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PERCUTANEOUS MANAGEMENT OF PARAVALVULAR LEAK-RELATED HEMOLYTIC ANEMIA AFTER TRANSCATHETER AORTIC VALVE REPLACEMENT

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ABSTRACT

We present a case of a patient with severe intravascular hemolytic anemia from a paravalvular leak after transcatheter aortic valve replacement and describe a technique of percutaneous repair of the leak with a vascular plug that resolved the patient's transfusion-dependent hemolysis.

KEYWORDS

Transcatheter aortic valve replacement, paravalvular leak, hemolytic anemia

LEARNING POINTS

- Paravalvular leak after transcatheter aortic valve replacement could lead to intractable hemolytic anemia.
- Percutaneous paravalvular leak repair with vascular plug implantation is a feasible treatment for aortic prosthesis leak-related hemolysis.

INTRODUCTION

The incidence of clinically evident hemolytic anemia following transcatheter aortic valve replacement (TAVR) is unknown. Potential mechanisms for hemolytic anemia post-TAVR include, paravalvular leaks (PVL). Hemolytic anemia post-TAVR has been linked to adverse long-term outcomes, including repeat hospitalization and death[1]. Percutaneous repair of PVL through vascular plug implantation is proposed as a treatment option for high-risk patients, as open repairs carry a high mortality risk[2]. However, there is limited literature on the feasibility and efficacy of transcatheter PVL correction with vascular plug implantation to resolve leak-related hemolysis. We present a case of a patient with refractory, transfusion-dependent, non-immune-mediated hemolytic anemia that completely resolved after percutaneous PVL repair with vascular plug implantation.

CASE DESCRIPTION

A 74-year-old female with severe calcific aortic stenosis (aortic valve area of 0.7 cm^2, a trans-aortic valve means gradient of 48 mmHg, peak velocity of 4.4 m/s) with baseline New York Heart Association (NYHA) class III-IV

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symptoms underwent elective TAVR with a 20 mm SAPIEN 3 Ultra RESILIA valve after being deemed as high-risk for surgical aortic valve replacement. Immediately following an uneventful transfemoral implantation of the prosthesis, a trace paravalvular leak (PVL) was identified on post-TAVR supra-valvular aortography. This was confirmed by transthoracic echocardiography (TTE) *(Fig. 1)*. The PVL was managed with post-dilatation of the prosthesis with the delivery balloon using an additional 2 ml of diluted contrast. Three weeks post-TAVR, the patient was admitted for symptomatic anemia, with a hemoglobin level of 6.6 g/dl. Her history was negative for the use of non-steroidal anti-inflammatory drugs or anticoagulants, and there was no evidence of overt bleeding. There was no lymphadenopathy or hepatosplenomegaly on physical examination. Laboratory tests for iron, folate, and vitamin B12 levels were within normal limits. Hemolysis laboratory findings indicated mild unconjugated hyperbilirubinemia, low haptoglobin (<8 mg/dl), and elevated lactate dehydrogenase (LDH 537 mg/dl), but the direct antiglobulin test result was negative. A peripheral blood smear showed normal platelet counts, reticulocytosis, and 2+ schistocytes. She was transfused with red blood cells, started on erythropoietin with iron supplementation, and discharged to follow up with hematology outpatient.

The patient's medical history included hypertension, hyperlipidemia, diet-controlled type 2 diabetes mellitus (HbA1c 6%), hypothyroidism, and stage 3 chronic kidney disease. At the time of admission, the patient was taking the following medications: atorvastatin 20 mg daily, carvedilol 12.5 mg every 12 hours, hydralazine 25 mg daily, losartan 100 mg daily, nifedipine 60 mg every 12 hours, levothyroxine 112 µg daily, plavix 75 mg daily, and pantoprazole 40 mg daily.

The differential diagnoses for the patient's anemia included anemia due to chronic kidney disease, autoimmune hemolysis (ruled out by a negative Coombs test), and cardiac prosthesis-mediated non-immune intravascular hemolysis.

During outpatient visits, the patient continued to receive intermittent blood transfusions every 2 to 3 weeks due to recurrent symptomatic anemia, which only showed temporal improvement in hemoglobin levels. An extensive gastroenterology evaluation, including upper and lower gastrointestinal endoscopies and capsule endoscopy, ruled out gastric or intestinal bleeding as the cause of her refractory anemia. Serum electrophoresis did not indicate gammopathy. TTE revealed a previously identified PVL, confirmed by transesophageal echocardiography (TEE) and cardiac-gated computerized tomography angiography. The leak was determined to be at the raphe of the right and non-coronary cusps *(Fig. 2)*.

Given the concern for intravascular hemolysis caused by the PVL, the patient underwent PVL closure under general anesthesia. Antibiotic prophylaxis was administered pre-procedure and heparin was administered to maintain activated clotting time >300 s through the procedure. A transfemoral approach was utilized. The closure was guided by fluoroscopy and TEE. A 6F angle glide wire and a multipurpose catheter were used to cross the PVL. Biplane imaging and 3D TEE was used to close the small leak. A 6mm Amplatzer Vascular Plug (AVP) II *(Fig. 3 and 4)* was then deployed through a 6F Shuttle sheath. Intraprocedural TEE revealed complete resolution of the PVL, which was confirmed by supra-valvular aortography *(Fig. 5)*.

