

Case Report

Hemolytic anemia caused by mild regurgitation after mitral valve plasty

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A 60-year-old woman underwent mitral valve plasty and annuloplasty with a prosthetic ring. She developed myocardial infarction of the left ventricular inferior wall with stable hemodynamic condition on the postoperative day of 2nd. On the next day, severe hemolysis became apparent. Echocardiography revealed mild mitral valve regurgitation with akinesis of the inferior wall of the left ventricle. The regurgitant flow hit the implanted prosthetic ring, and changed the flow direction. She underwent mitral valve replacement, and her subsequent course was uneventful with quick disappearance of hemolytic anemia.

(key words : mitral valve plasty, hemolytic anemia, myocardial infarction, mitral valve replacement)

Introduction

Hemolysis is a troublesome complication after valve surgery. Not only valve replacement but valve repair can cause apparent hemolysis. In case of valve replacement, transvalvular or paravalvular blood flow may contribute hemolysis. After valve plasty, however, residual regurgitation per se is related to hemolysis. Some anecdotal experiences with mitral valve plasty have been reported. Our case also shows that a mild residual regurgitation produced the severe hemolysis after mitral valve plasty, requiring reoperation.

Case

A 60-year-old house wife was admitted to our hospital because of surgical treatment of mitral valve regurgitation. Echocardiographic study and left ventriculography showed massive mitral valve regurgitation associated with prolapse of the posterior leaflet of the mitral valve. Cardiac catheterization showed the intact coronary arteries without abnormality of the left ventricular wall motion. Cardiac index was 5.4L/min/m². She underwent mitral valve plasty of quadrangular resection of the prolapsed portion and annuloplasty with a 26-mm Cosgrove-Edwards prosthetic ring (Edwards Lifesciences, Irvine, CA, USA). Moderate hypothermia and antegrade infusion of blood cardioplegic solution was used for myocardial protection. Intraoperative esophageal echocardiographic examination revealed trivial residual mitral

regurgitation without abnormality of the left ventricular wall motion, and weaning from cardiopulmonary bypass was uneventful.

She developed myocardial infarction of the left ventricular inferior wall with the highest creatinine phosphokinase (CPK) of 4114mU/ml on the postoperative day of 2nd. Her hemodynamic condition was not deteriorated, although postoperative electrocardiogram showed abnormal Q-wave in leads of II, III, and aVf. On the postoperative day of 3rd, severe hemolysis became apparent associated with brown to black urine color, and serum haptoglobin showed a low level below 10mg/dl. Hemoglobin (Hb) levels remained between 7.1 and 9.2g/dl without blood transfusion, and lactate dehydrogenase (LDH) rose to 5847mU/ml. A lot of kinds of peripheral red cell fragments were observed (Fig.1), and reticulocytes also increased to 5.1% in the peripheral blood. Total and direct bilirubin levels showed 2.78mg/dl and 0.4mg/dl. Blood urea nitrogen (BUN) and creatinine increased to 28mg/dl and 1.50mg/dl, respectively. Echocardiography revealed mild mitral valve regurgitation with akinesis of the inferior wall of the left ventricle. The regurgitant flow ran toward the posterior wall of the left atrium, hit the implanted prosthetic ring, and changed the flow direction (Fig.2).

On the postoperative day of 9th, reoperation was performed. Operative findings showed no apparent abnormalities of the repaired posterior leaflet of the mitral valve and the implanted ring. The anterior leaflet of the mitral valve opposed to the repaired portion of the posterior leaflet was slightly prolapsed. The prosthetic ring was removed and the anterior leaflet of the mitral valve was resected. The valve was replaced with a Bicarbon valve of 27mm. Her subsequent course was uneventful with quick normalization of Hb, LDH, BUN, and others.

Discussion

Mitral valve plasty is performed worldwide and is quite effective. The plasty has been also preferred to valve replacement from the point of patients' postoperative quality of life and hemodynamic superiority. However, there are risks for recurrent valve dysfunction and reoperation since it requires surgical skills based on surgeon's experiences.^{1,2}

Mitral valve plasty includes chordal shortening, transposition of chorda tendinea, resection of prolapsed leaflets, and annular remodeling using a prosthetic valve ring. Not only prosthetic valve leaflets per se but sewing rings may produce a higher share stress on the red cell

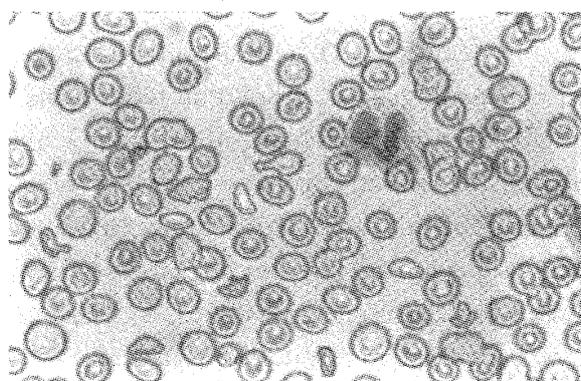


Fig. 1 Hemogram (Wright-Giemsa stain, 400x) when hemolytic anemia became apparent, Different kinds of fragmented red cells were observed in the peripheral blood.

