FIGURE 1. Digital subtraction angiogram showing filling of a branch of the right bronchial artery from the thoracic aorta (A) and filling of the hemangiomatic portion through the right bronchial artery (B).

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Early calcification of a stentless pericardial bioprosthesis in the elderly

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The Sorin Freedom pericardial valve (SFP, Sorin BiomedicaCardio, Saluggia, Italy), a stentless bioprosthesis made of glutaraldehyde-tanned bovine pericardium, was introduced clinically in 1991.¹ In 2000, detoxification treatment with homocysteic acid was added to eliminate glutaraldehyde toxicity and mitigate tissue calcification. The SFP has shown excellent overall results at medium term with no cases of structural failure.² We report the first case of early SFP calcification in an elderly recipient.

CLINICAL SUMMARY

An 84-year-old woman underwent aortic valve replacement in 2000 with a 25-mm SFP, implanted with 3 continuous sutures of 4-0 polypropylene to secure the inflow and outflow rims; intraoperative echocardiography showed a competent valve. Approximately 6 years later she was hospitalized for acute pulmonary edema. A harsh precordial systolic murmur was audible, and electrocardiography showed sinus rhythm and left ventricular hypertrophy. Echocardiography revealed calcified valve cusps with a peak transvalvular gradient of 100 mm Hg and a valve area of 0.8 cm². No evident risk factors for bioprosthetic calcification were detected; renal function was normal (serum creatinine 0.78 mg/dL) with no hyperlipidemia, and hyperparathyroidism was excluded by normal calcium and phosphate values. At reoperation, the prosthesis appeared incorporated into the aortic wall; all cusps were calcified (Figure 1), and calcification extended to the outflow of the prosthesis preventing its complete removal. The 3 cusps were separately excised leaving a rigid diminutive aortic

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FIGURE 1. Gross (A), x-ray (B), and histologic (C) views of the explanted cusps with massive calcification (von Kossa stain, magnification $\times 6$).

annulus; this was enlarged with a patch of bovine pericardium allowing the insertion of a 21-mm Perimount Magna pericardial bioprosthesis (Edwards Lifesciences, Irvine, Calif). The patient recovered uneventfully.

DISCUSSION

Intrinsic calcification is the leading cause of bioprosthetic failure and reoperation. In the elderly, mineralization of bioprostheses occurs slowly, thus justifying their extended use in this patient subset because the prosthesis will most likely outlive the recipient.³ Stentless bioprostheses have been reported to be hemodynamically superior to stented bioprostheses and with potentially longer durability, thus gaining popularity in recent years as a valve substitute in the elderly.⁴

The SPF is a stentless bioprosthesis made entirely of bovine pericardium with excellent hemodynamic performance and durability at medium term, with no reports of valve mineralization. The present case shows 2 interesting features. First, it describes early bioprosthetic calcification in an elderly patient without evident risk factors after aortic valve replacement with a first-generation SPF, a prosthesis without any antimineralization treatment. Bioprosthetic calcification in the elderly is considered exceptional.⁵ Because such a complication has not been reported even in large series of patients receiving an SPF,^{2,4} our case may be an occasional finding. Second, it underlines the potential complexity of replacing a stentless valve, which might require a more technically demanding procedure. In our case, the

presence of a small, rigid aortic annulus mandated an annular enlargement procedure to insert a prosthesis of adequate size, and calcification of the prosthesis outflow discouraged the use of a second stentless valve.

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Acute rupture of both papillary muscles after aortic valve replacement for infective endocarditis

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Surgical treatment of active infective valvular endocarditis is associated with substantial morbidity and mortality, particularly when the infection spreads below the primary infected valve. We report the case of a patient in whom acute mitral regurgitation (MR) developed secondary to the rupture of both papillary muscles (PMs) after aortic valve replacement (AVR) for infective endocarditis.

CLINICAL SUMMARY

A previously healthy 23-year-old morbidly obese man was transferred to our hospital with a 5-day history of left-sided chest pain, dyspnea, cough, and fever. On arrival, his blood pressure was 120/48 mm Hg, with a regular pulse rate of 148 beats/min, a temperature of 38.6°C, a respiratory rate of 26 breaths/min, and an oxygen saturation of 86% on room air. The only remarkable physical findings were a grade III/VI long, diastolic, blowing murmur at the right second inter-space and a progressively decreasing level of consciousness, with no focal neurologic signs. After intubation, a transesophageal echocardiogram revealed the presence of a 1 × 3-cm vegetation involving the right and left aortic leaflets and associated with severe aortic insufficiency with an ejection fraction of 30%. There was no evidence of abscess, and the anterior mitral leaflet was intact with minimal MR.

The patient was taken to the operating room for an emergency AVR. Under cardiopulmonary bypass, an aortotomy was performed and the aortic valve (AV) was excised. A 2-cm aortic abscess involving the ventricular septum was identified. After thorough debridement of all the infected tissue, reconstruction of the aortic annulus was achieved using bovine pericardium followed by insertion of a number 21 St Jude mechanical valve (St Jude Medical Inc, St Paul, Minn).

Postoperatively, the patient's condition gradually improved. However, on postoperative day 4, the patient's fever spiked to, his white count increased to 37.5, his systolic pressure decreased to 70 mm Hg, and his wedge pressure increased to 28 mm Hg. Shortly thereafter, cardiac arrest occurred and the patient died after attempted resuscitation for more than 30 minutes.

Examination of the heart at autopsy revealed a competent AV with no vegetation or thrombus. However, both the anterior and posterior PMs were ruptured and the posteroseptal myocardium contained a 2.5 × 1.5-cm abscess (Figure 1). Histologic examination showed myocardial necrosis and marked acute inflammation of both papillary heads, consistent with septic embolization of the small coronary branches supplying these muscles. Gram-positive cocci, consistent with Group B streptococcus found in blood cultures, were identified in the necrotic tissue. Additional foci of septic embolization were seen in the kidneys and spleen.

DISCUSSION

Aortic root infection occurs in one third of all patients with native valve endocarditis and is associated with substantial morbidity and mortality.¹ The major determinants for the successful surgical management of these cases are the complete exclusion and thorough debridement of the infected cavities.² MR in the setting of AV endocarditis is

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