Case Report

Do All Hemolytic Anemias That Occur After Mitral Valve Repair Require Surgical Treatment?

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ABSTRACT

We report on a 29-year-old woman with severe hemolytic anemia following mitral valve annuloplasty. Although hemolysis due to mechanical prosthetic mitral valve is well recognized, hemolytic anemia associated with mitral valve repair is an uncommon condition. Reoperation may be considered if the patient has serious and persistent anemia. Although valve replacement is suggested to be a unique intervention, it may not be the solution every time because of mechanical effects. Various mechanisms of hemolysis related to mitral valve repair were suggested, but sufficient and precise data is not available. In this case, we tried to emphasize whether all hemolytic anemias that occur after mitral valve repair require surgical treatment.

Case Report

A 29-year-old woman was referred to emergency services because of severe dyspnea developing into pulmonary edema. Medical therapy was given immediately and her clinical condition improved thereafter. She was consulted by the cardiology department and a diagnosis of severe mitral regurgitation associated with rheumatic heart disease was made. The patient was considered eligible for mitral valve repair. Mitral valve annuloplasty was performed at a private center. Intraoperative transesophageal echocardiography revealed minimal mitral regurgitation. Postoperative transthoracic echocardiography indicated +1 mitral regurgitation. Hematological findings were normal before surgery. She had no anemia and lactate dehidrogenase (LDH) level was in normal range. Following the surgery there was progressive fatigue, palpitation, and jaundice developed over a 2 mo period. Physical examination showed 2/6 pansystolic ejection murmur, pallor, icterus, and tachycardia. Other findings were normal.

Laboratory results revealed a Coomb's-negative hemolytic anemia with hemoglobin of 5.9 gr/dl (13.6–17.2 gr/dl), aspartate aminotransferase (AST) of 75 IU/L (0–40 IU/L), total bilirubin of 7.4 mg/dl (0.1–1mg/dl), conjugated bilirubin of 2 mg/dl (0–0.3 mg/dl), LDH of 1552 IU/L (207–414 IU/L), haptoglobin of 0.2 gr/L (0.3–2gr/L), ferritin of 4.9 ng/ml (5–148 ng/ml; Table 1). Levels of vitamin B12, folate, and hemoglobin electrophoresis were normal. Urine hemosiderin was found positive. Drug-induced hemolytic anemia was not considered because there was no recovery after cessation of any drug except furocemide which was given before surgery. The hemolysis workup of the patient was performed. Signs of intravascular hemolysis were evident. A history of infection, accident, and dark urine were not present, excluding numerous causes of intravascular red cell breakdown. Schistocytes were demonstrated in the peripheral blood smear of the patient (Figure 1). Fecal occult blood test was found to be negative 3 times. An abdomen ultrasonography was normal except for hepatic venous dilatation. Splenic size was normal. Since the patient had mitral valve repair with a unique history and clinical findings, the diagnosis of valvular hemolysis was reached. In transthoracic echocardiography, the dimension of the left atrium, end-diastolic, and end-systolic diameters of the left ventricle were 8.1 cm, 6.1 cm, and 4.2 cm respectively. The ejection fraction was 63%. The degree of mitral regurgitation was +3 (Figure 2). The patient refused transesophageal echocardiography, so that test was not performed. We suggested reoperation but our recommendation was not accepted by the patient. Iron therapy[1] and 4 units of blood transfusion were administered to compensate for anemia-induced tachycardia and dyspnea. Fortunately the hemolysis was not apparent after 3 mo. During follow-up, blood tests revealed hemoglobin

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Table 1. Laboratory findings comparing before and after operation

Laboratory Results	Before operation	After operation
Hemoglobin (13.6–17.2 gr/dl)	13.8	5.9
White blood cell (4.4–11.3 $\times 10^3/\mu L$)	5.1 $\times 10^3$	6.3 $\times 10^3$
Platelet (156–373 $\times 10^3/\mu L$)	158 $\times 10^3$	167 $\times 10^3$
Erythrocyte mean corpuscular volume (80–95.5 fL)	83.3	79
Red cell distribution width (11.5%–14.5%)	unknown*	27.8
Reticulocyte % (0.5%–3.5%)	2.7	4.51
Coomb's test	unknown*	negative
Haptoglobin (0.3–2 gr/L)	unknown*	0.2
Ferritin (5–148 ng/ml)	unknown*	4.9
Aspartate aminotransferase (0–40 IU/L)	36	75
Alanine aminotransferase (0–41 IU/L)	23	25
Lactate dehidrogenase (207–414 IU/L)	408	1552
Serum total bilirubin (0.1–1 mg/dl)	0.85	7.4
Conjugated bilirubin (0–0.3 mg/dl)	0.2	2
Urine hemosiderin	unknown*	3+

*These parameters were not evaluated before operation since the patient had a normal level of hemoglobin.

of 12.1 ngr/dl, LDH of 632 IU/L, and transthoracic echocardiography showed +3 mitral regurgitation.

