Intervalvular fibrosa pseudoaneurysm with projectile shunt flow to left atrium

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Abstract

We report a case of intervalvular fibrosa pseudoaneurysm, causing massive shunt flow from the left ventricle below the left coronary cusp to the left atrium above the anterior mitral annulus, which was clearly demonstrated on preoperative three-dimensional transesophageal echocardiography. Superior extension of the right-sided left atriotomy toward the pseudoaneurysm, combined with transection of aorta and main pulmonary artery, provided its sufficient exposure. Its opening to the left ventricle was closed with a patch, leaving no residual shunt flow.

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1. Introduction

The mitral-aortic intervalvular fibrosa is composed of thin fibrous and avascular tissue connecting the base of the anterior mitral leaflet to the posterior aortic root [1]. Infection of the native or prosthetic aortic valve, and even infected blood stream, can cause infectious damage on this specific region, developing perforation or pseudoaneurysm [2, 3]. When intervalvular fibrosa pseudoaneurysm (IFPA) communicates both to the left ventricle (LV) and left atrium (LA), eccentric jet flow from the anterior to posterior mitral annulus may occur during systole and mimic mitral regurgitation due to perforation in the anterior leaflet [4]. Because of its anatomical location, it is not easy to make a correct preoperative diagnosis. We report a case of such an IFPA, which could be identified clearly on the preoperative three-dimensional transesophageal echocardiography (3D-TEE).

2. Case

A 71-year-old woman had experienced long-lasting high fever without diagnosis in her early twenties. She had noticed gradually progressive (over 20 years) numbness and paralysis of the right arm and leg with unknown etiology. She had deterioration of dyspnea in the past few months. The transthoracic echocardiography (TTE) showed moderate aortic valve stenosis (aortic valve area of 1.4 cm² and peak pressure gradient of 27 mmHg) with moderate grade of regurgitation, eccentric jet flow originating from the anterior mitral annulus toward the posterior annulus, and a cavity-like lesion between the posterior aortic root and LA. The LV diastolic and systolic diameters were 49 and 29 mm. The LV ejection fraction was 72%. The 3D-TEE showed a large opening of the IFPA to the LV and quite thick wall of the IFPA (Fig. 1, left). The location of the IFPA, LV, and AML, and their anatomical relationship are visualized in Video 1. There is blood flow from the LV to the IFPA and from the IFPA to the left atrium above the AML (Video 1). A projectile blood flow coming from its opening to the LV toward the left atrium via its opening just above the AML is seen in Video 2. On coronary angiography, proximal left anterior descending and circumflex arteries appeared to override on partially calcified pseudoaneurysm.

After cold cardioplegic arrest, the aorta and main pulmonary artery were transected, and their stumps were pulled anteriorly for exposure of the pseudoaneurysm. The right-sided left atriotomy was extended superiorly behind SVC and on LA roof, directing toward the pseudoaneurysm. There was a small orifice (5–6 mm in diameter) in the LA wall, which was found to the opening of the pseudoaneurysm to the LA (Fig. 2, left). The left atriotomy was extended toward the opening and reached it. Then, the incision was spontaneously switched from the LA wall to the wall of the pseudoaneurysm, which was thick and atherosclerotic with patchy calcification. There was a round opening (3 cm in diameter) with a thick atherosclerotic margin, which communicated to the LV just below the left coronary cusp (Fig. 2, right). A round-shaped autologous pericardium (4 cm in diameter) reinforced by a Gore-Tex patch was created and sutured to the opening. The

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moderately calcified aortic valve was replaced by a pericardial bioprosthesis. The wall of the pseudoaneurysm, left atriotomy, transected aorta, and main pulmonary artery were closed by continuous 4–0 Prolene sutures. Weaning from CPB was uneventful with no residual shunt flow from the LV to LA via the pseudoaneurysm on intraoperative TEE and postoperative TTE. Pathologic examination of the resected wall of the pseudoaneurysm showed no bacterial colony.

3. Comment

Our case has several unique characteristics as an IFPA. First, the pseudoaneurysm and its anatomical relationship to surrounding tissue could be visualized clearly by preoperative 3D-TEE. Second, it was thought to be developed without relationship to aortic valve replacement and recent onset of infective endocarditis. Third, the patient had a history of long-lasting high fever in her youth. Fourth, the patient had suffered from gradually progressive right hemiparalysis with left cerebral infarction with unknown etiology. Fifth, extension of the upper right-sided left atriotomy toward the pseudoaneurysm via the LA roof with transection of the aorta and main pulmonary artery provided sufficient exposure of the pseudoaneurysm and its opening to the LV.

Afridi and colleagues demonstrated the superiority of TEE with Doppler flow over TTE or aortography in detecting IFPA [1]. Although the 2D-TEE can provide an accurate diagnosis of IFPA, the preoperative diagnosis of the LV–LA shunt via the IFPA was made not by 2D-TEE but by the 3D-TEE in our case. The 3D-TEE also offered quite useful information for the surgeons about its exact anatomical relationship to surrounding tissue.

Although there was no evidence to clarify whether long-lasting fever in her youth was related to infective endocarditis, with later development of the pseudoaneurysm and right hemiparalysis was caused by sporadic microemboli from it [2], a considerably thick and atherosclerotic aneurysmal wall with patchy calcification suggested its remote onset with quite a long time for its development. Because of the above-mentioned pathological nature of the orifices to the LA and LV, her symptomatic deterioration was supposed to be caused not by a new onset of LV–LA shunt, but by progression of moderate grade of aortic valve stenosis with regurgitation. The absence of any other episodes suggestive of infective endocarditis and surgical trauma to intervalvular fibrosa in the last decade may have excluded other possible causes for the IFPA and slowly progressed stroke in our case.

Because of the characteristic anatomical location of IFPA, it may not be easy to find out its exact location of openings both to the LA and LV through conventional oblique aorto-
tomy and right-sided left atriotomy [5]. Our surgical approach may provide sufficient exposure of the pseudoaneurysm and its openings to the LA or LV despite of additional cardiac arrest time to close the transected aorta and main pulmonary artery, and extended left atriotomy.

References


