Case Report

A Case of Transient Bioprosthetic Valve Regurgitation and Hemolysis Devoloping Early after Surgery Using Carpentier-Edwards Valve

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The Carpentier-Edwards pericardial bioprosthesis has been markedly improved in the long-term results and valve-related complications including valve dysfunction, compared to the previous generation bioprosthesis. We report a patient in whom transient prosthetic valve regurgitation and hemolysis occurred early after mitral valve replacement using a Carpentier-Edwards pericardial bioprosthesis and were resolved by preservative therapy.

The patient was a 77-year-old female diagnosed with severe mitral valve stenosis and insufficiency. She underwent mitral valve replacement with a Carpentier-Edwards pericardial bioprosthesis. Opening and closing of the three leaflets looked good on intraoperative transesophageal echocardiography (TEE). The only prosthetic valve regurgitation was evident at the central region where the leaflets form coaptation, and no abnormal findings were seen. Serum lactate dehydrogenase (LDH) was decreased to 405 U/l after surgery. However, LDH again began to increase on the 3rd day after surgery and it increased to 1,830 U/l on the 14th day after surgery. Hemolytic urine was detected on 10th day after surgery. PVL was not detected, but moderate abnormal regurgitation from the outside of the stent pocket was detected on TEE. Revision of valve replacement was considered, but LDH thereafter to 393 U/l on 41st day after surgery. The TEE was repeated, and only a trace of central jet was detected without abnormal regurgitation, unlike the previous examination. The patient did not develop any complications thereafter and was discharged on 47th day after surgery. LDH was nearly normal at the time of discharge. (Ann Thorac Cardiovasc Surg 2005; 11: 413–5)

Key words: valve replacement, hemolysis, bioprosthesis

Introduction

Since the durability of bioprosthesis has been improved and valve replacement has been increasingly utilised in elderly patients, valve replacement using bioprostheses has increased. Among bioprostheses, Carpentier-Edwards pericardial bioprosthesis (Edwards Lifesciences, Irvine, CA) has been markedly improved in its long-term results and valve-related complication including valve dysfunction, compared to the previous generation bioprosthesis. Here, we report a patient in whom transient prosthetic valve regurgitation and hemolysis occurred early after mitral valve replacement using a Carpentier-Edwards pericardial bioprosthesis and were resolved by preservative therapy.

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Received July 7, 2005; accepted for publication August 9, 2005.
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Case Report

The patient was a 77-year-old female receiving drug therapy for mitral valve stenosis and insufficiency from

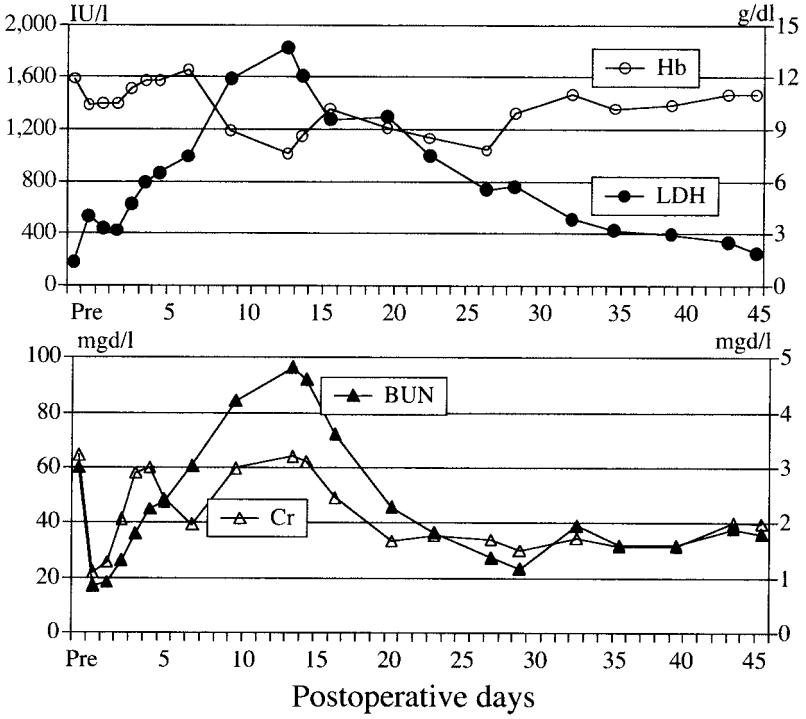

Fig. 1. Courses of LDH, Hb, BUN, and Cr after surgery.

