



Traumatic Hemolytic Anemia after Valve Surgery: a Case Report

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Abstract

Hemolytic anemia is an uncommon complication after mitral valve repair. We present a case of a 55-year-old man who presented with post-operative hemolytic anemia after mitral valve repair with prosthetic ring. The hemolytic anemia improved after the patient had the prosthetic ring removed and the valve replaced by a prosthetic mitral valve. However, the post-operative course of the redo operation was complicated by acute renal failure and respiratory dysfunction, but the hemolytic anemia was finally abolished and the patient was discharged 20 days post-operatively in good condition.

Keywords

complication, hemolytic anemia, mitral valve repair

INTRODUCTION

Hemolytic anemia (HA) is a rare but important complication of any repair or construction of the heart. HA should always be considered in patients with a history of mitral valve repair (MVR).¹ MVR is the preferred method in dealing with mitral insufficiency. Myxomatous degeneration evolves with a variety of clinical syndromes of which mitral insufficiency is the most common. The most frequent mechanism of regurgitation in this condition is the chordae tendineae elongation or rupture, and dilatation of the posterior ring of the mitral valve.² Currently however, the indication for MVR is well-known, but there is no precise indication for surgical or medical intervention in case of hemolytic anemia following MVR surgery.³ Double-Teflon posterior mitral leaflet repair is more susceptible to HA in comparison with a complete ring repair of the mitral valve. Regurgitated blood through irregularly shaped openings of the repaired mitral valve is associated with more hemolysis than through large round orifices. In addition, abnormal

flow in the presence of a serrated rim of the leaflet especially in myxomatous change of the mitral valve may result in HA. Severe hemolysis and intractable congestive heart failure require closure of leak or valve replacement.¹ We report a patient who developed postoperative hemolytic anemia after mitral valve repair with prosthetic ring.

CASE REPORT

A 55-year-old male with a history of dyspnoea (6 months) and chronic atrial fibrillation was referred to our hospital for further evaluation. His echocardiographic findings were severe mitral regurgitation and pulmonary hypertension (70 mmHg). He had severe limitation of daily physical activity. He did not have dyspnea at rest, but was breathless on exertion. Physical examination showed a grade 2/6 pansystolic murmur which was audible in the mitral valve zone. Chest exam was normal. Abdominal palpation revealed hepatomegaly with no ascites. Lower extremities were oedematous

with pitting edema. Body temperature was 37.4°C, pulse rate 110 beats/minute, respiratory rate - 19 breaths/minute, and his blood pressure was 135/70 mm Hg. An electrocardiogram was unremarkable. Laboratory studies showed his hemoglobin 13 g/dl, fasting blood glucose 85 mg/dl, blood urea nitrogen 33 mg/dl, and creatinine 1 mg/dl. On transthoracic echocardiography, severe mitral regurgitation was detected in the large left atrium, but stoppage to the diastolic transmitral backflow was not found (**Fig. 1**). The left ventricular myocar-



Figure 1. Echocardiography in a four-chamber view shows mitral regurgitation.

dium was hypertrophic with a maximum diameter; however, no wall motion abnormalities were detected. The left atrium was enlarged with a diameter of 60 mm but other cardiac chambers and aortic valve function were normal. The patient had normal native coronary artery disease. There was also evidence of severe mitral regurgitation with myxomatous change of the mitral valve. Ejection fraction was 30%. The patient was scheduled for the mitral valve repair. At surgery, the patient was found to have a myxomatous mitral valve with ring dilatation. The patient underwent mitral valvuloplasty with quadrangular resection of medial P2 and lateral P3. Annuloplasty was performed with pledged sutures, and the annulus was kept at the appropriate size by tying the stitch and prosthetic annuloplasty rings (memo CarboMedix, Sorin Group, number 30) together. The postoperative course of the patient was complicated by yellow sclera, renal failure, congestive heart failure (CHF) and fluid overload. They were successfully treated with fluid management, inotropic drug and diuretics usage. The patient also had low-grade fever and leukocytosis. Blood, urine, sputum, and stool cultures were negative for bacterial growth. An echocardiogram revealed a well-donned repair with near normal Doppler velocities and trivial evidence of regurgitation. The patient was empirically treated with broad-spectrum antibiotics. At this time, he was treated with diuretics and afterload reduction. A repeated echocardiogram revealed severe left ventricular systolic dys-

