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Mitral Paravalvular Leak: Caution in Percutaneous Occluder Device Deployment

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ABSTRACT

A frail 70-year-old woman presented with symptomatic mitral paravalvular leak 2 months after undergoing a double valve replacement for mixed mitral and aortic valve disease. There was no evidence of prosthetic valve endocarditis. Percutaneous closure of the paravalvular leak was attempted as an alternative to a high-risk surgical procedure. This therapy was successful in reducing the regurgitation but resulted in severe intravascular hemolysis and acute renal failure requiring hemodialysis.

The development of new hemolysis and acute renal failure directed our attention to the Amplatzer device as a possible etiology for these complications. The assumption that her kidney injury was recent and likely reversible compelled us to think of the surgical method as a definitive option. A re-operative surgery was performed, which included retrieval of the occluder devices, pericardial patch repair, and re-replacement using a new biological prosthesis. The patient's urine output gradually improved, and the patient was dismissed with normal renal function.

We present this case as an unusual complication of percutaneous device closure. It also highlights the reversible nature of acute renal failure due to intravascular hemolysis and demonstrates the importance of early surgical intervention for this condition.

INTRODUCTION

The incidence of paravalvular leak after mitral valve replacement ranges from 9% to 12.5% [Genoni 2000]. Although surgery is the traditional method of therapy, percutaneous device closure is gaining more popularity. The success of device closure for the management of atrial and ventricular septal defects had prompted many cardiologists to apply this technique for closure of paravalvular leaks.

We present the case of a patient who developed a paravalvular leak early after mitral valve replacement and was treated with percutaneous device closure. She then developed severe intravascular hemolysis and acute renal failure. Surgical intervention with re-replacement of the valve improved her symptoms and reversed the renal dysfunction and the hemolysis as well.

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CASE REPORT

A frail 70-year-old woman with severe rheumatoid arthritis taking methotrexate and steroid therapy presented to our service with mixed aortic and mitral valve disease and severe pulmonary hypertension. Further preoperative work up did not demonstrate any other significant abnormality (serum creatinine, 1.2 mg/dL; epidermal growth factor receptor [e-GFR], 41 mL/min per body surface area [BSA]). She underwent a double valve replacement using biological valves, and her postoperative recovery was uneventful.

After dismissal from the hospital, the patient did well initially but was readmitted with recurrent episodes of right-sided heart failure and pleural effusion. She underwent placement of a PleurX® Pleural Catheter (Denver Biomedical, Inc., part of Cardinal Health, Inc., Golden, CO, USA) to help manage her resistant effusion. Transesophageal echocardiogram (TEE) performed at that time demonstrated severe periprosthetic regurgitation along the base of the bioprosthesis without any evidence of vegetation. Surgery was considered to be a very high-risk endeavor given the patient's general condition and history of chronic steroid therapy. After a detailed discussion with the patient, percutaneous device closure of the leak was performed in the cardiac catheterization laboratory suite.

A few months after dismissal, she again presented to the emergency department with generalized weakness and fatigue. Baseline work-up revealed that the high serum creatinine had risen to 4.1 mg/dL with an e-GFR of 10 mL/min per BSA and a lactate dehydrogenase (LDH) serum level that peaked at 971 U/L. A peripheral blood smear demonstrated polychromasia and schistocytes. All these clues pointed to a diagnosis of acute renal failure secondary to intravascular hemolysis with the Amplatzer occluder devices as the probable cause of the red cell damage (Figure 1A). We had a detailed discussion with the patient regarding the high operative risk but felt that surgery was the only option to improve her kidney function.

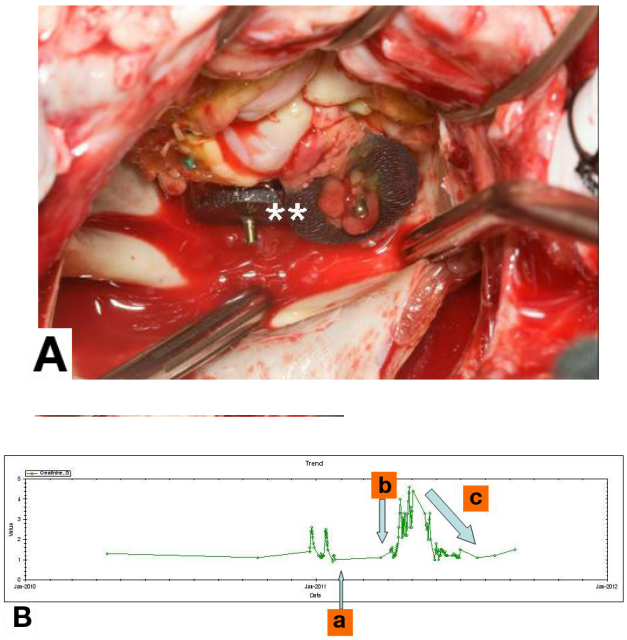

Figure 1. A, The intraoperative photograph depicting 2 Amplatzer devices situated at the posterior mitral annulus (**). B, A graph showing the level of serum creatinine at different time intervals: before the initial surgery (a), after the Amplatzer device deployment (b), and at the time of dismissal from the second surgery (c).