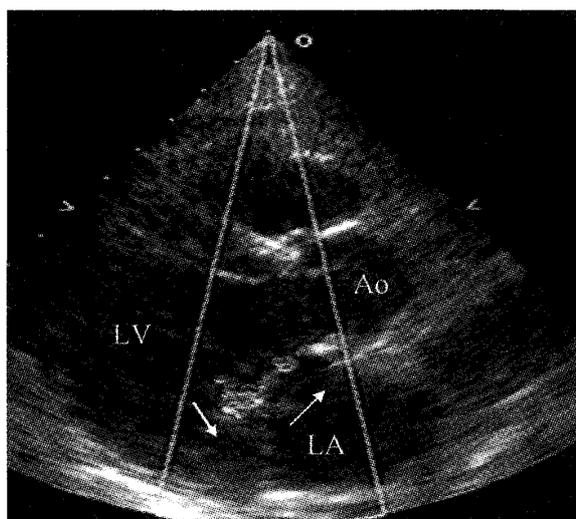


Fig. 2 Echocardiographic findings 7 days after the initial mitral valve plasty. Mitral valve regurgitation was mild. The regurgitant flow through the mitral valve ran toward the posterior wall of the left atrium (left arrow), hit the implanted prosthetic ring, and changed the flow direction (right arrow).

Abbreviation

Ao : Ascending aorta

LV : Left ventricle

LA : Left atrium

membrane than native valves. Some investigators reported cases of hemolysis after mitral valve plasty and hypothesized its mechanisms.²⁻⁹ The mechanisms of red cell destruction includes a high-velocity regurgitant jet and its collision at the cloth-covered annuloplasty ring. Red cells exposed by high shear stresses change their appearance showing red cell fragments. Fig.1 endorses the hypothesis, showing a small amount of regurgitant blood flow toward the ring and its reflection at the ring. Therefore, every valve replacement causes hemolysis to some extent, and valve plasty can also lead to hemolytic anemia, when residual regurgitation blood flow hit the prosthetic ring as our case. The hemolysis can be overcome without any clinical symptoms under normal hemopoietic function in most patients. However, some patients develop hemolytic anemia, when the hemolysis is severe or the hemopoietic function of patients is impaired. We administered haptoglobin since hemolysis became apparent, which led transient urine color normalization, but renal dysfunction gradually progressed without recovery of the hemolysis.

Our patient developed hemolysis after the latent period of 3 days. A transient decrease of LDH immediately after the initial operation was followed by a rapid increase of LDH, together with hemolytic anemia and colored urine. The myocardial infarction can explain the recurrence of the mitral valve dysfunction caused by papillary muscle dysfunction. In our case, myocardial infarction of the left ventricular inferior wall might be caused by embolism of air or thrombus because the preoperative study showed the coronary arteries were intact.

The indication of reoperation for hemolytic anemia after valve surgery should be carefully decided before multiple organ failure progresses. Before renal shutdown or liver dysfunction, which can lead lethal clinical results, mitral valve re-plasty or replacement can always relieve

the hemolysis, effectively and immediately.^{8,10,11}

Conclusion

Hemolytic anemia occurred after a few day latent period after mitral valve plasty, and echocardiography showed that the mild regurgitant flow ran toward the posterior wall of the left atrium, hit the implanted prosthetic ring, and changed the flow direction. Postoperative myocardial infarction was related with recurrence of mitral valve dysfunction. Subsequent mitral valve replacement quickly led to hematological normalization.

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僧帽弁形成術後に生じた溶血性貧血のため、弁置換術を 施行した1例

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要 約

患者は60歳の女性で変性による僧帽弁閉鎖不全のため、後尖の部分切除術と人工弁輪を用いた弁輪形成術を受けた。患者は第3病日に溶血が顕著となり、心エコー図上、軽度の僧帽弁閉鎖不全と左心室下壁の著明な低下が確認された。エコー図上では逆流した血流が左房後壁側の人工弁輪に向かい、そこで方向を変えて上行

大動脈方向に向かっていた。周術期心筋梗塞に合併した僧帽弁閉鎖不全の再発に伴う溶血性貧血と診断し、第9病日に再手術した。再手術後に溶血や逆流は認められず軽快退院した。

(キーワード：僧帽弁形成術，人工弁輪，溶血性貧血，僧帽弁置換術)