78-year-old man at high risk for aortic valve replacement (Euroscore, 44%; left ventricular ejection fraction, 30%; previous bypass graft surgery) had undergone a transapical aortic valve implantation (26-mm Edwards Sapien) with a good immediate clinical and echocardiographic outcome: peak transvalvular gradient, 15 mm Hg; aortic valve area, 1.6 cm²; and trivial aortic regurgitation (Figure 1). He was treated with dual-antiplatelet therapy (clopidogrel, 75 mg/d for 1 month; aspirin, 100 mg/d lifelong), as recommended after Sapien valve implantation. The clinical and echocardiographic follow-ups at 1 month were excellent (New York Heart Association class II; peak transvalvular gradient, 18 mm Hg; aortic valve area, 1.6 cm²).

The patient was admitted 4 months after the procedure to the emergency department with a non–ST segment elevation myocardial infarction, signs of left heart failure, and an aortic systolic murmur. There was no evidence of endocarditis: no fever, no rash, and normal inflammatory parameters as per laboratory findings.

The coronary angiogram showed graft patency, and the aortic angiogram (Figure 2) demonstrated an asymmetrical opening of the Sapien valve leaflets, despite the circular shape and transannular position of the stent, and the absence of a significant aortic regurgitation (supplemental movie in the online-only Data Supplement). During cardiac catheterization, a ViewFlex catheter (St. Jude Medical) was introduced through the femoral vein to perform intracardiac...
echocardiography, which showed a thickened aspect of the Sapien valve and thrombus on the leaflets, resulting in a significant aortic transvalvular gradient (Figure 3). These intracardiac echocardiographic findings were comparable with those of the transesophageal assessment (Figure 4): thickening of the leaflets, peak transvalvular gradient of 73 mm Hg, aortic valve area reduced to 0.4 cm², and trivial aortic regurgitation.

Screening tests for thrombophilia (including abnormalities of protein C, protein S, antithrombin III, homocysteine plasma level, factor V Leiden mutation, prothrombin G20210A gene mutation, and antiphospholipid antibodies) were performed, showing the absence of any coagulation disorder and a normal response to aspirin (ristocetin- and 2 μg/mL ADP–induced platelet aggregation reduced at 24% and 39%, respectively).

Because there was a clinical suspicion of an aortic bioprosthetic thrombosis, the patient received heparin followed by Coumadin therapy. After 1 month of anticoagulant therapy, the peak transvalvular gradient significantly decreased to 22 mm Hg, the aortic valve area increased to 1.2 cm², and the Sapien valve leaflets appeared more mobile and thinner (Figure 5).

Discussion

This case shows that bioprosthetic thrombosis after a successful transcatheter aortic valve implantation can occur even in the absence of a coagulation disorder or stopping antiplatelet therapy. As published by Cribier et al, antiplatelet therapy after transfemoral aortic valve implantation in the I-REVIVE and RECAST trials is clopidogrel 75 mg/d for 1 month and low-dose aspirin indefinitely. After Sapien valve implantation, the aortic endothelium is in contact with a stainless steel stent, and it seems logical to apply the same rules that are used after bare-metal stent implantation in coronary arteries. Recommendations after CoreValve ReValving system implantation are a prolonged dual-antiplatelet therapy: clopidogrel for 6 months and aspirin lifelong. The asymmetrical geometry and the incomplete apposition of the nitinol stent may predispose to a higher risk of thrombosis and leaflet distortion, compared with the short and circular stent of the Sapien valve.

The leaflets of the Sapien valve are bovine pericardium treated similarly to the surgical bioprosthesis, for which anticoagulation by warfarin is recommended during the first 3 months. The risk of thromboemboli after open-heart surgery is probably higher than after transcatheter aortic valve implantation. The transapical approach requires chest and a small heart opening as opposed to the transfemoral approach.
technique. This case suggests that preventive antithrombotic measures after transcatheter Sapien valve implantation could be different between the transapical and transfemoral approach.

The aortic bioprosthesis dysfunction was detected by using transesophageal echocardiography; the good clinical and echocardiographic outcome while the patient was receiving anticoagulant therapy suggested the thrombotic aortic restenosis, which was not confirmed by histology or by visual inspection because open-heart surgery was not needed. Moreover, we evaluated the ability of intracardiac echocardiography to detect a late aortic Sapien valve dysfunction and to
explain its origin. Intracardiac echocardiography is an attractive technique to assist the procedure of transcatheter aortic valve implantation because it can be done without requiring general anesthesia and the catheter can be left in place in the right atrium without interfering with the view of the native aortic valve. In addition, this case suggests that intracardiac echocardiography is an accurate imaging technique to detect and explain the reason for a late aortic bioprosthesis dysfunction.

**Conclusion**

Thrombotic aortic restenosis after transcatheter aortic valve replacement with the Sapien valve is an uncommon situation, not yet well described, which can be successfully treated by anticoagulant therapy. Intracardiac echocardiography, an accurate technique to assist the procedure of transcatheter aortic valve implantation, could also be instrumental in diagnosing late aortic bioprosthesis dysfunction.

**Disclosures**

None.

**References**


**KEY WORDS:** echocardiography, thrombosis, valvuloplasty, aortic valve, transcatheter aortic valve implantation
Thrombotic Aortic Restenosis After Transapical Sapien Valve Implantation
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doi: 10.1161/CIRCINTERVENTIONS.109.935031
Circulation: Cardiovascular Interventions is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-7640. Online ISSN: 1941-7632

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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