Discussion

Hemolysis due to mechanical prosthetic valvular replacement occurs in the majority of patients and the incidence of hemolytic anemia varies from 5% to 15%.[1] Hemolytic anemia caused by prosthetic valves can be managed effectively with iron therapy in almost all patients and reoperation, primarily for refractory hemolytic anemia, is seldom necessary and has not been consistently successful.[1] However, hemolysis following annular ring placement and mitral valve repair is uncommon. Only 7 patients among 1,548 who underwent mitral valve repair required reoperation for hemolytic anemia.[2] Garcia et al. reported mechanisms of hemolysis at 5 categories; fragmentation, collision, rapid acceleration, free jet, and deceleration.[3] In our patient, we considered that free jet of regurgitation may underlie this hemolytic condition. 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.[4]

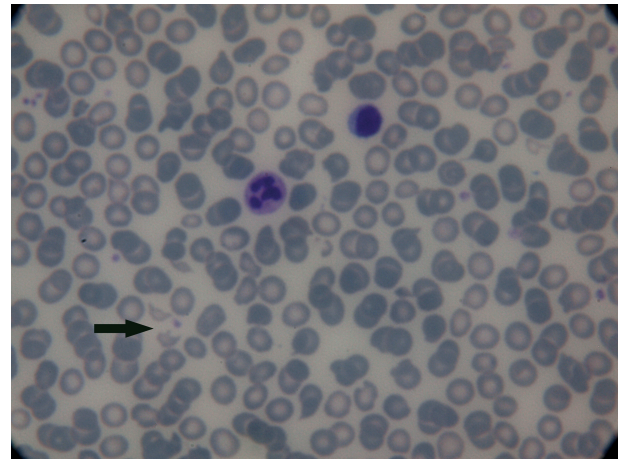

Figure 1. Peripheral blood smear showing schistocytes, as indicated by the arrow.

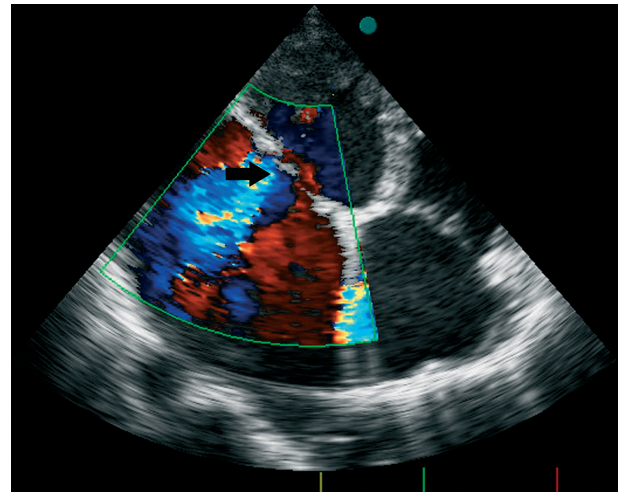

Figure 2. Transthoracic echocardiogram showing a free jet of mitral regurgitation in the apical four-chamber view.

Although our patient refused reoperation, hemolysis was not apparent and prosthetic materials may be endothelialized rapidly within several months. It has been reported that incomplete endothelialization of prosthetic material employed in the initial mitral repair was present in 11 of 12 patients reoperated on for hemolysis and that the site of incomplete endothelialization was not visible on transesophageal echocardiographic examination in any of these patients.[4] Inoue et al. reported a patient with severe hemolysis and gradually decreased hemolysis without reoperation following mitral valve repair, as in our case.[5] The question is whether or not all hemolytic anemias, as a consequence of mitral valve repair, require reoperation to prevent hemolysis. In such cases, the physician may observe the patient for several months to evaluate endothelialization of the prosthetic

material. Nevertheless, further investigations should be performed to manage that condition.

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