The patient's hemolytic panel improved within 24 hours post-PVL closure, with LDH decreasing from 520 mg/dl to 443 mg/dl, haptoglobin increasing from <8 mg/dl to 41 mg/dl, and hemoglobin rising from 7.5 g/dl to 8.5 g/dl. At 30 days following the PVL closure, an outpatient hemolysis work-up confirmed complete resolution of the intravascular hemolysis, with LDH normalizing to 215 mg/dl and

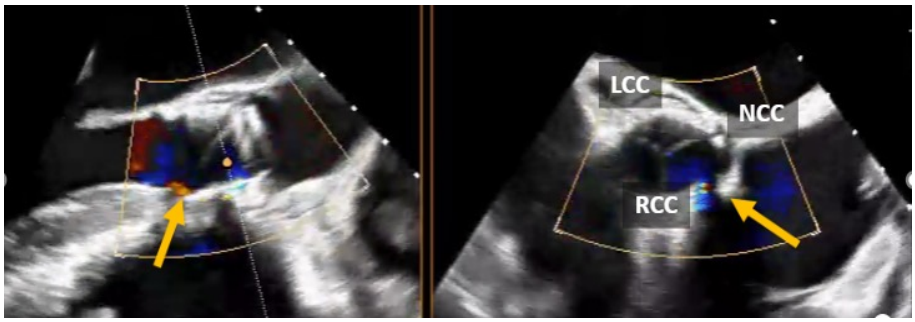

Figure 1. Parasternal long axis (left) and parasternal short axis aortic view (right) transthoracic echocardiography showing trace paravalvular leak (yellow arrows). Abbreviations: LCC, left coronary cusp; RCC, right coronary cusp; NCC, non-coronary cusp.

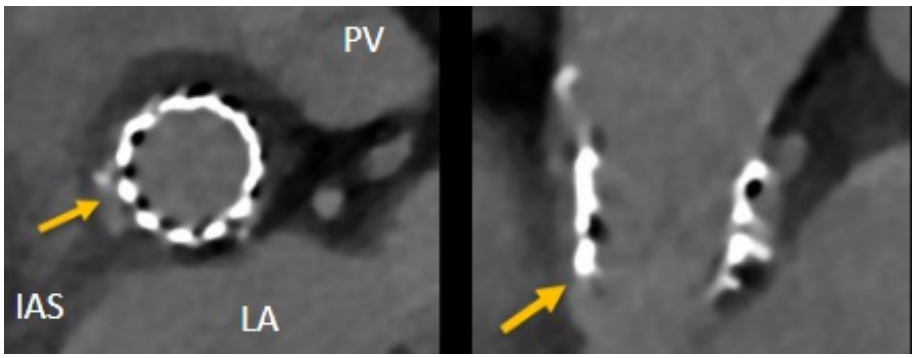

Figure 2. Cardiac computerized tomography angiography showing a small paravalvular leak (yellow arrow). Abbreviations: IAS, interatrial septum; LA, left atrium; PV, pulmonary valve.

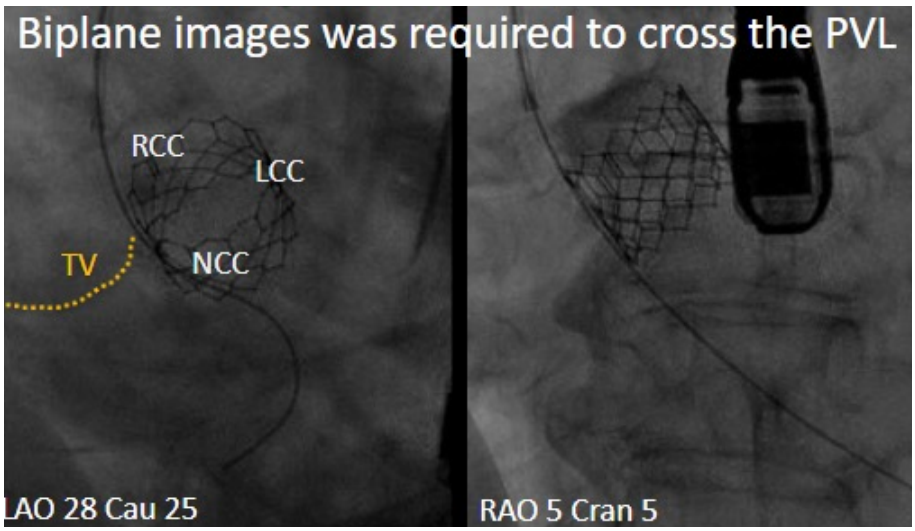

Figure 3. A 6F angle glide wire and a multipurpose catheter crossing the paravalvular leak. Abbreviations: TV, tricuspid valve; LCC, left coronary cusp; RCC, right coronary cusp; NCC, non-coronary cusp.

