

Video 1, 2. Normal coronary arteries were revealed in coronary angiography

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Refractory hemolytic anemia due to severe swirling flow pattern in chronic mitral regurgitation after myxoma surgery

Miksoma cerrahisi sonrası gelişen kronik mitral yetersizliğine bağlı şiddetli "swirling"e bağlı refrakter hemolitik anemi

Introduction

While hemolytic anemia due to the paravalvular leaks is common in prosthetic valves, hemolytic anemia due to native mitral jet flow has not been described yet. It is generally believed that acceleration of regurgitation jet flow or red blood cell hitting to prosthetic valve causes fragmentation of blood cells and leads to hemolytic anemia in prosthetic valves. We thought that similar mechanisms may be responsible

in our case who had unexplained refractory hemolytic anemia for about 7 years.

Case Report

A 74-year-old male applied to our clinic with the complaints of dyspnea, early fatigue and palpitation. He had previous history of repeated blood transfusions for last 7 years reaching up to 54 units in total. He underwent left atrial myxoma and atrial septal defect operation 27 years ago. At the physical examination, the following findings were determined: pale skin, irregular and tachycardia heart rhythm, jugular venous distension and pretibial 2+edema. Four years ago, bone marrow aspiration was performed for anemia etiology with the prediagnosis of myelodysplastic syndrome but it did not reveal any pathological findings. Blood smear examination demonstrated red blood cell fragmentation, a few schistocytes and polychromasia. The count of thrombocyte was slightly reduced. Number of reticulocyte was 3%. Laboratory test results were low hematocrit 28.2% (42-52) hemoglobin 8.6 g/dL (range 13.5-18), white blood cell 10.5 (4-10.5) 10^3 u/L, Platelets 145 (150-450) 10^3 u/L normal hemoglobin electrophoresis, low serum haptoglobin level 0.1g/L (30-200 g/L) and negative direct Coombs' test, high serum total bilirubin level (2.2 mg/dL range 0.2-1.1), indirect bilirubin 1.67 mg/dL (normal value <1), low serum iron level (29 μ g/dL range for normal values 45-182), normal iron binding capacity (266 μ g/dL range 110-370), normal ferritin level 41.1 ng/mL (22-322), normal B12 and folic acid levels (200 pg/mL and 6.8 ng/mL respectively). Glucose-6-phosphate dehydrogenase enzyme level was found to be within normal reference values: 16.75 U/HGB (4.6-13.5). But serum LDH was high 535 IU/L (98-192) and alanin aminotransferase 40 mg/dL, aspartat aminotransferase levels 34 mg/dL, BUN 29 mg/dL, (8-25) creatinin 0.9. (0.8-1.2). Spleen was determined to be normal in abdominal ultrasonography. There was atrial fibrillation at electrocardiography. Cardiothoracic ratio increased in favor of heart at telecardiography. Transthoracic echocardiography revealed eccentric moderate degree mitral regurgitation. Because of its eccentricity, we had suspicion of severe mitral regurgitation thus we decided to perform transesophageal echocardiography (Philips Envisor C-HD, IPx-1 S6-2mpt Bothell WA, USA). At TEE, it was noted that left atrium was markedly dilated and there was eccentric severe mitral regurgitation towards left atrial lateral wall with swirling motion (Fig. 1, 2). We also detected a 3 mm small interatrial septal defect. Left

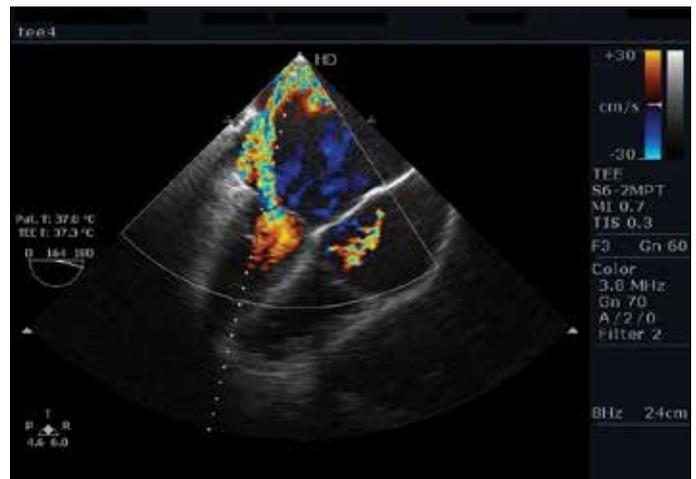


Figure 1. Transesophageal echocardiography view of the severe dilated left atrium and jet flow beginning from posterior mitral leaflet toward the septum

