

A Surgical Case for Severe Hemolytic Anemia after Mitral Valve Repair

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We report a rare case of severe hemolytic anemia accompanied by moderate renal insufficiency after mitral valve repair. Although the degree of the residual mitral regurgitation was less than 1+ during the first three weeks after the operation, the maximum lactate dehydrogenase (LDH) was up to 7,430 U/l and the minimum hemoglobin was 4.9 g/dl. The mitral valve replacement successfully resolved the hemolysis, but the renal function did not completely recover. (Ann Thorac Cardiovasc Surg 2005; 11: 198–200)

Key words: mitral repair, hemolytic anemia, thrombotic microangiopathy (TMA)

Introduction

Hemolysis in prosthetic mitral valve regurgitation has been recognized, but the mechanism of hemolysis following mitral repair is not well characterized. We present a case of severe hemolytic anemia accompanied by moderate renal damage after mitral valve repair. The focus of this report was how to identify the mechanism of hemolytic anemia. And we discuss the diagnosis and characteristics of it.

Case Report

A 61 year-old man was admitted to the other hospital for heart failure due to severe mitral regurgitation (MR). Further examination revealed prolapse of the medial portion of the posterior leaflet (P3) and rupture of the chordae tendineae. Coronary angiography showed 75% and 90% stenosis of the left anterior descending artery (LAD) and the diagonal branch, respectively. He was referred to our hospital for the mitral valve repair and coronary artery

bypass grafting (CABG). He underwent mitral valve repair of quadrangular resection of the P3. An artificial chordae tendineae of CV-4 Gore-Tex was placed at the incidentally damaged chord of the P3. The mitral annuloplasty was also done using a 28-mm Cosgrove ring. Before the procedure, the left internal thoracic artery was anastomosed to LAD and the free right internal thoracic artery (RITA) to the diagonal branch as a composite Y-graft because the RITA was partially damaged. The cardiopulmonary bypass (CPB) time and aortic cross clamp (AXC) time were 210 minutes and 93 minutes respectively. There was no need for blood transfusions during the operation.

The postoperative course was unremarkable. He was extubated on the following day in the intensive care unit (ICU). There was no heart murmur and the lactate dehydrogenase (LDH) level was 432 when he left the ICU on the postoperative day (POD) 3. All the chest tubes were removed on the POD 4. The LDH level elevated from 560 on the POD 6 to 985 on the POD 10. On the POD 16, he presented with port-wine urine. The LDH went up to 1,488 (LDH-1 dominant pattern). The hemoglobin (Hb) and total bilirubin were 9.4 g/dl and 1.7 mg/dl. We suspected the hemolytic anemia due to incomplete repair of the mitral valve. The transthoracic echocardiography (TTE) revealed no more than 1+MR and the jet from the medial to the anterior aspect of the left atrial cavity was not strong enough to induce the hemolysis. In a few days,

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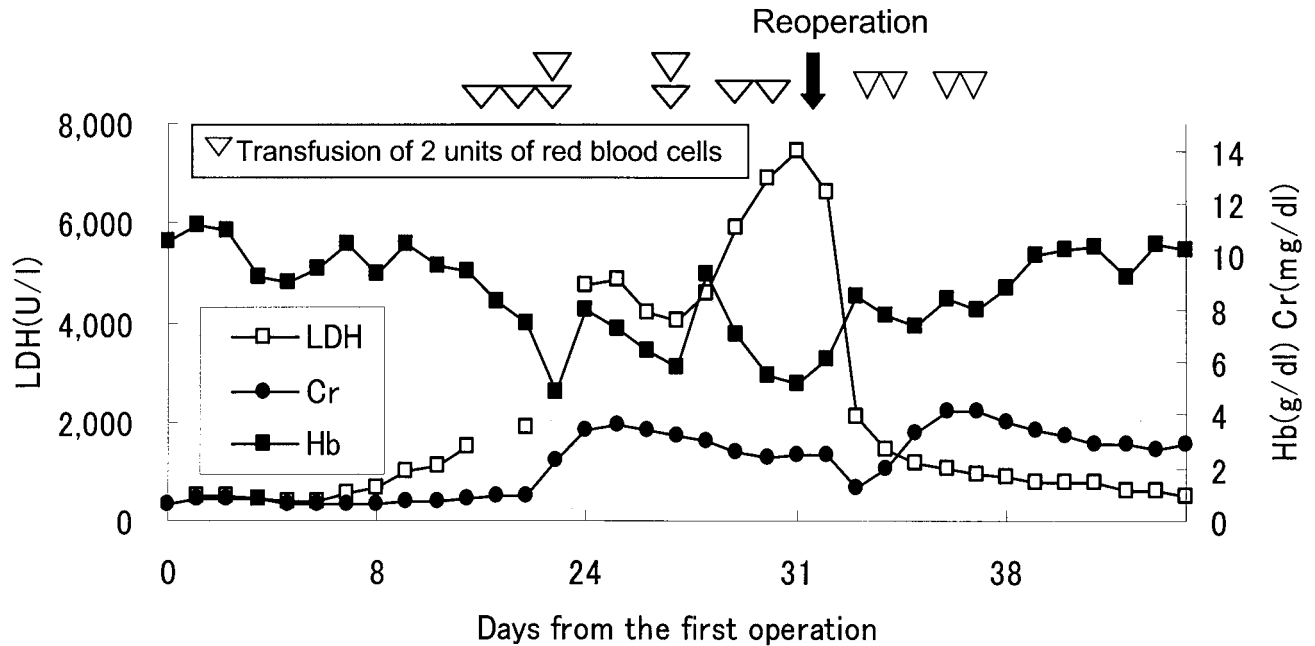


