emolytic anemia is uncommon after mitral valve repair and only a few cases have been reported. Although it is thought that rapid acceleration, fragmentation and collision of mitral regurgitation jets may be associated with high shear stress and cause hemolysis,1,2 the precise mechanisms are not understood. In addition, the course of severe hemolysis after mitral valve repair has not been established, although in most cases the hemolysis improves with reoperation.2–11 We report a patient who developed a severe hemolytic anemia after mitral valve repair that was reduced without the need for reoperation.

**Case Report**

A 60-year-old man suffered from shortness of breath because of severe mitral regurgitation secondary to the prolapse of the posterior mitral leaflet. Transesophageal echocardiography revealed severe mitral regurgitation filling the whole left atrium from the medial scallop of the posterior or mitral leaflet. The dimension of the left atrium in systole phase and the left ventricle in diastole was 53 mm and 58 mm, respectively. Surgery was performed under cardiopulmonary bypass, in mild hypothermia. Myocardial protection was afforded with cold crystalloid cardioplegia. A left paraseptal atriotomy was performed. The prolapsing medial scallop of the posterior mitral leaflet was resected, and the edge of leaflet was sewn onto a mitral annulus. A Duran flexible ring was sutured and fixed along the mitral annulus. A water injection test confirmed no regurgitation. Transthoracic echocardiography 10 days after the mitral valve repair showed mild mitral regurgitation, reaching the deep left atrium from the posterior mitral commissure. The dimension of the left atrium in systole and the left ventricle in diastole was 34 mm and 45 mm, respectively. Three weeks after the mitral valve repair, the patient was discharged with no complaints. After 2 months, he was re-admitted complaining of fatigue and shortness of breath. His palpebral conjunctivae were anemic and bulbar conjunctivae icteric, but splenomegaly was not apparent. Laboratory data were as follows: red blood cell (RBC): 206×10^4/μl; hemoglobin: 6.2 g/dl; haptoglobin: <10 mg/dl; reticulocytes: 15.6×10^4/μl; lactate dehydrogenase (LDH): 3,235 IU/L; total bilirubin: 1.6 mg/dl. All these values had been within the normal range 5 months earlier. The Donath-Landsteiner test, Coombs test, Ham test and sugar water test were all negative. Drug-induced hemolytic anemia was not apparent, because there was no reduction of hemolysis after stopping the administration of any drug without furosemide, which was administered before the mitral valve repair.

**Key Words:** Hemolysis; Mitral valve repair; Reoperation

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**Fig 1.** Peripheral blood smear showing many fragmented red blood cells.
Discussion

Increased intravascular hemolysis occurs in the majority of patients with mechanical prosthetic valvular replacement and the incidence of hemolytic anemia varies from 5% to 15%. In contrast, RBC trauma and hemolysis following mitral valve repair and annular ring placement is uncommon because there are no moving mechanical parts or prosthetic leaflets. Only 7 patients among 1,548 who had underwent mitral valve repair needed reoperation for hemolytic anemia. The mechanisms of hemolysis following mitral valve repair have been reported to include dehisced annuloplasty rings producing para-ring regurgitant jets; protruding perivalvular suture material that provided a site of impact for circulating RBCs; ‘whiplash motion’ of residual, free-floating chordae tendineae within a hyperkinetic left ventricular chamber; nonendothelialization of foreign materials such as sutures or rings; and a small but turbulent regurgitation jet against the left atrial wall. Rapid acceleration, fragmentation and collision jets are associated with high shear stress, and may cause mitral prosthetic hemolysis, whereas free and slow deceleration jets are not! The mechanism of hemolysis observed after mitral valve repair most commonly involves direct collision of the regurgitation jet with an annuloplasty ring and appears to be independent of the severity of mitral regurgitation.

In the present case, a high velocity, paravalvular mitral regurgitation jet collided with the Duran ring and may have caused the hemolysis. Hemolytic anemia caused by prosthetic valves can be managed effectively with oral iron therapy in almost all patients, and reoperation, primarily for refractory hemolytic anemia, is seldom necessary and has not been consistently successful. On the other hand, an improvement in the hemolytic anemia following reoperation has been reported in many cases. Because of the severe hemolysis requiring a blood transfusion, we recommended that our patient undergo reoperation, but unexpectedly, the hemolysis reduced without reoperation, although a moderate mitral regurgitation jet remained. Slowed velocity caused by pranopinol may reduce the shearing stress between RBCs and foreign material but in this case, the hemolysis was reduced without an apparent change in velocity or blood pressure. Endothelialization of the Duran ring may have reduced the damage to the RBCs; prosthetic materials usually become endothelialized rapidly, within several weeks, but recurrent or residual mitral regurgitation jets may prevent the endothelialization and cause hemolysis at this site. In addition, it has been reported that incomplete endothelialization of the prosthetic material employed in the initial mitral repair was present in 11 of 12 patients reoperated for hemolysis, that the interval between initial repair and reoperation was 2.9 months, and that the site of incomplete endothelialization was not visible on transeosophageal echocardiographic examination in any of these patients! Although it is difficult to know without inspection whether endothelialization really has occurred in the present patient, the important outcome is that hemolysis was reduced over the span of several months and that reoperation was not necessary.

It is important that the appropriate time for reoperation is not lost during refractory hemolytic anemia, but we had a rare case whose severe hemolysis after mitral valve repair was reduced without reoperation, over the span of several months.
References


