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Periaortic Hematoma Formation Leading to Aortic Valve Failure*

A Complication of Homograft Placement for Second Valve Surgery

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The aortic homograft has become the replacement valve of choice in the treatment of complicated endocarditis involving native and prosthetic aortic valves. Complications are rare, typically involving chronic leaflet degeneration causing valvular insufficiency or rarely chronic calcific stenosis. We present a case in which functional stenosis of the homograft valve was caused by compression and distortion by blood transmitted directly from the left ventricle into a space between the homograft and an external cavity formed by a Dacron wrap. The latter had been placed to help control suture-line bleeding. This case presentation demonstrates an unusual cause of homograft failure and suggests that wrapping of a homograft conduit by native aorta or an external Dacron wrap is not a substitute for meticulous surgical technique to assure a hemostatic suture line. (Chest 1992; 102:1299-1301)

Experience with aortic valve homografts began nearly 35 years ago and advances in graft preservation and storage procedures have stimulated more widespread use of this prosthesis.1 The aortic valve homograft has been particularly valuable in the treatment of native and prosthetic aortic valve endocarditis complicated by valvular insufficiency or perivalvular abscess formation.2 These prostheses have the potential for failure principally related to gradual valve wear leading to aortic insufficiency and rarely calcific stenosis.3,5

Meticulous surgical technique is an essential component to placement of the homograft valve, but this may prove extremely difficult even for the experienced surgeon in the presence of extensive tissue destruction associated with complicated aortic valve endocarditis. We present a patient who developed relatively early aortic homograft stenosis and insufficiency due to external compression and distortion of the homograft conduit by an external Dacron wrap that had been placed to assist with hemostasis.

Case Report

A 45-year-old man initially presented eight years prior to the current hospital admission with enterococcal endocarditis complicated by acute aortic insufficiency and left ventricular failure. Aortic valve replacement was uneventfully performed with a No. 27 Bjork-Shiley prosthesis. Seven years later, he presented with significant prosthetic aortic valvular insufficiency. Evaluation revealed a subvalvular abscess cavity, partial dehiscence of the prosthetic valve, and aortic annular destruction. Considering the extensive destruction of the aortic annulus in the face of prosthetic valve infection, the aortic root was replaced with a 24-mm aortic valve homograft conduit into which the left and right coronary arteries were

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Figure 1. Simultaneous pressure tracings from the left ventricle (LV), central aorta (Ao), and pulmonary artery (PA) demonstrate a 100 mm Hg peak-to-peak pressure gradient across the stenotic homograft aortic valve.
irregular luminal contour (Fig 2). An ill-defined collection of contrast was seen extrinsic to the homograft, extending from above the coronaries into the left ventricle. Mild narrowing of the distal end of the homograft was noted at the anastomosis with the ascending aorta. A ring of nonopacification was seen at the proximal end, where the homograft was anastomosed to the left ventricular outflow tract. Dynamic contrast-enhanced computed tomography of the chest demonstrated an area of localized extrinsic compression of the homograft at the level of the aortic valve ring (Fig 3).

The patient was taken to the operating room for surgical revision of the homograft. An extracardiac chamber, in continuity with the left ventricle, was found between the aortic homograft and a Dacron wrap surrounding the aortic root. The aortic valve within the homograft was friable in appearance, but not in itself stenotic. Functional aortic stenosis appeared to result from distortion of the homograft by blood flow into the extrinsic chamber from the left ventricle. This process also resulted in noncoaptation of the aortic valve leaflets and significant aortic insufficiency. The homograft and Dacron wrap were excised and a valve conduit (Medtronic-Hall) containing a No. 24 valve was placed, with reimplantation of the coronary arteries. The patient’s postoperative course was uneventful.

**DISCUSSION**

The aortic valve homograft is considered by many investigators to be the replacement valve of choice in complicated endocarditis involving a native or prosthetic aortic valve.\(^5\) Reported complications of aortic homograft placement include trauma to the conduction system, bleeding from the suture line, intracardiac fistulas; malalignment and torsion of the coronary arteries, and obvious mortality risks related to surgery.\(^6\)

Long-term valve failure is typically gradual, with mild valvular insufficiency present for two or more years before progressing.\(^7\) Significant homograft valve stenosis is rare and gradients across aortic valve homografts generally do not increase over time.\(^8\) When leaflet calcification does develop, it usually occurs as small, discrete, cauliflower-like spicules\(^1\) that predispose to leaflet rupture, so that patients generally present with valvular incompetence rather than stenosis.