Fig. 1. Postoperative clinical course. LDH; lactate dehydrogenase, Cr; serum creatinine, Hb; hemoglobin. 10 units of red blood cells were transfused intraoperatively.

the Hb dropped to 4.9 g/dl, and the LDH and serum creatinine (Cr) elevated up to 4,855 U/l and 3.63 mg/dl despite enough hydration (Fig. 1). The count of thrombocyte was slightly depressed. We consulted our hematologists about this pathology. On the peripheral blood smear, there were not so many schistocytes and red cell fragmentations enough to suggest such a severe mechanical shearing of red blood cells. The Coombs' test and cold agglutination were negative. So they suspected thrombotic microangiopathy (TMA) including hemolytic uremic syndrome (HUS) rather than autoimmune hemolytic anemia (AIHA) or valve-related mechanical hemolysis. We rehydrated and transfused red blood cells for the anemia and used prednisolone for the possible microangiopathy. Though he never presented with heart failure, 3+MR was detected by TTE on the POD 27. The jet was directed from the repaired posterior leaflet to the anterior aspect of the left atrium. We decided to do reoperation for mitral valve replacement, despite some risks of renal insufficiency and the possibility of exacerbation of TMA if it existed. The LDH was 7,430 U/l just before the reoperation (Fig. 1).

On the POD 33, the reoperation was done. The intraoperative transesophageal echocardiography (TEE) showed 2+MR from the medial posterior leaflet. The regurgitant jet had collided with the annular support ring and was divided by it. By the inspection of the valve,

there was no ring dehiscence, suture-related tear of the valve tissue, or chord break. We conducted the mitral valve replacement with a 27-mm SJM. Hemodialysis was performed during the surgery. The CPB and AXC time were 134 minutes and 84 minutes respectively. The clinical course after the valve replacement was very good. The hemolytic anemia resolved immediately after the operation. The LDH level gradually fell to 502 U/l in the two weeks after the operation. Though the renal function did not improve completely, he did not need postoperative hemodialysis. The postoperative TTE showed good left ventricular and valve function. He left the hospital to his local institution for further recuperation on the 24th-day after the second operation.

Discussion

In our case, very severe hemolytic anemia did occur about two weeks after the mitral valve repair and moderate renal insufficiency followed. Considering the 1+MR, unaccountable damage of the renal glomeruli, and slight thrombocytopenia, we suspected microangiopathic pathology at the time. The reoperation was done mainly because the MR increased to 3+ on TTE. The hemolytic anemia subsided after that. The preexisting fragility of red blood cells by TMA may have been related to the hemolysis. But now we believe that the fragmentation or

acceleration of the regurgitant jet was the major cause of this rare case severe hemolysis.

The occurrence of hemolysis in prosthetic mitral valve regurgitation has been well recognized. Ionescu et al. investigated prevalence and clinical significance of incidental paraprosthetic valvar regurgitation regarding the LDH level.¹⁾ Garcia et al. reported the mechanism of it using TEE. They suggested five regurgitant patterns — fragmentation, collision, rapid acceleration, free jet, and slow deceleration.²⁾ There are a few reports about severe hemolytic anemia following mitral repair.³⁻⁶⁾ Lam et al.⁷⁾ characterized the hemolysis after mitral repair in 32 patients as did Gracia and associates.²⁾ In Lam's study, most patients had high grade MR and jets were fragmented or accelerated, but 23% of them had only 1 or 2+MR. They insisted the severity of the hemolysis does not depend on the echocardiographic variables, including mild degrees of MR.⁷⁾

We presented a case of severe hemolytic anemia accompanied by moderate renal damage after mitral valve repair with only grade I MR. When we see a patient with persistently low or falling hematocrit after mitral repair, we should consider the mechanical hemolysis even if the MR is not so severe and do more examinations such as transesophageal echocardiography. Though the clinical presentation dramatically improved after the valve replacement, we should do further follow up of the remaining renal insufficiency.

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