Three mechanisms of early failure of transcatheter aortic valves: Valve thrombosis, cusp rupture, and accelerated calcification

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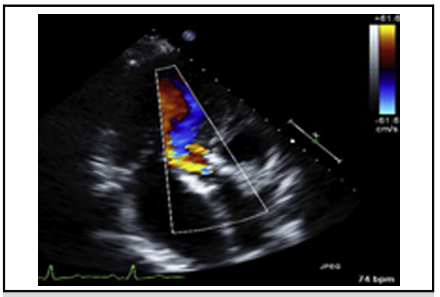

TTE of a distinct mechanism of early TAVR failure: thrombosis.

Central Message

We describe 3 cases of overt, early TAVR failure via 3 distinct mechanisms and review the literature on TAVR degeneration.

Video clip is available online.

Transcatheter aortic valve replacement (TAVR) is now a well-established therapy for patients with high or prohibitive surgical risk and severe aortic stenosis. Expanding indications include valve-in-valve treatment of bioprosthetic valve degeneration, predominant aortic regurgitation, and, more recently, treatment of patients at increasingly lower surgical risk and younger ages. Although short and intermediate follow-up studies of early-generation Edwards SAPIEN[1,2] (Edwards Lifesciences, Irvine, Calif) and SAPIEN XT[3] valves consistently have shown that clinically significant early valve degeneration is exceedingly rare, given the continued increase in TAVR volume, clinicians should be aware of the mechanisms for these uncommon presentations. Accordingly, we present 3 clinical cases of early TAVR degeneration from 3 distinct mechanisms.

CASE 1: VALVE THROMBOSIS

A 92-year-old woman presented for evaluation of progressive, mild dyspnea on exertion. She had undergone TAVR 4 years previously with a 23-mm Edwards SAPIEN XT valve for severe, calcific aortic stenosis. To evaluate her symptoms, a transthoracic echocardiogram (TTE) was obtained, which demonstrated a 0.8 × 0.5-cm supravalvular thrombus on the anterior cusp of the bioprosthetic valve (Figure 1 and Video 1). Peak and mean transaortic valvular gradients were elevated at 42 and 22 mm Hg, respectively (peak velocity 3.3 m/s; dimensionless index 0.33). Moderate aortic regurgitation was noted with a larger central jet and a smaller anterior paravalvular jet. On review of the patient's TTE 1 year previously, the bioprosthetic valve was functioning well with trivial aortic regurgitation, as well as peak and mean transprosthetic aortic valvular gradients of 26 and 15 mm Hg, respectively, compared with gradients of 14 and 8 mm Hg, respectively, immediately post-TAVR. Clinically, she had no features suggestive of infectious endocarditis, and her blood cultures were negative. The patient had been maintained with dual antiplatelet therapy after TAVR. On discovery of valve thrombosis, anticoagulation was started with plans to follow her clinically and with serial echocardiography.

CASE 2: CUSP RUPTURE

A 95-year-old woman presented for evaluation of rapidly progressive dyspnea, initially present with minimal exertion and now with symptoms at rest. She underwent TAVR 3 years previously with a 23-mm Edwards SAPIEN XT valve for severe calcific aortic stenosis. A TTE demonstrated severe aortic regurgitation secondary to a flail posterior prosthetic valve leaflet (Figure 2 and Video 2) that was new compared with the most recent TTE from 1 year previously. Her TTE was otherwise notable for mild left ventricle dilation with mildly reduced systolic function (left ventricular ejection fraction 45%-50%). There were no visible vegetations, she had no systemic or constitutional symptoms, and blood cultures were negative. Severe leaflet calcification was absent, and the mobility of the other leaflets

