Successful Mitral Valve Replacement in Patient with Severe Hemolytic Anemia Due to Tight Mitral Stenosis

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Abstract

A 43 year-old woman complained of palpitation and shortness of breath admitted to our hospital. Transthoracic echocardiography revealed severe mitral stenosis and moderate mitral regurgitation. In routine blood tests hemoglobin and lactate dehydrogenase levels were 7.6 g/dL, 520 IU/L respectively. Coombs test was negative. After studying the other intrinsic or extrinsic causes of hemolytic anemia patient's anemia was diagnosed as intravascular hemolytic anemia due to tight mitral stenosis. Therefore, mitral valve replacement planned and performed. Hemolysis was resolved after the mitral valve replacement. In conclusion, we report a case of hemolytic anemia due to mitral stenosis resolved by mechanical mitral valve replacement.

Keywords: Hemolytic anemia, acquired, mitral valve stenosis, implantation, heart valve prosthesis

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Introduction

Hemolytic anemia following valve replacement or repair is well recognized [1]. However, intravascular hemolysis with native valve is seldom. Although, there are a few reports of intravascular hemolysis with aortic stenosis, there is no report with mitral stenosis [2,3]. We report a case of hemolytic anemia due to mitral stenosis, resolved by mitral valve replacement.

Case Report

A 43 year-old woman was admitted to our clinic with symptoms of palpitation and shortness of breath. Her blood pressure was 125/75 mm Hg and heart rate 78 beats per minute. There was a 4/6 sistolic murmur at mitral area. Transthoracic echocardiography showed a mitral stenosis with maximal pressure gradient of 37 mm Hg and moderate mitral regurgitation. Mitral valve area was 0.8 cm^2 and pulmonary artery pressure was 100 mm Hg. Left ventricular wall motion was normal with an ejection fraction of 60%. The dimension of the left atrium in systole and the left ventricle in diastole was 55 mm and 47 mm, respectively. There were no signs of valve perforation or vegetation on the mitral valve. Coronary angiography showed no coronary artery disease. The patient had a hemoglobin of 7.6 g/dL, mean corpuscular volume of 63.8 femtoliter, mean corpuscular hemoglobin of 21 picogram, mean corpuscular hemoglobin concentration of 32.9 g/dL. Peripheral blood smear displayed fragmented erythocytes. The patient's lactate dehydrogenase was 520 IU/L. Total and indirect bilirubins were 4.39 mg/dL and 4.08 mg/dL respectively. Serum iron was 45 µg/dL, and ferritin was 47.9 ng/dL. Coombs test was negative. Renal and hepatic functions were normal. After studying the other intrinsic or extrinsic causes of hemolytic anemia patients anemia was diagnosed as intravascular hemolytic anemia due to tight mitral stenosis. Therefore, we decided to perform mitral valve replacement.

Mitral valve exposure provided with median sternotomy and incision of the left atrium close to the atrial septum. Mitral valve was fibrocalcific, cusps of the mitral valve were thickened and stiffened. There was no vegetation or valve perforation. The mitral valve (29-mm St Jude mechanical valve) was inserted with everting interrupted mattress sutures. Postoperative course was uneventful. Postoperatively, her hemoglobin level increased, her lactate dehydrogenase and bilirubin gradually approached the normal range. Hemoglobin was 11

g/dL, lactate dehydrogenase was 343 IU/L, total and indirect bilirubins were 1.56 mg/dL and 0.94 mg/dL at 10 days after the mitral valve replacement. The patient was discharged on postoperative day 13. Control echocardiography revealed that maximal and mean gradients were 10 mm Hg and 6 mm Hg. Pulmonary artery pressure was 55 mm Hg. The third month follow up of the patient was uneventful.

Discussion

Intravascular hemolysis and hemolytic anemia can be encountered sometimes as complications, such as perivalvular leakage after prosthetic valve replacement or residual regurgitation after valve repair [2]. However, intravascular hemolysis related to native valve is rare.

Hemolytic anemia was clinically diagnosed by the combination of persistent severe anemia (hemoglobin ≤10 g/dL, hematocrit ≤ 33%), elevated lactate dehydrogenase (≥ 440 U/L), and the presence of schistocytes, fragmented cells, and polychromasia on peripheral blood smear [4]. In this case, there was no evidence for noncardiac cause of hemolytic anemia.

In patients with valvular heart disease, intravascular hemolysis is caused by flow disturbances. High-velocity jets increase shear forces that disrupt erythrocyte membranes and result in hemolysis [5]. Also rapid acceleration, fragmentation and collision of mitral regurgitation jets cause hemolysis [6]. In our case, echocardiography revealed severe mitral stenosis and moderate mitral regurgitation. We speculated that mitral stenosis and regurgitation may cause hemolytic anemia in this case, as was evidenced by the laboratory findings and clinical presentation. After the mitral valve replacement hemolysis was eliminated.

In conclusion, we report a case of hemolytic anemia due to mitral stenosis, resolved by mitral valve replacement. Although it is rare, hemolysis secondary to valve pathology should be kept in mind, while researching etiology of hemolytic anemia in a patient with valvular disease.

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