Via a re-sternotomy, the left atrium was approached through the Waterston's groove. Upon opening of the left atrium, the devices were located at the posterior mitral annular aspect (Figure 1B). Extraction of the mitral prosthesis and the Amplatzer device with bovine patch repair of the posterior atrioventricular groove site and re-replacement of the mitral prosthesis using biological valve were performed.

Postoperative recovery was uneventful, and urine output improved to normal; the patient was dismissed from the hospital with a serum creatinine of 1.2 mg/dL (Figure 1C).

DISCUSSION

Intravascular hemolysis due to an unrepaired mitral valve cleft was first described by Sayed in 1961 [Concepcion 2008]. Subclinical intravascular hemolysis is occasionally found even with a normal functioning valve prosthesis, although significant hemolysis is always pathological and is generally associated with a paravalvular leak or failed mitral valve repair [Mecozzi 2002]. The high velocity flow through the small orifice of a paravalvular defect or an unrepaired cleft can cause shearing of the red blood cells with subsequent intravascular hemolysis. The resulting hemoglobinuria leads to acute renal failure due to complex mechanisms of tubular injury [Concepcion 2008]. This complication is more common in the early period of the postoperative course with the majority of patients presenting within 3 months after surgery [Mecozzi 2002]. Infective endocarditis or suture dehiscences are the usual etiology in patients who present late after mitral valve replacement. In our patient, the devices created a narrow orifice that promoted high velocity jets to impinge on the Dacron cloth covering the device; this promoted red cell destruction and the subsequent renal failure. This is akin to the hemolysis seen in patients with an annuloplasty ring associated with mitral valve repair.

Surgical repair of mitral paravalvular leakage is definitely a major undertaking with significant in-hospital mortality as reported by Jindani et al [Jindani 1991]; however, Genoni and his colleagues showed in their series that the mortality rate in the follow-up period for the surgically treated group was significantly lower compared to the conservatively treated patients [Genoni 2000]. Despite these facts, it was more attractive to us to pursue a lesser invasive method to manage our patient

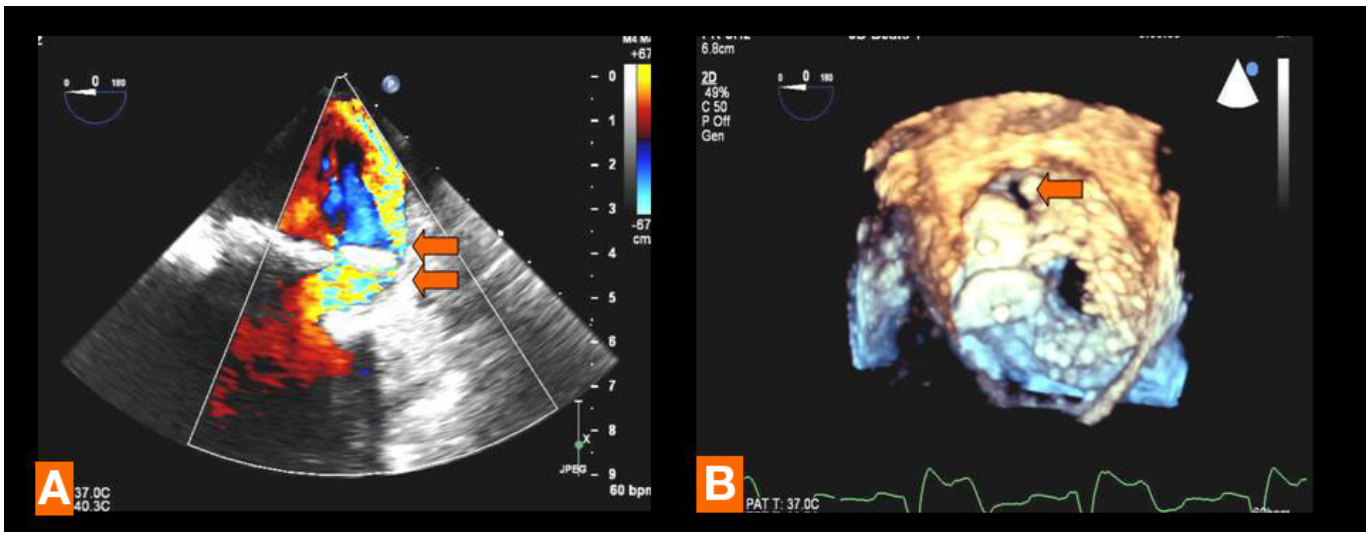

Figure 2. A, Color Doppler transesophageal echocardiogram (TEE) shows the eccentric high-velocity paravalvular jets (orange arrows) after device deployment. B, Three-dimensional TEE view from the same patient showing the residual space (orange arrow) after the Amplatzer deployment.

