INTRODUCTION

Angina is commonly seen in patients with severe pulmonary hypertension and may signal coronary compression by a dilated pulmonary artery, a well-recognized condition. Percutaneous coronary intervention, which has been the mainstay treatment for this condition, may not be the solution given the high-pressure, non-compliant nature of the pulmonary artery. We describe a patient with an atrial septal defect (ASD) and severe pulmonary hypertension who experienced coronary arterial compression not relieved by implantation of a coronary stent.

CASE REPORT

A 38-year-old female presented with breathlessness and angina on exertion. Echocardiography revealed a large ASD with severe pulmonary arterial hypertension (PAH). Ensuing right heart catheterization showed an aortic saturation of 98%, a mean pulmonary arterial pressure of 49 mm Hg, pulmonary vascular resistance index of 21.9 Woods unit/m², left to right shunt size of 1.6, and a pulmonary-to-systemic vascular resistance ratio (Rp:Rs) of 0.494. She was started on sildenafil 25 mg thrice daily. Her breathlessness decreased, but angina persisted. She underwent an exercise stress test during which she developed giddiness, hypotension, and ST segment changes at stage II. Coronary angiogram showed compression of the left main coronary artery (LMCA) by the dilated pulmonary artery, and the lesion was stented using a 4.5×17 mm drug eluting stent (Stentys, Paris, France) (Fig. 1).

Despite left coronary artery stenting, her angina persisted. A repeat stress electrocardiogram 3 months after coronary stenting showed widespread ST depression at stage II during a treadmill test. Echocardiogram demonstrated protrusion of the coronary artery into the aorta with abnormal angulation of the stent, suggestive of coronary stent fracture. Subsequent CT of the coronary arteries confirmed stent fracture.

DISCUSSION

Extrinsic compression of the LMCA has been recognized as an important cause of angina in patients with PAH [1]. Unlike patients without pulmonary hypertension, the pulmonary arteries in patients with pulmonary hypertension are severely diluted and non-compliant. Coronary arterial flow, which occurs during diastole, may be compromised by the non-compliant nature of the pulmonary artery. In addition, patients with ASD have increased right ventricular filling during diastole, which further pushes the coronary artery toward the pulmonary trunk during...
Fig. 1. Angiogram pictures the left coronary artery before and after the coronary artery stenting. (A) Kinking of the proximal LMCA due to compression by the dilated pulmonary artery (arrow) before stenting; and (B) resolution of the proximal LMCA stenosis after stenting. LMCA: left main coronary artery.

Fig. 2. Echocardiographic images which raised the suspicion of a fractured coronary stent. (A) Parasternal long axis view showed a coronary stent protruding into the ascending aorta; (B) short axis view demonstrated the stent (arrows) kinked into an angle; (C) transesophageal echocardiogram at mid-esophagus and 50° showed an angulated stent (arrows) with the proximal segment protruding into the ascending aorta; and (D) transesophageal echocardiogram at high esophagus showed non-continuity of the coronary stent (arrow).
Fig. 3. CT scan images showed features suggestive of a stent fracture caused by the dilated pulmonary artery. (A) and (B) presence of discontinuity of the coronary stent (arrows); (C) the distal segment of the coronary stent (arrow) was pushed caudally by the dilated pulmonary trunk; and (D) volume-rendering images showed that the coronary stent (arrow) was kinked by the dilated pulmonary trunk. LCX: left circumflex artery.

ventricular filling.

When present, the LMCA is depressed caudally by the dilated main pulmonary trunk or the right pulmonary artery. Studies have demonstrated that a small angle between aortic sinus and LMCA is a predictor of significant compression [1,2]. In the present case, diastolic movement of the heart pushed the LMCA toward the dilated pulmonary trunk, which may have compromised coronary flow during diastole and caused angina. Coronary compression was aggravated by the presence of a shunt, resulting in significant right heart dilatation.

In the present case, compression was so severe that a well-positioned coronary stent succumbed to fracture. Clues for stent fracture include ongoing chest pain and migration of the proximal portion of the stent into the ascending aorta. Galiè et al. [2] found that 2 out of 5 restenoses were secondary to extrinsic compression. It is unclear whether the coronary stents in those two patients will experience fatigue fracture in the long run. We opined that restenting the coronary artery was likely to yield a similar outcome; hence, coronary bypass surgery was performed instead. The present case demonstrates that cardiovascular imaging coupled with clinical symptoms may provide valuable clues to pathology in patients who underwent coronary stenting.

ASD closure is controversial. In the present case, we opted for closure with a fenestration for the following reasons: 1) there was a relatively large left-to-right shunt, 2) the Rp:Rs was <0.5, and 3) the fenestration allowed for possible enlargement of interatrial communication in the future.

In conclusion, a diagnosis of extrinsic compression to the LMCA should be entertained when patients with PAH complain of angina. After coronary stenting surveillance of stent
position via echocardiography is crucial, as the stent may fracture or be rendered non-functional by the non-compliant pulmonary trunk.

Conflicts of Interest
The authors have no potential conflicts of interest to disclose.

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