the cardiology department of our hospital since 12 years ago. Cardiac failure progressed and the patient was referred to our department for surgery. Cardiac catheter examination and transthoracic (TTE) and transesophageal echocardiography (TEE) detected severe mitral regurgitation. The left ventricular ejection fraction was 70%. The maximum range of pressure at the mitral valve was 10 mmHg and the valve area was 2.0 cm^2. Seventy-five percent stenosis was detected at the mid position of the anterior descending branch (LAD) of the left coronary artery. On preoperative examination, Ccr was 20 ml/min, BUN was 60 mg/dl, and Cr was 3.23 mg/dl, showing renal dysfunction. Since the patient was elderly, mitral valve replacement with a bioprosthesis and coronary artery bypass at LAD #8 with the left internal thoracic artery were performed. Both anterior and posterior cusps of the mitral valve were resected and mitral valve replacement with a Carpentier-Edwards pericardial bioprosthesis 27 mm (Model 6900) was performed at the intra-annular position using everting mattress of pledjeded Ethibond. Insertion of the valve was easy, and opening and closing of the three leaflets were good on intraoperative TEE. The only prosthetic valve regurgitation was evident at the central region at which the leaflets coapt, and no abnormal findings were recognized. Hemodynamics and renal function were stable after surgery. Serum lactate dehydrogenase (LDH) was decreased to 405 U/l after surgery. However, LDH began to re-increase on the 3rd day after surgery. Since hemolytic urine was detected on the 10th day after surgery and BUN and Cr were 84.4 mg/dl and 3.0 mg/dl, respectively, continuous infusion supplemented with furosemide and administration of haptoglobin was given. TTE was performed on 11th day after surgery. Prosthetic valve regurgitation was transvalvular without PVL, and regurgitation was moderate. The movements of three leaflets were not abnormal. The urinary volume was maintained at 1,500 ml/day, but hemolytic urine persisted. LDH increased to 1,830 U/l on the 14th postoperative day (Fig. 1). On TEE performed on the 18th day after surgery, PVL was not detected, but moderate abnormal regurgitation from the outside of the stent pocket was detected (Fig. 2). Revision of valve replacement was considered, but LDH decreased thereafter to 393 U/l on the 41st day after surgery and renal function improved: BUN, 36.0 mg/dl, Cr, 1.75 mg/dl. TEE was repeated, and only a trace of central jet was detected without abnormal regurgitation, unlike the previous examinations. The patient did not develop complications thereafter and was discharged on the 47th day after surgery. LDH was nearly normal at the time of discharge.

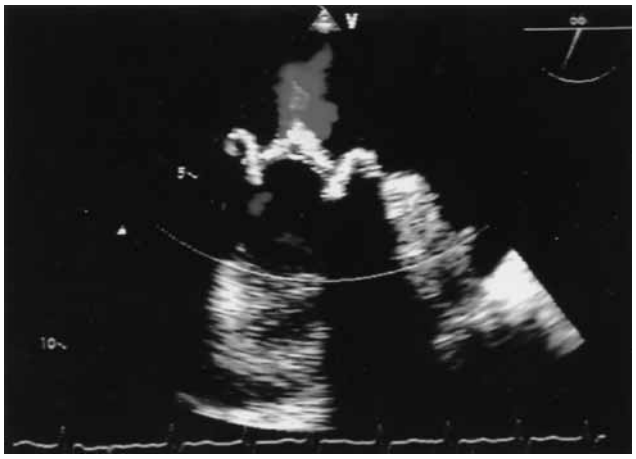

Fig. 2. Postoperative transesophageal echocardiography.

