Aortic valve replacement in the setting of aortic valve endocarditis frequently is managed with an aortic homograft. However, late operation is not uncommon and may be very difficult because of calcification of the wall of the aortic homograft and the valve leaflets.1–3 We report a patient with a heretofore unreported complication with near-complete obstruction of his aortic homograft.

A 38-year-old man was referred to the Bluhm Cardiovascular Institute at Northwestern University for surgical management of suspected aortic prosthesis dysfunction. He had undergone aortic valve replacements at 16 and 24 years of age, and at 25 years of age, he developed prosthetic valve endocarditis, which was treated with cryopreserved homograft aortic valve and root replacement. He required partial sternectomy for sternal osteomyelitis weeks later and an implantable cardioverter-defibrillator for perioperative ventricular arrhythmias. He recovered fully. In 2009, the patient developed weekly episodes of numbness, parasthesia, and weakness lasting from 15 minutes to several hours. In the fall of 2010, progressive fatigue and dyspnea on exertion progressed to near syncope. Echocardiographic (Figure 1) and computed tomography (Figure 2) scans suggested obstruction of the aortic homograft. At Northwestern, echocardiographic imaging from the apical transducer position demonstrated no aortic stenosis or regurgitation with a mean gradient of 6 mm Hg across the valve.

Figure 1. Preoperative continuous-wave Doppler studies of the aortic valve and ascending aorta from various transducer positions. A, Aortic valve from the apical transducer position showing a peak velocity of ~1.7 m/s, indicating a normal peak gradient (12 mm Hg) across the aortic valve. B, Aortic valve and ascending aorta from the left clavicular transducer position showing a peak velocity of ~2.8 m/s, indicating a mildly increased peak gradient (31 mm Hg) across the aortic valve and ascending aorta. C, Aortic valve and ascending aorta from the right clavicular transducer position showing a peak velocity of ~3.5 m/s, indicating a moderately to severely increased peak gradient (49 mm Hg) across the aortic valve and ascending aorta. D, Aortic valve and ascending aorta from the suprasternal notch transducer position showing a peak velocity of ~4 m/s, indicating a severely increased peak gradient (64 mm Hg) across the aortic valve and ascending aorta. Peak velocity is best recorded by continuous-wave Doppler from the suprasternal notch approach, illustrative of extensive obstruction in the aortic graft.
However, imaging from the suprasternal transducer position detected a peak velocity of 4 m/s in the ascending aorta, consistent with a high-grade stenosis above the aortic valve (Figure 1D). A gated computed tomography scan demonstrated a densely calcified aortic graft with focally narrowed lumen (as narrow as 1 cm; Figure 2) in its mid to distal portion at the level of the pulmonary artery bifurcation. There was no indication of coronary artery stenosis. A brain computed tomography scan showed numerous small old embolic lesions. In January 2011, he underwent a fourth cardiac operation with femoral artery and vein cannulation. The aorta was cross-clamped distal to the aortic homograft at the base of the innominate artery. The native aorta was transected just beyond the prior distal anastomosis. The distal several centimeters of the homograft were 80% obstructed with granular, exophytic, sand-like calcification (Figure 3 and Movie I in the online-only Data Supplement). The proximal homograft was transected where the lumen was normal diameter, just distal to the sinotubular junction (Movie I in the online-only Data Supplement). Calcification of the proximal remaining wall of the homograft extended to the base of the aortic valve leaflets, but otherwise, the leaflets appeared normal. The wall of the proximal portion of the homograft was decalcified with the Cavitron Ultrasonic Surgical Aspirator (Tyco Healthcare, Mansfield, MA) to facilitate anastomosis. A 28-mm graft was used to replace the ascending aorta from the level of the sinotubular junction to the distal ascending aorta. Pathology disclosed medial nodular calcification and severe hyalinization of the aortic wall with luminal calcific debris (Figure 4). The patient was discharged 4 days after surgery. At follow-up at 3 months, he had no further neurological episodes and had returned to New York Heart Association functional class I.

Aortic valve homograft durability has been shown to be similar to that of bovine pericardial aortic valve replacement, but many consider it the valve of choice when faced with endocarditis. Reoperation is known to be technically very challenging as a result of calcification of the homograft wall. However, we have not previously encountered luminal obstruction of the homograft, and we were not able to find prior reports by searching the literature search or by contacting other experts in valve surgery. Why this patient developed granular calcification that embolized is unknown but perhaps is related to the infection at the time of homograft implantation and an ongoing inflammatory response. However, we found neither inflammation nor microorganisms in the specimen. Although there is some debate about inflammation and valve homograft dysfunction, numerous reports implicate inflammatory processes in bioprosthetic valve failure. We believe that inflammatory processes were responsible for the pathology seen in this case owing to the severe hyalinization and the rapid development, luminal projections, and severe extensive calcification of the aortic graft. The extent of calcification and hyalinization is rare after homograft implantation. Unfortunately, there is no way to deduce the origin of these processes at this stage. Echocardiography was difficult in this setting because of the atypical location of obstruction,
and a computed tomography scan clarified the location and calcific nature of the obstruction.

**Disclosures**

None.

**References**


**Figure 4.** Hematoxylin and eosin stain demonstrating extensive calcification and severe hyalinization of the aortic homograft. Inflammation was absent from sections of the aorta. **A**, Nodular calcification was seen focally in the aortic wall graft. **B**, Some additional sections of the aortic homograft exhibited indistinct layers and severe hyalinization containing granular, exophytic calcification causing marked distortion and projecting into the luminal graft. Original magnification ×100.
Near-Complete Obstruction of an Aortic Homograft

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