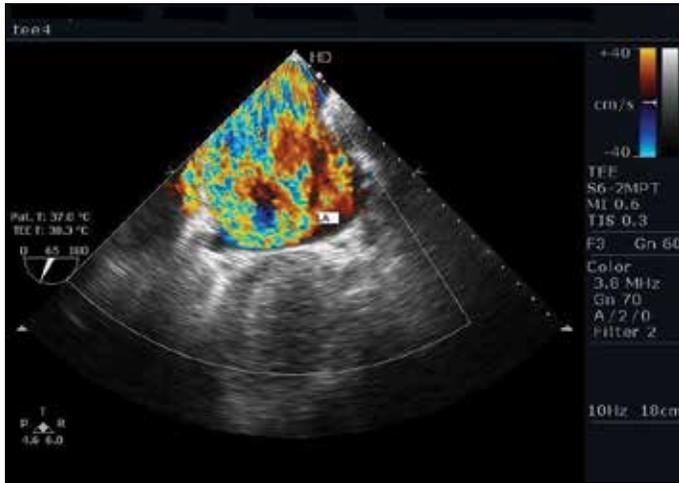


Figure 2. Transesophageal echocardiography view of an annular movement with 270 degree as swirling pattern toward interatrial septum

ventricle was dilated and ejection fraction was calculated as 56%. Right heart chambers were dilated and severe pulmonary hypertension was determined (pulmonary arterial pressure of 91 mmHg). Taking into account of patient clinical and laboratory situations, mitral valve surgery was advised and cardiac catheterization was planned but patient refused to go ahead. He was using furosemide 40 mg 1x1, spironolactone 25 1x1 and digoxin 0.25 mg 1x1.

Discussion

Hemolytic anemia in mechanical valve prostheses was encountered most frequently in situations of paravalvular leak (1) and less commonly after annular ring annuloplasty, mitral valve repair (2), percutaneous mitral valvuloplasty (3) and chordal rupture (4). The potential mechanisms of hemolysis in these cases includes fragmentation due to accelerated blood flow through a narrow orifice and direct hit of erythrocytes to surrounding native or foreign structures (left atrial wall, sutures, prosthetic valves ie.) (5, 6). However, hemolytic anemia has not been reported in native valve insufficiencies. Serum LDH value >500 IU/L is an important criterion for severe hemolysis (1). We could not explain hemolytic anemia in our patient with the presence of other diseases. Other possible hemolytic anemia or enzyme deficiencies causes were excluded (negative Coombs test, normal glucose-6-phosphate dehydrogenase enzyme level, normal hemoglobin electrophoresis and bone marrow aspiration). As a possible cause, we thought that high velocity mitral regurgitation jet passing through regurgitant orifice and directly impinging upon left atrial lateral wall may predispose erythrocytes to fragment. Furthermore, abrupt change of erythrocyte's motion within the left atrium due to swirling motion may also enhance susceptibility to fragmentation. We can also speculate that previous myxoma and atrial septal defect operation with patch makes interatrial septal surface rough and irregular which otherwise facilitates erythrocyte breakdown. We recommended surgery to patients for the hope that surgery may alleviate hemolytic anemia but he refused. We added a beta blocker to his drug therapy because there are reports suggesting potential use of beta blocker agents to decrease the hemolysis in these patients (7).

Conclusion

In this case, we have reported refractory hemolytic anemia that is likely to be caused by native mitral insufficiency. To the best of our knowledge, such an association has not been reported previously.

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Sağ ventrikül lead'i korunarak yapılan triküspit kapak cerrahisi sonrası intrakardiyak defibrilatör ve biventriküler Pacemaker'in (CRT-D) implantasyonu

Implantation of cardioverter-defibrillator and cardiac resynchronization device in a patient with preserved right ventricular lead after tricuspid valve surgery

Giriş

Triküspit kapak protezi olan hastalarda, transvenöz endokardiyal pacemaker (PM) ve kardiyoverter defibrilatör implantasyonu kontraendikedir (1). Bu nedenle sağ ventrikülde (RV) endokardiyal lead varsa