function with evidence of a mild mitral regurgitation. Three days after surgery, the patient's urine color changed to a dark scheme and blood urea nitrogen and creatinine levels were raised. Physical examination revealed jaundice, hepatic enlargement, and bilateral lower extremity edema. Laboratory values showed anemia (Hb 8 g/L), hepatic function test disturbances showed an elevated lactate dehydrogenase level of 3000 IU/L, low haptoglobin, hemoglobinuria, and the presence of fragmented cells in blood smear. A thoracic X-ray revealed cardiomegaly with pulmonary edema. MVR was complicated by hemolysis and severe congestive heart failure that was resistant to medical therapy. The clinical picture of HA with right heart failure progressed to multiple organ failure. Investigation of other etiology that might explain the patient's condition and HA was performed by a hematologist. The possibility of autoimmune HA and other etiologies that might explain the patient's condition was excluded. These diseases included hemoglobin SC disease (similar in symptoms to sickle-cell anemia), HA due to glucose-6-phosphate dehydrogenase deficiency, hereditary elliptocytosis, ovalocytosis, spherocytosis, idiopathic autoimmune HA, malaria, microangiopathic HA, non-immune HA caused by chemicals or toxins, paroxysmal nocturnal hemoglobinuria, secondary immune HA, sickle-cell anemia, thalassemia and transfusion of blood from a donor with a different blood type. The patient received multiple blood transfusion and despite the use of a high dose of inotropic drugs and intra-aortic balloon pump, his hemolysis continued and CHF did not improve. At day 6 of primary operation with improvement of cardiac function and tapering of the inotropic drug dose, multiple attempts to wean the patient from the ventilator was unsuccessful. His serum creatinine concentration increased to 2.2 mg/dL and he became oliguric. The patient underwent cardiopulmonary bypass. The prosthetic ring was removed and with preservation of the posterior mitral valve's papillary muscle, the mitral valve replaced with a prosthetic mitral valve (29 CarboMedic, Sorin Group). Intraoperative finding revealed grossly normal and intact mitral valve repair (**Fig. 2**). Despite abolishing the hemolysis with mitral valve replacement, it had postoperative complications such as renal and respiratory failures, tracheostomy, and prolonged intensive care unit stay. The patient was discharged on day 20 postoperatively in a good condition.

DISCUSSION

Hemolytic anemia is a rare complication after mitral valve repair and has been reported in few studies. Mitral regurgitation is usually caused by a prosthesis failure. Despite the preference of mitral repair to replacement, repair results in some complications such as stroke, reoperation, thromboembolism, endocarditis, residual regurgitation, mitral stenosis, hemolysis, and reduced posterior annular contraction in the rigid annuloplasty ring.²

In the present study, hemolytic anemia occurs after mitral valve reduction annuloplasty technique with prosthe-