because she was friable with the added comorbidities mentioned before. The patient did not have any evidence of infection, and we thought that a device closure via a percutaneous approach would satisfy our goals and avoid a repeat sternotomy.

Percutaneous closure of paravalvular leak has been described for patients with severe heart failure, transfusion-dependent hemolysis, and patients requiring prolonged ventilatory support. Sorajja and colleagues have performed device closure in 115 patients, which is the largest single center series to date [Sorajja 2011]. Even with appropriate device deployment, they reported a complication rate of 8.7% such as sudden and unexplained death, stroke, emergency surgery, and bleeding and device embolization. Experience with the technique is limited, and more data are needed before we plan to use this method as the preferred modality for therapy of paravalvular leak.

Mild degrees of intravascular hemolysis after percutaneous interventions is reportedly common, and severe hemolysis is a rare complication and it is usually a result of erythrocyte fragmentation in the high shear velocity jets that come in contact with the device [Webb 2005]. Thus we think that the development of the overt degree of intravascular hemolysis with organ dysfunction in our case after the percutaneous occluder deployment is a result of the narrowing of the paravalvular leak, which creates more jet forces across the small space in addition to the trauma induced by the device itself as previously mentioned. The tubular injury induced by hemolysis is reversible in the early stages before repetitive accumulation of hemosiderin in the tubular cells causes an irreversible renal insult [Chow 2001]. It is highly prudent to detect subclinical hemolysis and paravalvular leaks in the early course to prevent irreversible kidney injury, so prompt surgery is the key to saving the renal function once intravascular hemolysis due to a valvular pathology is detected.

Surgical intervention remains the gold standard for the therapy of mitral paravalvular leak in symptomatic patients [Kirali 2001]. Despite the operative mortality of 6% mentioned in the previous reports, open surgical repair entails prolonged survival and improves symptoms in patients with mitral paravalvular leakage after mitral valve interventions [Genoni 2000]. We feel that although device closure definitely has a place in the management of patients with a prohibitive surgical risk, open intervention gives the best chances for definitive success.

TEE is an important modality in assessing the anatomy of the paravalvular defect that should determine the approach taken.

CONCLUSION

Surgical repair of paravalvular leak provides the best chances of a successful outcome. Acute renal failure due to intravascular hemolysis is reversible if corrected early, and hence aggressive therapy is warranted.

REFERENCES

Chow KM, Lai FM, Wang AY, Chan YL, Tang NL, Li PK. 2001. Reversible renal failure in paroxysmal nocturnal hemoglobinuria. Am J Kidney Dis 37:E17.

Concepcion B, Korbet SM, Schwartz MM. 2008. Intravascular hemolysis and acute renal failure after mitral and aortic valve repair. Am J Kidney Dis 52:1010-5.

Genoni M, Franzen D, Vogt P, et al. 2000. Paravalvular leakage after mitral valve replacement: improved long-term survival with aggressive surgery? Eur J Cardiothorac Surg 17:14-9.

Jindani A, Neville EM, Venn G, Williams BT. 1991. Paraprosthetic leak: a complication of cardiac valve replacement. J Cardiovasc Surg (Torino) 32:503-8.

Kirali K, Mansuroglu D, Yaymaci B, et al. 2001. Paravalvular leakage after mitral valve replacement: is left atrial enlargement an additional indication for reoperation? J Heart Valve Dis 10:418-25.

Mecozzi G, Milano AD, De Carlo M, et al. 2002. Intravascular hemolysis in patients with new-generation prosthetic heart valves: a prospective study. J Thorac Cardiovasc Surg 123:550-6.

Sorajja P, Cabalka AK, Hagler DJ, Rihal CS. 2011. Percutaneous repair of paravalvular prosthetic regurgitation: acute and 30-day outcomes in 115 patients. Circ Cardiovasc Interv 4:314-21.

Webb JG, Pate GE, Munt BI. 2005. Percutaneous closure of an aortic prosthetic paravalvular leak with an Amplatzer duct occluder. Catheter Cardiovasc Interv 65:69-72.