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Percutaneous intervention is not always problem-solving in prosthetic paravalvular leakage

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Introduction

Many studies have shown the long-term durability and gratifying results of the Cooley-Cutter valve (1, 2). Nonetheless, many of the complications cannot be prevented or predicted despite optimal prosthesis function in the individual patient; so careful clinical follow-up are, therefore, essential. We report the case of a well-functioning Cooley-Cutter prosthetic mitral valve (PMV), complicated almost four decades after implantation.

Case Report

A 54-year-old man with history of mitral valve replacement (MVR) was admitted to Department of Cardiothoracic Surgery, Day General Hospital; Tehran, Iran. The MVR was performed with a caged-disk Cooley-cutter valve for severe rheumatic involvement in 1975. He reported no serious complaint until the last few years. The patient had undergone two percutaneous interventions for paravalvular leak (PVL) in the last two years.

At admission, the patient presented with increasing dyspnea with New York Heart Association functional Class III. His evaluation revealed a systolic murmur, mild hemolytic anemia, increased lactate dehydrogenase (LDH) and negative blood cultures. The International Normalized Ratio was within target range.

Transthoracic echocardiography (TTE) illustrated a PMV with increased mean gradient (12 mm Hg), Doppler velocity index (DVI)=0.4, effective orifice area (EOA)=1 cm², severe pulmonary hypertension (systolic pressure=100 mm Hg), left ventricular ejection fraction about 48%, and moderate tricuspid regurgitation. The detailed transesophageal and real-time three-dimensional echocardiography demonstrated two side-by-side Amplatzer ductal occluder devices (Fig. 1) and confirmed significant stenosis (Video 1) and moderate PVL at the posterior segment of the prosthesis and significant annular calcification.

At operation, the PMV was intact without any dysfunction and no abscess or evidence of endocarditis (Fig. 2). The annulus was heavily

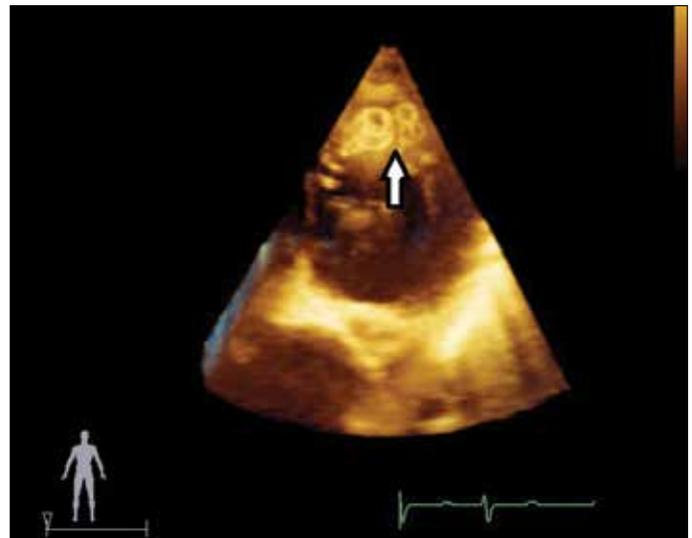


Figure 1. Real-time three-dimensional echocardiography demonstrated two side-by-side Amplatzer ductal occluder devices (white arrow)

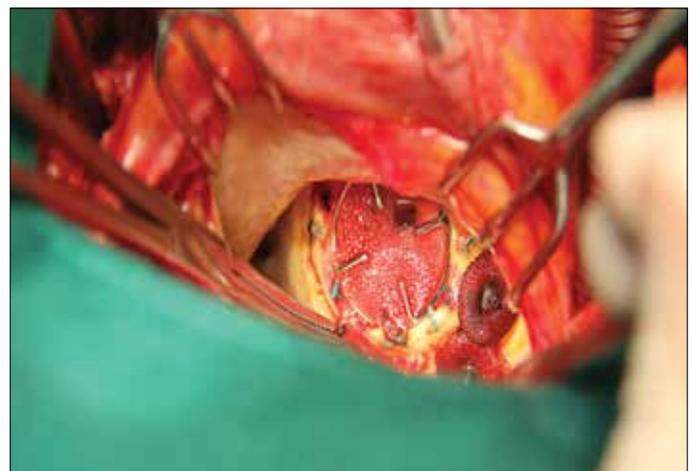


Figure 2. The operational view of mitral prosthesis and two Amplatzer occluder devices

calcified, and the sutures were neither cut nor loosened (Fig. 3A). The valve was replaced with a new mechanical valve and retrieval of the occluder devices was performed (Fig. 3B). The tricuspid valve ring annuloplasty was also performed. On postoperative studying, the PMV had mean gradient of 5 mm Hg, the pressure of the right ventricle decreased to 40 mm Hg and there was no residual PVL. LDH decreased dramatically. The recovery was uneventful, and he was discharged 8 days after surgery.

Discussion

Most PVLs become apparent in the first half-year after the operation (3, 4), although our patient was complicated with PVL more than 35 years after MVR. The suggested possible causes of late PVLs include long-term degenerative change of the suture site, small tears in the calcified portion, and accumulated stress on the annulus-allowing a small area of detachment and unidentified cured infective process in the remnant valve tissue (4).

The presence of a severely increased gradient cannot be equated with intrinsic prosthesis dysfunction. Hence, a high gradient can be due



Figure 3. A, B. Removed mitral caged-disk (Cooley-cutter) prosthesis (A). Removed Amplatzer occluder devices (B)

to an associated subvalvular obstruction or a high-flow state; such occurrences can be suspected when the DVI is normal (5). Conversely, in this case-the combination of a high valve pressure gradient and a low DVI suggested intrinsic prosthesis dysfunction or prosthesis-patient mismatch (PPM); nonetheless, the persistent high transvalvular gradient and high pulmonary pressure before two percutaneous interventions were misinterpreted as PVL. Therefore the best initial treatment could be surgery rather than intervention. Distinction must be made between obstruction resulting from PPM and intrinsic prosthesis dysfunction by calculating the projected indexed EOA of the prosthesis implanted (5). Unfortunately, we could not calculate this index due to unavailable reference values for the old generated patient's prosthesis; consequently, we considered it as obstructive status.

Surgery is regarded as the gold standard of dehiscence repair (6, 7). Recently- percutaneous transcatheter closures of PVLs using a wide array of devices have been reported (6, 8). Such techniques are less invasive and can be employed in most high-risk patients instead of performing repeat surgery (6, 7). The failures of percutaneous procedure in previous studies were mainly attributed either to deployment failure, to the presence of a persistent leak or both (5, 9).

In our case, the pathological fibrous tissue between the sewing cuff and the annulus could conceivably have induced the weakening of the suture sites and produced the PVL. If the PVL is small and the surrounding tissue is clear, direct suture closure or device closure may be possible. If the leak is large or degenerative calcified tissue is present - specifically in old generated prosthetic valves, the effective treatment is valve replacement.

Conclusion

Redo replacement of PMV is the accepted method of care in most complicated prosthesis with PVLs. The device closure of PVLs should be limited to high risk patients and be performed only in the absence of other complications like infective endocarditis, valvular degeneration, and calcification.

Video 1. The transesophageal echocardiography demonstrated two side-by-side devices and confirmed significant stenosis

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Hereditary thrombophilia (factor V R2-mutation) as a contributing factor in premature myocardial infarction associated with pregnancy

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