after closing the ASD in an earlier surgical phase. In high-risk patients, percutaneous coronary stenting may be the acceptable treatment of choice. As we described earlier, the risk of restenosis after stenting the proximal part of the LMCA is low, possibly because of the

large diameter of the vessel and the rare occurrence of atherosclerosis. Patients with chronic and severe pulmonary artery hypertension, however, are prone to develop pulmonary apoplexy and hemoptysis, and the use of long-term antiplatelet therapy such as clopidogrel after stent placement is considerably risky for such patients. After discussion with members of the surgical team, we decided to perform the coronary artery bypass surgery (LIMA to LAD) and tricuspid ring annuloplasty in conjunction with ASD closure.

CONCLUSION

We believe that this single case report might add valuable information to the literature. First, extrinsic compression of the LMCA by a much-dilated main pulmonary artery is a reversible cause of angina and should be considered in patients with a history of progressive angina pectoris and exertional dyspnea. Second, cardiac MDCT (256-slice), which is a fast, noninvasive, and accurate test, provides valuable information regarding this clinical entity. The diagnosis, however, should be confirmed with invasive angiography. Third, LMCA compression rarely occurs in patients with an ASD. This information may be relevant in evaluating patients with an ASD. Furthermore, a multicenter comparable trial is needed to identify the optimal treatment modality for extrinsic LMCA due to pulmonary hypertension.

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