Successful Percutaneous Closure of Paraprosthetic Aorto-Right Ventricular Leak Using the Amplatzer Duct Occluder

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A 55-year-old male with persisting aorto-right ventricular paraprosthetic leak after mitroaortic valve replacement was hospitalized for recurrent heart failure. Depressed left ventricular ejection fraction and severe pulmonary hypertension with increased right and left ventricular filling pressures were associated with significant left to right shunting through the leak. Elective closure of the leak was obtained with a 6–4 mm Amplatzer duct occluder. No complications were observed, and the patient experienced complete resolution of heart failure symptoms, with NYHA class I heart failure 12 months after discharge.

Key words: paravalvular leak; amplatzer duct occluder; transcatheter occlusion

INTRODUCTION

Paraprosthetic leak (PPL) complicates 2–17% of prosthetic valve replacements [1–3]. Rupture of one or more stitches securing the prosthesis to the valvular ring, usually during the first year after operation, is the cause of this complication. Small PPLs can be asymptomatic or cause hemolysis. Larger leaks can be a contributing cause of heart failure.

Reoperation is frequently required in symptomatic patients, but carries additional risks of mortality and complications in this group of sick patients [3]. Thus, effective percutaneous procedures are an attractive alternative for these patients. Successful percutaneous transcatheter closure of these defects was first reported with the use of the double umbrella Rashkind device by Dr. James Lock and coworkers in 1992 [4]. Since then, experience with closure of congenital and acquired intracardiac defects has continually grown. Major technical improvement has been provided by the family of Amplatzer occluder devices, which are extensively used for the treatment of various intracardiac defects in children and adults [5].

In this report, we describe the successful closure of an aortic PPL with an Amplatzer duct occluder.

CASE REPORT

Past History

A 55-year-old man was admitted to our university hospital for recurrent and progressive heart failure. He had a first mitroaortic valve replacement for rheumatic heart disease 9 years earlier. Two years earlier, he was admitted with NYHA class III heart failure due to significative prosthetic dysfunction and underwent repeat bivalvular replacement with Carbomedics mechanical valves (# 29 and # 21) plus tricuspid annuloplasty. Three days later, he underwent reoperation for a severe mitral PPL. The

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Fig. 1. A: shows pig-tail catheter in aortic root, pacemaker cable and aortic and mitral mechanical prosthesis in an anterior oblique fluoroscopic view. In (B), a contrast aortogram shows aorto-right ventricular PPL (arrow).

postoperative echocardiogram showed mild left ventricular dysfunction (LVEF = 48%), and an abnormal flow entering the right ventricle from aortic root was noted.

One month after hospital discharge, the patient was rehospitalized for recurrent heart failure. Echocardiography noted aorto-right ventricular PPL and a new severe mitral PPL. The patient was compensated and the new surgery was postponed and performed only 11 months before the present admission. In this 4th operation, an 8mm mitral prosthetic detachment was repaired with an autologous pericardial patch. No closure of the aortoright ventricular fistula was attempted, because of friable tissues and uncertain clinical importance. Three months after discharge, he developed severe bradycardia that alternated with tachyarrythmic atrial fibrillation and underwent AV node ablation and a VVIR pacemaker implantation. Subsequently, he maintained class II heart failure symptoms.

Present History

He presented after 1 week of dyspnea at rest and predominantly right-sided congestive signs. He was rapidly compensated on medical therapy. The new echocardiogram showed biventricular dilatation (LVDD = 70 mm, LVSD = 54 mm, RVDD = 46 mm) and mild left ventricular dysfunction (LVEF = 45%); both mechanical valves had no evidence of dysfunction, and moderate tricuspid regurgitation was noted, which allowed pulmonary systolic pressure estimation at 55 mm Hg. The aorto-right ventricular PPL was again noted and caused a significant left to right shunt (Qp:Qs = 1.5:1). Nuclear medicine shunt measurement with Technetium 99-MAG-3 showed Qp:Qs = 1.7:1. Cardiac catheterization showed severe pulmonary hypertension (PAP = 82/33 mm Hg) and increased left and right ventricular filling pressures (PCWP = 25 mm Hg, RAP = 16 mm Hg). An aortogram showed systolic and diastolic flow from the aortic root through a fistula connecting the base of the right coronary cusp (in close relationship with the aortic valve) to the right ventricle (Fig. 1). After written consent was obtained, the patient was scheduled for percutaneous closure.

