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Closure of a paravalvular communication originating from the pseudoaneurysm of the mitral-aortic intervalvular fibrosa following prosthetic valve infective endocarditis

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Introduction

Pseudoaneurysm of the mitral-aortic intervalvular fibrosa (PMAF) is a rare but potentially life-threatening event, which is usually caused by infective endocarditis and surgical trauma following cardiac surgery. Although there is a likelihood of an asymptomatic course, the most frequent clinical manifestations are signs of infection from active endocarditis, chest pain, heart failure, and cerebrovascular events; and the common dreaded complications of PMAF were reported as fistulas into the left atrium and aorta (20%) and coronary artery compression (10%) (1). Here, we present a case of successful percutaneous closure of a paravalvular communication originating from PMAF into the left ventricle.

Case Report

A 76-year old woman with a previous history of redo aortic valve replacement presented with methicillin-sensitive Staphylococcus aureus (MSSA) infective endocarditis one month after a second operation. During the fourth week of therapy, she experienced decompensated heart failure. Transesophageal echocardiography (TEE) revealed a pulsatile cavity in the mitral-aortic junction with bulging into the left atrium indicating PMAF and severe paravalvular communication with a systolic flow toward both the PMAF and into the aorta and diastolic flow from PMAF into the ventricle leading to de facto aortic regurgitation (Fig. 1a and 1b) (Video 1). As she was deemed ineligible for a third surgery, percutaneous closure of the entrance and exit of the fistulous communication and thus sealing of PMAF and severe aortic regurgitation was planned. A left coronary system angiogram showed the pseudoaneurysm with contrast filling adjacent to the ostium of the left system; therefore, a JL 4 6 Fr catheter was parked to ensure the flow in the left system during intervention (Video 2). We used a floppy wire with NAVICROSS® microcatheter and Amplatz left 1 catheter to cross the defect. A 8 Fr 90 cm shuttle was introduced through the defect over a 0.035 inch Amplatz stiff wire (Video 2) followed by a 14 mm Amplatzer™ vas-

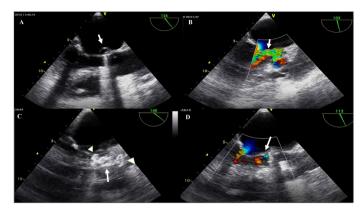


Figure 1. a) Transesophageal echocardiography (TEE), mid-esophageal long-axis view demonstrating PMAF (white arrow). b) Color Doppler showing paravalvular communication from pseudoaneurysm of the mitral-aortic intervalvular fibrosa into the left ventricular outflow tract (LVOT) resulting in severe aortic regurgitation. c) Long-axis TEE image of the deployed Amplatzer vascular plug (AVP) device II (arrow) with discs protruding towards aorta and LVOT (arrowhead). d) Mild paravalvular aortic regurgitation after AVP II deployment (arrow)

cular plug II deployment (Fig. 1c). TEE and angiography showed diminished flow into the pseudoaneurysm with mild paravalvular aortic regurgitation and good patency of the left coronary system (Fig. 1d, Video 2). The patient remained asymptomatic at the six-month follow-up.

Discussion

The expansion of PMAF can impinge on the neighboring structures leading to compression of the left atrium and coronary vessels (2). The left circumflex coronary artery is the most commonly involved, but the other arteries can also be affected. Mitral regurgitation is caused by displacement of anterior mitral leaflet owing to PMAF (3). Aortic regurgitation has been proposed as a risk factor for the progression of PMAF (4). On the basis of this observation, sealing of the paravalvular aortic regurgitation reduced persistent flow into the PMAF.

The clinical spectrum of PMAF can be unpredictable and varies from asymptomatic to rupture or fistula into the left atrium, aorta, pericardium, and left ventricular outflow tract (LVOT). Therefore, mostly surgical intervention is mandated. As surgical correction can be challenging in various clinical scenarios, less invasive methods such as percutaneous closure via the transfemoral and transseptal approach were performed previously (5, 6). The risk of a third surgery was expected to be prohibitive; thus, we elected to perform an endovascular approach in our patient. To minimize the mechanical effects of the AVP II device on the left coronary system and mechanical prosthetic valve, fluoroscopy along with contrast injections were repeated before deployment of the device within the paravalvular defect. Our case illustrates the feasibility and safety of percutaneous closure of PMAF-related paravalvular

communication. Deployment of AVP II device within the communication of PMAF with LVOT also provided diminished flow into the PMAF, and the pressure variation within the aneurysm was blunted

Conclusion

Percutaneous closure of PMAF-related paravalvular aortic regurgitation could be an alternative approach only when the patients are not operative candidates, given the risks of leaving a foreign material at the site of infectious tissue.

Informed consent: Informed consent was signed and given by the patient's parent.

Video 1. Long-axis mid-esophageal two-dimensional transesophageal echocardiogram view showing the pseudoaneurysm of the mitral-aortic intervalvular fibrosa with systolic expansion toward the left atrium and diastolic collapse creating de facto severe aortic regurgitation

Video 2. Fluoroscopy and transesophageal echocardiographic images of the procedure with opacification of the PMAF and left ventricle as a result of severe aortic regurgitation followed by successful closure of paravalvular communication. Note that following successful closure, color Doppler showed no blood flow from PMAF into the left ventricular outflow tract during diastole with mild paravalvular aortic regurgitation and aortography revealed mild opacification of the PMAF and left ventricle after sealing the connection and no coronary artery compression

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