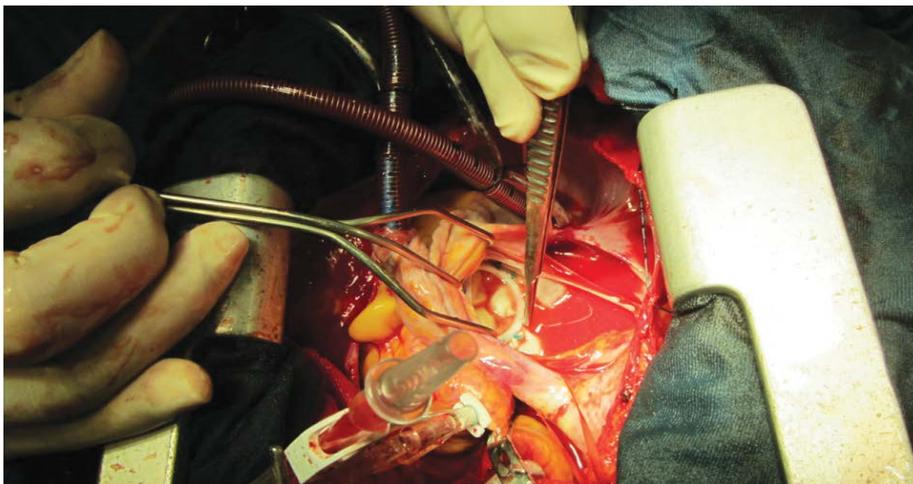


Figure 2. Apparently intact mitral valve repair with a prosthetic ring.

tic ring in patients with mitral insufficiency secondary to myxomatous degeneration. In patients with myxomatous degeneration with ruptured or elongated chordae tendinae, surgical repair of the valve may be associated with some degree of residual regurgitation. With the annuloplasty technique, the posterior leaflet of the mitral valve becomes a “frame” against which the anterior leaflet opens and closes. Regurgitation of blood with the closing and opening of the anterior leaflet against fixed posterior mitral leaflet caused hemolytic anemia. HA can also occur due to collision of blood flow against the corda or papillary muscle and crest of the leaflet. In this case, we hypothesize that free jet mechanisms against the serrated edge of the valve may be attributed to HA. Other cause of HA such as dehiscence of rigid annuloplasty ring, collision of blood to valvular apparatus or disrupted suture of the quadric angular repair of leaflets were not seen in this patient. Demirsoy et al. reported that the most important mechanism of HA was shear stress of regurgitated jet across the repaired valve.⁴ The result of echocardiographic study showed that hemolysis after MVR had hydrodynamic patterns which cause high shear stress.⁵

We supposed that HA developed by red cell fragmentation caused by regurgitated blood collision across of repaired mitral valve apparatus. The regurgitated blood not only collides on valve apparatus but also pressure effect of jet flow, delayed endothelialization of the ring and suture line that itself perpetuates hemolysis.⁶ The mechanical collision of erythrocytes to the edge of the valve, release hemoglobin into plasma, then hemoglobin is absorbed by haptoglobin, thereby forming a toxic complex. This large complex was not filtered by the glomerulus, but scavenged by the reticuloendothelial cells of the liver, spleen, and bone marrow. With severe HA, plasma haptoglobin is saturated with hemoglobin complexes, then free plasma hemoglobin degraded from the tetrameric globin structure to dimeric hemoglobin.

CONCLUSIONS

Mitral valve repair with annuloplasty technique and a prosthetic ring employed in the patient with mitral insufficiency secondary to myxomatous degeneration presented with hemolysis and a serious clinical outcome at the early postoperative period. Despite redo operation with revising the repaired mitral valve and replacing it with the prosthetic valve, postoperative complication with multiple organ failure was reported.

Conflicts of interest

The authors have none to declare.

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Травматическая гемолитическая анемия после операции на клапане: клинический случай

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Резюме

Гемолитическая анемия – редкое осложнение после операции на митральном клапане. Это случай 55-летнего мужчины, который обратился с послеоперационной гемолитической анемией после операции на митральном клапане с использованием протезного кольца. Гемолитическая анемия улучшилась после удаления протезного кольца и замены митрального клапана протезом. Однако послеоперационный курс повторной операции осложнился острой почечной недостаточностью и нарушением функции дыхания, но гемолитическая анемия была преодолена, и пациент был выписан через 20 дней после операции в хорошем состоянии.

Ключевые слова

осложнение, гемолитическая анемия, восстановление митрального клапана