Comment

Hemolysis due to PVL and hemolysis long after surgery to correct valve dysfunction have been reported.[1,2] There have been no reports of hemolysis occurring early after surgery and resolving as in this patient. In this patient, hemolysis due to extracorporeal circulation resolved early and LDH decreased after reaching a peak on the day of surgery. However, LDH re-increased gradually after the 3rd day and reached a peak of 1,830 U/l, and hemolytic urine was observed. TTE did not detect significant abnormal regurgitation, but TEE detected abnormal regurgitation generated at the outside of the stent pocket. LDH then decreased after reaching a peak on 14th day after surgery, and returned to nearly normal at the time of discharge. On TEE at discharge, previously detected abnormal regurgitation had resolved, and only a streak of regurgitation was generated in the center at which three leaflets coapt, which is generally observed in this valve. There have been no previous reports of transient abnormal prosthetic valve regurgitation early after surgery of this type. When the operative procedure compresses and distorts the stent, abnormal regurgitation may occur early after surgery. However, echocardiography did not detect regurgitation during surgery and on the day of surgery. It is interesting that decreased LDH re-increased then decreased. Generally, when a Carpentier-Edwards valve is used, physiological prosthetic valve regurgitation occurs in the center where the three leaflets coapt in many cases. Firstenberg et al. investigated regurgitation in this valve by TTE in 57 patients who underwent mitral valve replacement.[3] Trace, mild, and moderate regurgitation was detected in 83%, 16%, and 1.7% of the patients, respectively, and the regurgitation jet location was central in 55 and indeterminate in three patients. The regurgitation volume affects left ventricular function, but there was no significant difference in the left ventricular function with time, indicating that changes in regurgitation volumes are unlikely to be due to changes in left ventricular function. The mechanism of regurgitation in this patient is unknown, but the findings of TEE suggested that the regurgitation was generated at the outside of the stent pocket. The oversizing, non-uniform arrangements of suturing threads and ligation cause stent change and loss of correct coaptation of the valve cusps. This phenomenon is called stent distortion. Regurgitation may occur as a consequence of this, but left ventricular systolic pressure is loaded onto the valve and may spontaneously correct the distortion of the stent, resolving regurgitation. However, regurgitation may occur from an outer region of the stent with structurally thin PTFE, rather than from stent distortion. Since regurgitation resolved within a short period after surgery, it is unlikely that newly formed intima covered the generation site and resolved regurgitation. The region may have been filled with blood components, resolving regurgitation. Although the site and cause regurgitation could not be definitely determined, when a case similar to this is encountered, it may be necessary to perform transfusion, maintenance of urinary volume using diuretic agents, and administration of haptoglobin and follow the course of LDH while considering revision surgery. Regarding postoperative monitoring, TEE should be performed as TTE did not adequately detect regurgitation. Since many patients having a bioprosthesis are elderly, repeated surgery is hazardous and accurate diagnostic measures are required.

References

1. Eric Jamieson WR, Marchand MA, Pelletier CL, et al. Structural valve deterioration in mitral replacement surgery: comparison of Carpentier-Edwards supra-annular porcine and perimount pericardial bioprostheses. *J Thorac Cardiovasc Surg* 1999; **118**: 297–304.
2. Marchand MA, Aupart MR, Norton R, et al. Fifteen-year experience with the mitral Carpentier-Edwards PERIMOUNT pericardial bioprosthesis. *Ann Thorac Surg* 2001; **71** (5 Suppl): S236–9.
3. Firstenberg MS, Morehead AJ, Thomas JD, et al. Short-term hemodynamic performance of the mitral Carpentier-Edwards PERIMOUNT pericardial valve. Carpentier-Edwards PERIMOUNT Investigators. *Ann Thorac Surg* 2001; **71** (5 Suppl): S285–8.