Procedure

Five and six French introducers were inserted in the left femoral artery and right femoral vein, respectively, and 5,000 IU of unfractionated heparin was given IV. A root aortogram showed free pass of contrast from the right coronary sinus into the right ventricle, through a short fistulous tract with a minimal diameter measuring 3 mm. After a 5F hydrophilic multipurpose catheter was successfully placed in the fistulous tract (Fig. 2A), a 0.035" Magic Torque wire was used to advance the catheter into the right ventricle (Fig. 2B). Then, a 0.035" 260 cm length J wire was advanced through the multipurpose catheter into the right ventricle. A Microvena snare was advanced to the right ventricle and was used to capture and exteriorize the exchange wire through the right femoral vein (Fig. 3A) establishing an arteriovenous loop. The multipurpose catheter was removed and readvanced

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Fig. 2. A: shows contrast hand injection of fistulous tract with 5F multipurpose catheter. In (B), the catheter has entered the right ventricle.



Fig. 3. A: shows capture of the exchange wire (white arrow) with a Microvena snare (black arrow) in the right ventricle. In (B), the multipurpose catheter is passed through the fistulous tract (arrow) into the aorta.

from the femoral vein, through the fistula into the aorta (Fig. 3B). The wire was then replaced with an Amplatzer Super Stiff 260 cm wire and the multipurpose catheter was replaced with a 7 French Amplatzer delivery sheath and the tip advanced from the femoral vein into the aortic root. This allowed placement of the Amplatzer duct occluder with its single distal cap on the higher pressure

side of the fistula, the aorta. Then, a 6–4 mm Amplatzer duct occluder was screwed to the delivery cable, loaded into the sheath, and passed across the fistula. After the distal cap or disk was opened and apposed to the aortic side of the fistula, a root aortogram showed no flow across the fistula (Fig. 4A). The proximal disk was opened and the device delivered (Fig. 4B). No interfer-

Paraprosthetic Leak Closure



Fig. 4. In (A), with an opened distal disk, the Amplatzer duct occluder (arrow) is wedged into the fistulous tract. In (B), the device remains in place after removal of the delivery cable.



Fig. 5. After device (arrow) implantation, a contrast aortogram shows no flow across the fistulous tract with complete PPL closure.

ence with aortic valve functioning was noted. The final aortogram showed no flow across the device (Fig. 5). Catheters and sheaths were removed and the patient returned to the cardiology ward.

Follow-up

Five days after PPL closure, the patient had a new pacemaker electrode implanted through the coronary

sinus into a left posterolateral vein, achieving adequate electromechanical resynchronization. Two days later, the patient was discharged in NYHA class I heart failure. Twelve months later, he remains asymptomatic and a new echocardiogram showed the device in place with permanent PPL closure.

DISCUSSION

PPLs usually appear during the first year after operation and are more frequent in the mitral than aortic location, and with mechanical than bioprosthetic valves. According to a large cooperative study, 8% of mechanical aortic valves have perivalvular regurgitation [1]. However, when specially looked for, aortic PPLs can be seen in 47.6% of patients on Doppler echocardiographic assessment, but 90% are small [6]. This patient required surgical repair twice for severe mitral paraprosthetic regurgitation. Though, the aortic PPL was seen early on, other clinically significant problems delayed its recognition as an important contributor to heart failure. A new surgical procedure was discarded and percutaneous closure was proposed.

Percutaneous closure of PPLs has been attempted since 1987. Hourihan et al. reported 3 patients with successful closure of aortic PPL with the double umbrella Rashkind device [4]. One of them, a 73-year-old male, had a paraprosthetic aorto-right ventricular leak similar to our case, but suffered embolization of two Rashkind devices before successful closure of the defect [4]. Since

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then, several devices have been used for this purpose: the Bard PDA Occluder, coils, and Amplatzer devices used for atrial septal defects or arterial duct closure [7–12]. However, there have been few publications of percutaneous closure of prosthetic paravalvular leaks. Most of them have involved single case reports of mitral PPLs [8-12]. The Amplatzer duct occluder has been successfully used in two of these cases [10,12]. More recently, the percutaneous closure of an aortic PPL causing aortic insufficiency with the Amplatzer duct occluder has been reported [13]. In the present case, the PPL caused a fistulous communication between the sinus of valsalva and the right ventricular chamber determining a left to right shunt. The procedure we used for closure is similar to what has been described for transcatheter closure of ruptured sinus of valsalva aneurysm [14–16].

The family of Amplatzer occluders includes five devices specially designed for the closure of atrial septal, muscular and membranous ventricular septal defects, patent foramen ovale, and peristent ductus arteriosus. Successful results have been obtained in the treatment of these conditions. The variety in shapes and sizes, ease of use, safety, and delivery control of these devices has allowed their successful and increasing use in other cardiac and extracardiac conditions [16–22]. This case is another example of the novel use of the device. In this case, after establishing the arteriovenous loop, the device was easily advanced, positioned, and safely delivered after checking the desired position. However, in this case, the defect was relatively small. Probably larger or crescent-shaped PPLs would require larger or multiple devices.

We believe that the Amplatzer PDA occluder is a useful device for closure of small paraaortic leaks, provided the shape and size are appropriate for the available sizes. However, more experience is needed, especially in larger and crescentic defects.

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