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Mitral valve repair-related hemolysis: a report of two cases

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Abstract

Two patients are described who suffered from progressive intravascular hemolysis following different kinds of reconstructive surgery of the mitral valve. Within the context of increasing numbers of operations aimed to preserve the mitral valve, the importance and difficulty of prompt recognition and adequate treatment of this very uncommon but potential lethal complication are emphasised. © 1997 Elsevier Science Ireland Ltd.

Keywords: Mitral valve repair; Intravascular hemolysis

1. Introduction

During the past two decades, mitral valve repair has become a well-established therapeutic option for treating mitral valve disease [1,2]. In the postoperative course, severe mechanical intravascular hemolysis is a very uncommon cause of morbidity [3]. In a report reviewing the first decade (1969–1978) of mitral valve reconstruction, 59% (22/37) of the reoperations were due to surgical technical factors [2]. Hemolysis as a cause for reoperation within the first two years after the initial reconstruction contributed 11% (2/18). In the report reviewing the second decade (1978–1988), hemolysis was no longer a cause for reoperation [4]. Nevertheless, several case reports have dealt with this complication which may cause multiple organ failure. [3,5]. We describe two patients. The first patient represents a

“clear-cut” case of early postoperative hemolysis due to ruptured tendinous cords following supra-annular suturing of the annuloplasty ring. The second case is an illustration of progressive hemolysis due to a rupture of the leafletplasty which occurred months after surgery. Hemolysis was treated by mitral valve replacement in both cases.

2. Case 1

A 70 year old male was referred to the Academic Medical Center of the University of Amsterdam for treatment of dyspnea, New York Heart Association (NYHA) class III, combined with angina, NYHA class I. Echocardiography showed prolapse of the posterior mitral leaflet due to chordal rupture and annulus dilation. Cardiac catheterization revealed coexistence of a significant stenosis of the left anterior descending (LAD) coronary artery. During

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surgery, quadrangular resection of the posterior leaflet was performed, followed by annuloplasty by using a flexible 35 mm Duran ring. A saphenous vein graft was used for revascularization of the LAD. Weaning from extracorporeal circulation was uneventful. On the seventh postoperative day, slight urine darkening was noted, followed by jaundice. Diuresis remained satisfactory and stools showed no abnormality. On physical examination, a grade II/VI holosystolic murmur was heard at the apex cordis. The abnormal laboratory results comprised hemoglobin (Hb) 4.5 mmol/l and hematocrit (Ht) 0.21 l/l; normal values 8.6–10.7 mmol/l and 0.41–0.55 l/l, respectively. Total serum bilirubin had risen to 35 μ mol/l (normal value <17 μ mol/l) and serum haptoglobin had dropped to 0.0 g/l (normal value 0.3–1.8 g/l). Lactate dehydrogenase (LDH) was 2475 U/l (normal value 300–620 U/l) and the reticulocyte count was elevated. Myocardial infarction, hepatitis, infection, and rhabdomyolysis could be excluded. Transesophageal echocardiography (TEE) revealed ruptured tendinous cords of the central scallop of the anterior mitral leaflet and supra-annular insertion of the annuloplasty ring. The patient's hemodynamic condition deteriorated and multiple blood transfusions were needed. On the 13th postoperative day the mitral valve was replaced by a St. Jude no. 27 mechanical valve. Postoperatively, sustained normalization of Hb and serum LDH were observed.

3. Case 2

A 54-year-old male was admitted to the University Hospital Utrecht for treatment of dyspnea, NYHA class II, based on mitral valve regurgitation combined with stable angina pectoris, NYHA class II. Cardiac catheterization showed significant triple-vessel coronary artery disease. Echocardiography revealed a prolapse of the anterior mitral leaflet. Surgery comprised triangular resection of the prolapsing central scallop of the anterior leaflet. The cleft was closed with interrupted sutures. Posterior annular remodeling was established with a bovine pericardial strip, sized to a 30 mm Carpentier–Edwards ring. Myocardial revascularization comprised grafting of the LAD, the second obtuse marginal branch, and the posterior descending coronary artery by using the left internal

