A Surgical Case for Hemolytic Anemia after Ascending and Total Arch Replacement

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A 61-year-old man presented with consistent hemolytic anemia 15 months after ascending and total arch replacement for DeBakey I type acute aortic dissection. The cause of hemolysis turned out to be mechanical damage of red blood cells at the inverted felt of the proximal anastomosis. Reoperation of resection of the felt and repair of the proximal anastomosis successfully resolved this problem. We report a rare case of hemolytic anemia at the site of inverted felt strip after total arch replacement. (Ann Thorac Cardiovasc Surg 2005; 11: 416–8)

Key words: hemolytic anemia, total arch replacement, inverted felt

Introduction

Hemolysis by perivalvular leakage after mitral valve replacement is well recognized. However, hemolysis related to kinked prosthetic graft or felt strip is a very rare complication after aortic surgery.1) We experienced a case of hemolytic anemia due to inversion of the felt strip which was used at the proximal anastomosis of total arch replacement.

Case Report

A 61-year-old man underwent emergent ascending and total arch replacement because of DeBakey I type acute aortic dissection in July, 2003 at another hospital. The postoperative course was unremarkable except for compartment syndrome of the left leg which improved with conservative treatment. He was referred to our institution for the follow up in September, 2003. He presented with systolic ejection murmur of Levine II/VI at the left sternal border of second intercostal space. The computed tomography (CT) scan showed a dissecting aorta from the descending aorta to the left common iliac artery. The left subclavian artery and the left common carotid artery were also dissected. The left kidney was not enhanced.

Although he was totally asymptomatic, the laboratory data indicated hemolytic anemia and mild renal insufficiency. The hemoglobin (Hb), lactate dehydrogenese (LDH) and serum creatinine were 8.2 g/dl, 2,457 IU/L and 1.56 mg/dl, respectively. There was fragmentation of red blood cells by microscopy. He was admitted for further examinations in October, 2004. We also consulted our hematologists about this pathology. The Coombs’ test and cold agglutinin were negative. Surface IgG and IgM of red blood cells was negative by flowcytometry. These findings and close examination of bone marrow suggested a mechanical rather than immunological destruction of red blood cells in this case. The magnetic resonance angiography (MRA) showed severe stenosis at the proximal and the distal anastomosis and kinking of the prosthetic graft. (Fig. 1) We did not perform angiography in order not to damage renal function. The Hb dropped to 6.7 g/dl during hospitalization and we transfused 3 packs of red blood cells. The transthoracic echocardiography revealed no asynergy and normal valve function. Although we suspected fragmentation of red blood cells by turbulent flow at the distal anastomosis, there was not so much difference in blood pressure between arm and legs. We decided to do a reoperation for repair of the proximal anastomosis only and exploration for the stenotic parts.
inducing any hemolysis.

In November, 2004, he underwent the second operation. Before the re-sternotomy, the right subclavian artery was exposed for the cannulation and the right femoral artery and vein were also exposed and taped. Median sternotomy was carefully done using a harmonic scalpel. Extracorporeal circulation (ECC) was established together with the right subclavian cannula and the two-staged venous cannula of the right atrium. A thrill was felt at the ascending aorta. The proximal anastomosis had been wrapped by the prosthetic graft and the first cerebral branch of the graft was fairly close to the proximal anastomosis. Aortic cross clamp was done just proximal to the first branch when rectal temperature reached 28°C. The wrapping prosthesis was removed, and the graft was opened. Exploring the proximal anastomosis, the aortic wall was sandwiched by two felt strips and local dissection was absent. The outside felt was unremarkable and there was no rupture of sutures. However, the inner felt had been turned up and protruded to the internal lumen. (Fig. 2) The internal diameter of that level was only 12 mm. We attributed hemolysis to the collision or acceleration of blood flow at the level of the inverted felt strip. The inner felt was removed all around the lumen and the injured parts of the tunica intima were reinforced by two mattress sutures of proline. At 19.4°C, we declamped under circulatory arrest (CA) and selective cerebral perfusion (SCP) was established by balloon cannula from the inside of the graft. The proximal kinked graft was stretched. We also explored the distal kinking and found enough lumen, so we did nothing with that part. Because the stretched graft reached to the proximal anastomosis, we directly sutured to it using the remaining outside felt strip. The weaning from the ECC was relatively easy. The thrill had disappeared. The time of ECC, aortic cross clamp, CA and SCP were 225 minutes, 85 minutes, 40 minutes and 37 minutes, respectively.

The clinical course after the operation was very good. He was extubated the next day. Fragmented red blood cells disappeared. There was no progression of anemia and need for any blood transfusions. The postoperative MRA showed no obstruction in the ascending aorta. (Fig. 3) The LDH level gradually dropped to 258 IU/L by postoperative day (POD) 13. The renal function improved and serum creatinine went down to 1.17 mg/dl. He was discharged on POD 18.

Discussion

In this case report, we presented a rare case of hemolytic anemia at the site of inverted felt strip after total arch replacement. A similar case was previously reported by Izumi et al. They attributed hemolysis to dilation of the proximal aorta and graft constriction or the reversed inner felt. Firstly, the mechanism of hemolysis in our case was due to collision of red blood cells to the inverted felt and their fragmentation. Secondly, severe aortic stenosis at the site may have also contributed to the hemolysis to
some extent. The surgical repair of only the proximal anastomosis of the total arch replacement resolved his hemolytic anemia dramatically.

Hemolytic anemia by perivalvular leakage after mitral valve replacement is well recognized. Aortic prosthesis and subvalvular stenosis are also reported to induce intravascular hemolysis.\(^2\) Hemolysis is one of the complications after coil occlusion for patent ductus arteriosus.\(^3,4\) There are several studies about mechanical hemolysis after implantation of ringed intraluminal graft for type I aortic dissection.\(^5,6\) They are mainly because of kinking of the grafts and the resultant high pressure gradient. Although we did not measure the pressure gradient in the ascending nor descending aorta before the operation, we attributed hemolysis to the inverted felt which resulted in severe aortic stenosis, not the graft kink. From the intraoperative findings, the proximal graft kink was not more severe than the felt-induced stenosis. We also ruled out the possibility of hemolysis at the distal site of the graft by the normal ankle-brachial pressure index.

We successfully performed reoperation for a very rare case of consistent hemolytic anemia at the site of inverted felt strip after total arch replacement. Estrera and colleague reported that renal dysfunction and chronic obstructive pulmonary disease are the independent predictors of late mortality after reoperation of the proximal aorta.\(^7\) Thus we must continue close follow up of the renal function and late dilatation of the descending aorta. We should also pay attention to the possibility of hemolysis at the felt strip of the distal anastomosis in the future.

References