Our patient experienced both bleeding from the suture lines, which necessitated the placement of a Dacron wrap around the aortic root, and the development of intracardiac fistulas, presumably associated with the perivalvular tissue infection. We speculate that the intracardiac fistulas leading to communication between the left ventricle and the chamber between the Dacron aortic root wrap and the aortic root evolved over approximately 16 months following homograft placement. The continuity between the left ventricle and this extra-aortic space caused compression of the homograft, disrupting the valve architecture and resulting in hemodynamically significant stenosis and insufficiency of the valve.

In conclusion, this report emphasizes the potential for homograft failure due to extrinsic compression resulting in distortion of the valve cusps, causing stenosis and insufficiency. When this valve is used in patients with complicated endocarditis, the infected friable tissues may be predisposed to persistent postoperative bleeding as well as later fistula formation. This case argues against using an external wrap for control of intraoperative bleeding. Meticulous surgical techniques, including careful placement of sutures and the use of a felt strip around the proximal suture line, might permit better hemostasis and obviate the need for an
external wrap.

REFERENCES

Diffuse Alveolar Hemorrhage Secondary to Superwarfarin Ingestion*

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A 27-year-old woman with severe vitamin K deficiency presented with hemoptysis and diffuse pulmonary infiltrates. She rapidly developed respiratory failure requiring ventilatory support. Surrupitious ingestion of brodifacoum, a long-acting warfarin derivative, was ultimately found to be the cause of her coagulopathy and DAH.

(Chest 1992; 102:1301-02)

APPT = activated partial thromboplastin time; DAH = diffuse alveolar hemorrhage; JVD = jugular venous distention

A new class of rodenticides with powerful anticoagulant effects has been developed. These derivatives of warfarin have been called "superwarfarins" and exhibit enhanced potency and prolonged duration of action. Hemorrhagic complications of superwarfarins recently have been presented; however, no cases of pulmonary hemorrhage have been reported.1,3 We present a case of brodifacoum (D-Con) ingestion leading to prolonged, severe coagulation abnormalities and DAH. This case also illustrates the difficulty that can arise in diagnosing surreptitious superwarfarin ingestion.

CASE REPORT

A 27-year-old Hispanic woman, a ½ pack-per-day cigarette smoker, presented with fever, dyspnea and cough productive of ⅔ cup of blood. She denied chest pain, prior hemoptysis, or ingestion of aspirin, anticoagulants or illegal drugs.

Seven months previously she developed profuse vaginal bleeding and a coagulopathy was discovered. Factor analysis showed levels of factor II, 3 percent; factor VII, 5 percent; factor IX, 2 percent; and factor X, 1 percent. Severe vitamin K deficiency was found to be the cause; however, the etiology of her vitamin K deficiency could not be established. She had a rapid but short-lived response to vitamin K1 and required 40 mg intravenously three times a day for control of her coagulopathy. In subsequent months she suffered a thrombotic stroke, epistaxis and soft tissue hematomas. She did not take her vitamin K in the five days prior to the current admission.

On examination she was an ill-appearing woman with a respiratory rate of 50 breaths per minute, blood pressure of 140/90 mm Hg, a heart rate of 150 beats per minute and a temperature of 40° C. There were no skin lesions, JVD, third heart sound or edema. Lung examination showed bilateral basilar crackles. Laboratory values revealed a hemoglobin value of 10.1 mg/dl, WBC, 16.4/cu mm with 89 percent neutrophils and 8 percent band cells. The platelet count was 389,000 /cu mm. Her PT was 84 s (control of 12 s), and her aPTT was 65 s (control of 28 s). Arterial blood gas values, while breathing 50 percent O2 by face mask, showed a pH of 7.43; Po2, 27 mm Hg; Pco2, of 59 mm Hg. A chest x-ray showed diffuse alveolar infiltrates (Fig 1).

She continued to have hemoptysis and rapidly developed respiratory failure requiring intubation. Large amounts of blood were suctioned from the endotracheal tube. Postintubation ABC values were breathing 60 percent O2 were pH, 7.42; Pco2, 36 mm Hg; and Po2, 60 mm Hg. She was treated with fresh frozen plasma and 60 mg of vitamin K, intravenously. Her PT returned to 15.8 s (control of 12.9 s) within 30 min.

She required ventilatory support with 15 cm H2O of PEEP and an Fio2 of 0.6 for ten days. Her coagulopathy was controlled with 40 mg of vitamin K, given intravenously three times a day. She had no further episodes of bleeding. She was extubated on her 13th hospital day and discharged nine days later while receiving 40 mg of vitamin K, SQ three times a day. Her room air ABG prior to discharge showed a pH of 7.48; Pco2, 31 mm Hg; and Po2, 67 mm Hg.

Subsequently, she admitted ingesting D-Con rat poison, containing brodifacoum, because of depression. Blood samples showed high levels of brodifacoum. She is currently undergoing psychiatric treatment.

DISCUSSION

Diffuse alveolar hemorrhage is characterized by wide-