mammary artery and two saphenous vein grafts, respectively. Intraoperatively performed TEE showed no residual regurgitation. The postoperative course was uneventful and the patient was discharged from hospital on the 10th postoperative day. Three months later, the patient complained of fatigue and urine darkening. The abnormal laboratory data showed that Hb had dropped from 7.9, at the day of discharge, to 6.3 mmol/l and Ht from 0.37 to 0.31 l/l. Serum haptoglobin had decreased to 0.1 g/l, and serum LDH had increased to 2500 U/l. However, TEE showed no changes compared with the intraoperative TEE. An immunological or toxic cause was excluded. The same applied for endocarditis and hemoglobinopathy. Finally, a bone marrow specimen revealed marked erythroid hyperplasia. Vitamin B₁₂, folic acid, and ferrous fumarate were administered. However, after six weeks of therapy, the patient had to be admitted to hospital due to progressive dyspnea and jaundice. On physical examination, a Grade III/VI holosystolic murmur could be heard at the apex cordis. LDH had increased to 5190 U/l, Hb and Ht had decreased further to 5.6 mmol/l and 0.27 l/l, respectively. Peripheral blood smear now showed markedly deformed micropoikilocytes, indicative of mechanically destructed erythrocytes. TEE revealed a distinct preavalvular acceleration of the blood stream at the central site of apposition of the anterior and posterior leaflet (Fig. 1). Although the patient remained hemodynamically stable, multiple blood transfusions were necessary. Approximately seven months after the initial operation, the patient was reoperated on. During surgery, good endothelialization of the bovine pericardial strip and a ruptured suture at the central point of apposition of the leaflets were seen. The valve was replaced by a 29 mm Björk–Shiley mechanical valve. Anemia and hemolysis disappeared rapidly. The patient was discharged from hospital on the 11th postoperative day.

4. Discussion

After open-heart surgery, intravascular hemolysis is mainly caused by mechanical destruction of erythrocytes in the presence of a mechanical heart valve, primarily in the aortic position. However, paravalvular leakage, infection, and blood transfusion reaction,

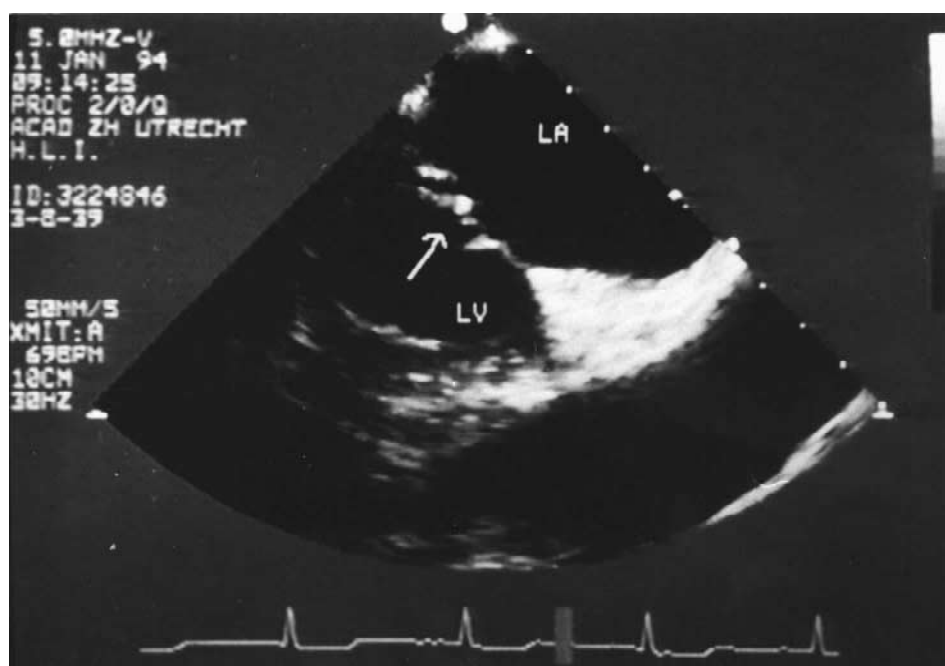


Fig. 1. Photograph showing a longitudinal view of the mitral valve, assessed by TEE. The arrow is pointed at the central site of apposition where the ruptured suture of the plasty of the anterior leaflet was located and a distinct preavalvular acceleration of the blood stream was identified by color-Doppler analysis. LA=left atrium, LV=left ventricle.

have to be ruled out [6]. Causes for reoperations within two years following the initial mitral valve repair include, dehiscence of the prosthetic mitral ring, oversizing of the ring, and residual regurgitation in the presence of interrupted sutures on pledgets of felt [4]. Case one of this paper illustrates the importance of surgical accuracy. The chordal cords had probably ruptured due to the shearing-stress, induced by the altered geometry of the mitral valve apparatus [5]. As reported by Mestres et al. [3], detection of the cause of hemolysis can be difficult which is illustrated by case two. Nonendothelialization of foreign bodies and the presence of a regurgitant jet, even of small hemodynamic significance, may play an important role in the origin of intravascular hemolysis. However, even in the absence of a regurgitant jet, assessed by TEE with color mapping and Doppler analysis [3], one has to consider the possibility of a cardiac cause of intravascular hemolysis. Up to now, only a few case reports have dealt with this subject. However, increased surgical experience has led to modification of repair techniques and expansion of the indications for mitral valve repair [1,4]. This is

represented by series in which up to 81% of the mitral valves subjected to surgery, could be preserved [1]. Thus, a small percentage of reoperations due to intravascular hemolysis might reappear in the report reviewing the third decade (1989–1999) of mitral valve repair. Based on the two presented cases, prompt recognition and adequate treatment of post-operative mitral valve repair-related intravascular hemolysis are emphasised.

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