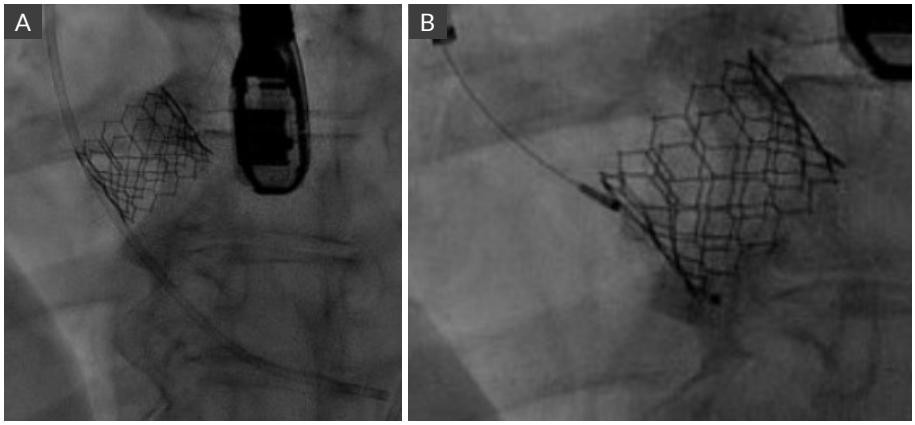

Figure 4. A 6F Shuttle sheath was placed in the left ventricle (A) and a 6 mm Amplatzer Vascular Plug II deployed in paravalvular leak (B).

haptoglobin rising to 208 mg/dl. At the 6-month follow-up, the patient's hemoglobin remained stable between 9 to 10 g/dl without needing new blood transfusions, and her NYHA classification improved to class II.

DISCUSSION

PVL is a defect between a prosthetic valve and the surrounding native tissue. This complication is common post-TAVR; however, newer generation TAVRs devices have decreased large PVL. Several risk factors are associated with PVL post-TAVR, including patient-prosthesis mismatch, native valve calcification, underinflation or malposition of the prosthetic valve, and incomplete apposition to the annulus[3]. PVL is linked to poorer clinical outcomes after TAVR, regardless of the type of prosthesis used, whether balloon-expandable or self-expandable[4]. Approximately 5% of patients with PVL experience symptoms of congestive heart failure or mechanical hemolytic anemia due to PVL, necessitating further surgical or interventional treatment.

In cases of PVL complicated by intractable hemolysis, where patients are at prohibitive surgical risk, transcatheter repair of the PVL is recommended with a class II indication[5]. Percutaneous PVL repair is a complex procedure, and currently, no specific devices are designed to treat PVL after TAVR.

In this index case, after thorough pre-procedural planning by a heart team and intraprocedural cardiac imaging guidance, we successfully used the AVP II device—originally intended for closing peripheral vessels—to repair the PVL. This resulted in immediate and sustained improvement in the patient's hemolysis. As the eligibility criteria for TAVR continue to expand, the need to address PVL may

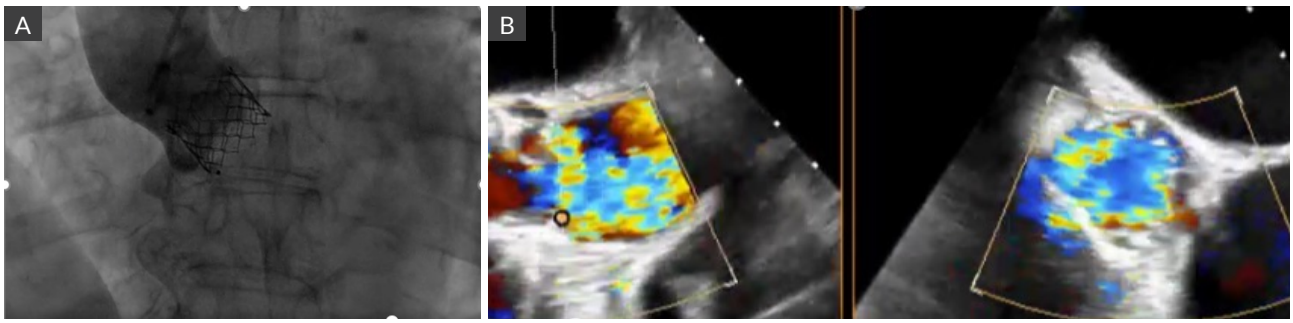

Figure 5. Supra-valvular aortography (A) and trans-esophageal echocardiography (B) confirmed the complete resolution of the paravalvular leak.

increase. Therefore, this case report is clinically relevant, demonstrating that percutaneous closure of PVL using AVP II is feasible and effective.

CONCLUSION

PVL-mediated hemolysis can complicate TAVR, but there is currently no standardized transcatheter treatment for post-TAVR PVL-related hemolytic anemia. Our case demonstrated that transcatheter AVP plugging is feasible and effective in resolving PVL and its associated hemolytic anemia.

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