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.

References

1. Stacchino C, Bona G, Rinaldi S, Vallana F. Design and performance characteristics of the Pericarbon stentless valve. *J Heart Valve Dis.* 1995;4(Suppl I):S102-5.
2. D'Onofrio A, Auriemma S, Magagna P, Favaro A, Cannarella A, Piccin C, et al. Aortic valve replacement with the Sorin Pericarbon Freedom stentless prosthesis: 7 years' experience in 130 patients. *J Thorac Cardiovasc Surg.* 2007;134:491-5.
3. Bortolotti U, Sciotti G, Milano A, De Carlo M, Codecasa R, Nardi C, et al. Performance of 21-mm size Perimount aortic bioprosthesis in the elderly. *Ann Thorac Surg.* 2000;69:47-50.
4. Nyawo B, Graham R, Hunter S. Aortic valve replacement with the Sorin Pericarbon Freedom stentless valve: five-year follow-up. *J Heart Valve Dis.* 2007;16:42-8.
5. Polo ML, Legarra JJ, Vilar M, Cabrera A, Duran D, Pradas G. Early calcification of a Carpentier-Edwards Perimount mitral valve in an elderly woman. *J Thorac Cardiovasc Surg.* 2002;124:1043-4.

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

Rony Atoui, MD, MSc,^a Tarek Hijal, MD,^a Richard Fraser, MD,^b and Dominique Shum-Tim, MD, MSc,^a Montreal, Quebec, Canada

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

From the Division of Cardiac Surgery^a and Department of Pathology,^b McGill University Health Center, Montreal, Quebec, Canada.

No financial associations or conflicts of interest to disclose.

Received for publication March 12, 2008; revisions received March 12, 2008; accepted for publication March 21, 2008.

Address for reprints: Dominique Shum-Tim, MD, MSc, Division of Cardiothoracic Surgery, McGill University Health Center, 1650 Cedar Avenue, Montreal, Quebec, H3G 1A4, Canada (E-mail: dshumtim@yahoo.ca).

J Thorac Cardiovasc Surg 2009;137:1275-6
0022-5223/\$36.00

Copyright © 2009 by The American Association for Thoracic Surgery
doi:10.1016/j.jtcvs.2008.03.043

FIGURE 1. A, Mitral valve showing rupture of posterior and anterior PMs (*). Note the ragged borders of the rupture sites and the tan, infarcted areas with hyperemic borders. B, Cross-section of mid-heart showing an abscess in the posteroseptal region. C, Section of myocardium showing coagulative necrosis surrounded by a dense neutrophil infiltrate ($\times 25$). D, Gram stain demonstrating abundant gram positive cocci predominantly in chains ($\times 600$).

a potential complication but has not been described as occurring secondary to bilateral PM rupture.

The clinical presentation of PM rupture is closely linked to the blood supply of the anterolateral and posteromedial PMs. Because of its single blood supply, the posteromedial PM is most frequently involved. Both muscles, however, are vulnerable to ischemic insult resulting from embolic occlusion or hypoperfusion of the terminal intramyocardial arterial branches. Our patient's course is similar to that of 2 other cases of PM rupture caused by coronary embolism in patients who underwent AVR for endocarditis.^{3,4} The main difference is that our patient had rupture of both PMs, explaining the postoperative deterioration of the patient's condition. There is only 1 previously reported fatal case of rupture of both PMs in the setting of acute myocardial infarction.⁵

The exact mechanism of bilateral PM rupture in our patient is difficult to explain and could not be defined by echocardiography because of the sudden onset of cardiac arrest. However, the autopsy findings and temporal evolution of MR suggest that its cause is most likely a combination of an occult infection of the PM and an ischemic insult caused by small-vessel embolism from AV endocarditis.

CONCLUSIONS

Infective endocarditis of the AV may be complicated by a subvalvular abscess involving the intervalvular fibrosa, a coronary embolism, or the rupture of one PM. Each of these complications has been described individually, but the rate at which they occur simultaneously is low, especially when both PMs are affected, and survival in such a case is unlikely. Nevertheless, a high index of suspicion allowing early recognition and prompt surgical intervention is the only hope to try to salvage this rare complication of bacterial endocarditis.

References

1. Siniawski H, Grauhan O, Hofmann M, et al. Aortic root abscess and secondary infective mitral valve disease: results of surgical endocarditis treatment. *Eur J Cardiothorac Surg.* 2005;27:434-40.
2. David TE, Bos J, Christakis GT, Brofman PR, Wong D, Feindel CM. Heart valve operations in patients with active infective endocarditis. *Ann Thorac Surg.* 1990;49:701-13.
3. Jinno T, Tago M, Yamane M, Nakagawa J, Ishiai S. A case of infective endocarditis with subtotal rupture of the posterior papillary muscle. *Kyobu Geka.* 1995;48:487-90.
4. Edwards J. Mitral insufficiency resulting from "overshooting" of leaflets. *Circulation.* 1971;43:606-12.
5. Patel AD, Abo-Auda W, Chowdhury N, et al. Rupture of both papillary muscles after acute myocardial infarction: a case report. *Heart Dis.* 2002;4:285-7.