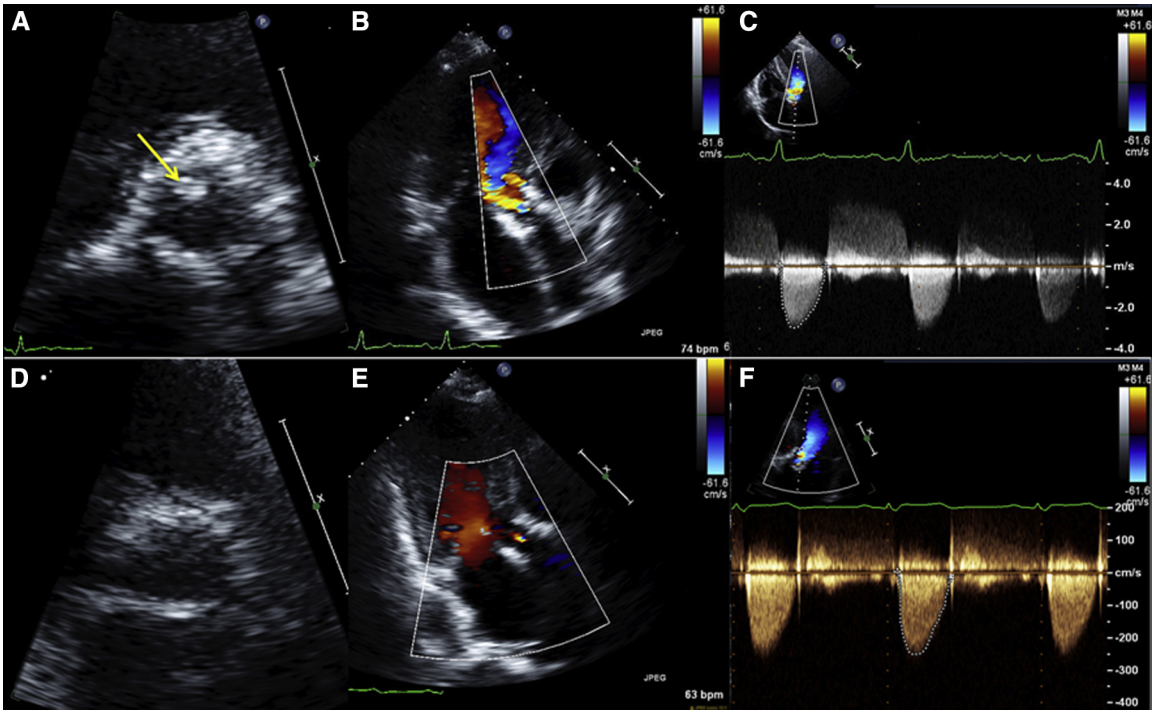

FIGURE 1. TTE showing 0.8 × 0.5-cm supravalvular thrombus (*yellow arrow*) on bioprosthetic SAPIEN XT (Edwards Lifesciences, Irvine, Calif) valve in parasternal short-axis view (A). TTE with color Doppler during diastole in apical long-axis view demonstrating associated aortic insufficiency (B). TTE with continuous-wave Doppler in apical long-axis view demonstrating mild-moderate aortic insufficiency and increased velocity across the valve (C, peak velocity of 3.3 m/s and peak and mean gradients of 42 and 22 mm Hg, respectively). Correlative TTE images from 1 year previously (3 years post-TAVR) demonstrating the lack of thrombus, valve dysfunction, and lower transvalvular peak velocity and gradients (D-F).

was normal. Therefore, the mechanism was presumed to be structural failure. The patient was managed expectantly on the basis of goals of care discussions.

CASE 3: ACCELERATED CALCIFIC DEGENERATION IN RADIATION-ASSOCIATED HEART DISEASE

A 62-year-old woman presented for evaluation of dyspnea on exertion and orthopnea that had progressed over a 6-month period. She underwent TAVR 5 years previously with a 23-mm Edwards SAPIEN valve for severe aortic stenosis. Her valvular disease developed in the setting of prior mantle radiation for the treatment of Hodgkin's lymphoma at age 15 years. She had several comorbidities, including severe chronic obstructive pulmonary disease, systemic scleroderma, and paroxysmal atrial fibrillation with a prior stroke. A TTE demonstrated new severe mixed bioprosthetic regurgitation and stenosis (peak and mean gradients of 80 and 46 mm Hg, respectively; peak velocity 4.5 m/s; dimensionless index of 0.15), secondary to severely restricted prosthetic valve leaflet motion from accelerated calcific degeneration (Figure 3 and Video 3). There was also moderate (2-3+) mitral regurgitation and moderate mitral stenosis with thickening of the aorto-mitral intervalvular fibrosa. There was no echocardiographic or clinical findings to suggest prosthetic valve endocarditis. Given the comorbidities and radiation-associated heart disease that limited her surgical candidacy during her native valvular degeneration, she again was deemed suitable for only percutaneous valve therapy and underwent successful transfemoral, valve-in-valve implantation of a 23-mm Edwards SAPIEN XT valve. She had an uneventful postprocedural course, and her symptoms improved to New York Heart Association class I.

DISCUSSION

With increasing indications for TAVR, identifying and understanding associated complications, such as early valvular dysfunction, are increasingly important. Short and intermediate follow-up studies of early-generation Edwards SAPIEN[1,2] and SAPIEN XT[3] valves have consistently shown that clinically significant early valve degeneration is rare and has decreased further with improved valve design in newer-generation valves and with increased experience with TAVR.[4,5] Nonetheless, distinct, rare causes of early failure have been identified, including structural dysfunction, thrombosis, prosthetic endocarditis, compression, and late migration.[6]

In the surgical literature, valve degeneration is invariably defined by the necessity for reoperative valve replacement