An 86-year-old man with severe symptomatic aortic stenosis (additive Euroscore 7) underwent a transcatheter aortic valve implantation with a CoreValve bioprosthesis (26 mm, CoreValve Revalving Technology, Medtronic, Inc., Minneapolis, MN). The patient was discharged on aspirin and clopidogrel and advised to stop the clopidogrel after 3 months. This first 6-month follow-up was uneventful. Repeated echocardiograms obtained during this period revealed a slight increase in mean transprosthetic aortic pressure gradient without significant concomitant aortic regurgitation. At the last visit, 1 year later, the patient became symptomatic (New York Heart Association class II–III). He had no fever. Between the 6th and 12th month of follow-up, there was a documented increase in transprosthetic aortic peak velocity (4.11 m/s) and mean gradient (41 mm Hg) and a significant reduction in aortic effective orifice area (0.69 cm²), which indicated severe prosthetic valve stenosis. To evaluate the shape of the CoreValve, both transesophageal echocardiogram and cardiac computed tomography were performed. Although computed tomography revealed no change in prosthesis position (when compared with pre–transcatheter aortic valve implantation computed tomography) and the absence of significant deformation of the stent by any calcifications (Figure 1A–1D), transesophageal echocardiogram evidenced thickened hypomobile aortic valvular leaflets, with aliasing on Color Doppler imaging beginning just at the level of valve leaflets (Figure 1E–1F; Movie in the online-only Data Supplement). No vegetation was identified, and there was no appearance suggestive of thrombus, and no attempt was made to manage the obstruction with anticoagulant therapy. The patient underwent successful surgical bioprosthetic valve replacement. The in situ visual inspection of the implant revealed a normal seating of the CoreValve with a translucent neointimal sheath covering the upper portion of the nitinol frame. The free edges of the valve leaflets were thin, and no calcifications were noticed. The leaflets were almost immobile because of the presence of a brown thrombotic host tissue covering exclusively the aortic side (Figures 2 and 3). Glistening white fibrous tissue covered the fabric skirt of the inflow portion of the device on outer and inner surfaces and formed a ridge of pannus extending into the inflow orifice and partially onto the ventricular surface of the valve cups. Pannus remained attached to the frame on the outflow, extending to the commissural areas, partially covering the free margins, resulting with restricted leaflet movement. After a long stay in the intensive care unit, the patient was discharged. Several

**Figure 1.** Cardiac computed tomography (CT; A–D) and transesophageal echocardiography (TOE; E–F) obtained late after CoreValve implantation showing no prosthesis displacement, no leaflet calcifications, and the absence of thrombus. Sagittal (A), coronal (B), and short-axis (C) CT views showing no prosthesis displacement and the absence of leaflet calcifications. Short- (E) and long-axis (F) TOE views showing thickened aortic cusps (arrows), no leaflet calcifications, no thrombus, limited leaflet mobility, and abnormal flow pattern across the aortic valve.

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months after the surgical procedure, the patient was well and the control echocardiogram showed good function of the new implanted bioprosthesis. Although we are not aware of any prior descriptions of severe thrombosis of a CoreValve aortic bioprosthesis, this case clearly demonstrates that this complication can occur early after successful implantation. A surgical extraction of the CoreValve was successfully performed, and the initial postoperative period was relatively uneventful. Although CoreValve degeneration has mainly been described during long-term follow-up, little is still known on the natural history of percutaneous implanted valve. Currently, 2 types of percutaneous valves are available: the CoreValve and the Edwards Sapien valve (Edwards Lifesciences Inc, Irvine, CA), which have different structural (bovine valve for Edwards Sapien; porcine pericardium for CoreValve) and hemodynamic (lower leaflet stress with the CoreValve) properties. Whether these differences will affect durability is unknown. In the present case, the reduced performance of the CoreValve was mainly attributed to mural thrombus on aortic surfaces of valve cusps and partly to fibrous pannus overgrowth on stent frame inflow orifice. The reason for such valve deterioration remains to be determined, and there was no evidence of inflammatory disease or prothrombotic disease. Of note, radiography of the explanted materials showed no evidence of mineralization/calcification in the valve or host tissue. This latter observation contrasted with the 2 cases of CoreValve degeneration reported after 5-year implantation, in which extensive calcifications were noted. Instead, it is in line with recently reported cases of reversible Edwards Sapien XT dysfunction attributable to prosthesis thrombosis. In our case, the progressive increase in aortic pressure gradients and the absence of imaged thrombosis first suggested an early prosthesis structural degeneration, and no anticoagulation treatment was attempted. It is possible that anticoagulation therapy would have resolved the problem. On the basis of this experience, valve thrombosis should be suspected in cases of early transcatheter aortic valve implantation dysfunction with recurrent obstruction, and anticoagulation therapy may be indicated as the first line therapy in hemodynamically stable patients, even if thrombus is not visualized on imaging studies.

Disclosures
Dr Legrand is a proctor for Medtronic. The other authors have no conflict to report.

References

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SUPPLEMENTAL MATERIAL

Video Legend

Long- (video: Top) and short-axis (video: Bottom) TOE views showing thickened aortic cusps, no leaflet calcifications, no thrombus, limited leaflet mobility and abnormal flow pattern across the aortic valve.