Mitral Valve Repair for Mitral Insufficiency Due to Infective Endocarditis in a Patient with Idiopathic Thrombocytopenic Purpura

Akira Marumoto, MD,1 Yasushi Ashida, MD,2 Hiroaki Kuroda, MD,3 Takafumi Hamasaki, MD,4 Satoshi Kamihira, MD,1 Shingo Ishiguro, MD,1 and Shigetsugu Ohgi, MD1

From 1Department of Surgery, Division of Organ Regeneration Surgery, Faculty of Medicine, Tottori University, Tottori, 2Department of Cardiovascular Surgery, Matsue City Hospital, Shimane, 3Department of Cardiovascular Surgery, San-in Rosai Hospital, Tottori, and 4Department of Cardiovascular Surgery, Hamada National Hospital, Shimane, Japan

A 63-year-old woman with an 18-year history of idiopathic thrombocytopenic purpura (ITP) was admitted with a persistent fever of unknown cause. Blood culture was positive for α-Streptococcus and echocardiography revealed severe mitral regurgitation and vegetation on the mitral valve. After antimicrobial therapy for six weeks, she underwent mitral valve repair using a Cosgrove ring. The platelet count increased and remained stable by perioperative treatment with intravenous high-dose gamma-globulin and platelet transfusion without steroids therapy or splenectomy. The hospital course was uneventful. Perioperative high-dose gamma-globulin therapy and platelet transfusion for the cardiac operation were useful to increase and maintain the platelet count for an ITP patient complicated with infective endocarditis. (Ann Thorac Cardiovasc Surg 2005; 11: 48–50)

Key words: idiopathic thrombocytopenic purpura, infective endocarditis, gamma-globulin, mitral valve repair

Introduction

Critical factors for cardiac surgery in a patient with an autoimmune coagulation disorder such as idiopathic thrombocytopenic purpura (ITP) are to avoid the depression of platelets and achieve hemostasis by intensive perioperative management. We report the case of a patient with ITP who successfully underwent mitral valve repair for severe mitral valve regurgitation due to infective endocarditis (IE).

From 1Department of Surgery, Division of Organ Regeneration Surgery, Faculty of Medicine, Tottori University, Tottori, 2Department of Cardiovascular Surgery, Matsue City Hospital, Shimane, 3Department of Cardiovascular Surgery, San-in Rosai Hospital, Tottori, and 4Department of Cardiovascular Surgery, Hamada National Hospital, Shimane, Japan

Received January 22, 2004; accepted for publication September 20, 2004. Address reprint requests to Akira Marumoto, MD: Department of Thoracic and Cardiovascular Surgery, Tottori Prefectural Central Hospital, 730 Ezu, Tottori 680-0901, Japan.

Case Report

A 63-year-old woman was admitted with a persistent fever of unknown cause for about 4 weeks. She had been followed for ITP for 18 years without any treatment. The patient had no history of bruising or bleeding manifestations. She was in NYHA class II at admission, and had holosystolic murmur at the apex. Body temperature was elevated at 39.0°C and a bacteriological examination of blood was positive for α-Streptococcus. Echocardiography revealed severe mitral regurgitation and dilated left atrium of the heart. Multiplane transesophageal echocardiography showed a mobile mass that originated on the surface of the anterior mitral-valve leaflet and a prolapse of the antero-lateral scallop of the posterior mitral-valve leaflet. After antimicrobial therapy, intravenous administration of Penicillin G sodium 16 million U/day for 6 weeks, blood culture was negative, inflammatory reactions improved, and heart failure had not progressed. Left ventricular ejection fraction of transthoracic echocardiography was 75%. The platelet count was 20,000 cells/mm3 on admission. Coagulation screen (prothrombin time
was 118.5% and activated partial thromboplastin time was 26.7 sec) was normal, but the function of platelets had declined slightly. No anti-platelet antibody was detected. For the mitral valve operation, the patient received 5 days of intravenous high-dose gamma-globulin therapy at 0.4 g/kg/day. At the operation, cardiopulmonary bypass was established with a centrifugal pump (Termo Inc., Tokyo, Japan) and heparin-coated extracorporeal circuits (Termo Inc.). Heparin was given (250 units/kg) and the activated coagulation time (ACT) was monitored by Hemochron (International Technidyne Corp., Edison, NJ), and ACT was kept over 400 seconds during cardiopulmonary bypass. Anticoagulation was managed using the Hepcon HMS PLUS (Medtronic, Minneapolis, MN), a device that calculates an individual’s heparin dose response and permits assessment of the heparin concentration throughout the operation. A conventional median full sternotomy was made, and superior transseptal approach was performed. No vegetation was found on the anterior mitral-leaflet, and there was severe mitral regurgitation due to two perforations (3 by 3 mm) on the postero-medial scallop (P3) and a perforation on the prolapsed antero-lateral scallop (P1) of the posterior mitral-leaflet, and mitral annula was dilated to 31 mm. The patient underwent mitral valvuloplasty using a quadrangular resection of the perforated antero-lateral scallop of the posterior mitral-leaflet and ring annuloplasty with a 28 mm Cosgrove-Edwards ring. The perforated P3 leaflet was repaired with interrupted sutures of 5-0 polyester. The patient came off the cardiopulmonary bypass without difficulty, and the aortic cross-clamp time was 105 min and the extracorporeal circulation (ECC) time was 148 min. Thirty units of platelets were transfused perioperatively. Intravenous high-dose gamma-globulin therapy was administered for 5 days after operation and the platelet count increased to 63,000 cells/mm² (Fig. 1). The postoperative course was uneventful. The chest tubes were removed on the third postoperative day without excessive bleeding, with a total drainage of 550 ml and oral anticoagulant therapy with warfarin was begun. Microscopic findings revealed the usual scars of endocarditis without degenerative mitral valvulopathy or myxomatous degeneration.

On postoperative day 15, cardiac catheterization was performed and no mitral regurgitation was observed. The patient’s clinical course was unremarkable without infectious complications and she was discharged on postoperative day 36. Platelet count on discharge was 78,000 cells/mm².

Discussion

ITP is an autoimmune disorder of increased platelet destruction mediated by autoantibodies to platelet-membrane antigens and characterized by a reduced number of circulating platelets that is often fewer than 50,000 cells/mm². The most notable clinical manifestations are spon-

![Fig. 1. Perioperative platelet count and PAIgG. The solid line shows platelet count and the broken line shows PAIgG. Plt: platelet transfusion](image-url)
taneous hemorrhage and excessive posttraumatic bleeding. Thus the critical factor for surgical treatment in a patient with ITP is to achieve adequate hemostasis by intensive therapy. In particular, perioperative therapy is important for cardiac surgery under cardiopulmonary bypass leading to platelet dysfunction. Reports of patients with ITP undergoing cardiac operations are scarce. And to our knowledge there has been only one report including a patient complicated with IE, in which hemostasis was difficult because the therapy for ITP was only platelet transfusion. Steroids, immunosuppressive drugs, Danazol, high-dose gamma-globulin therapy and splenectomy have all been advocated as treatments for ITP. Steroids and immunosuppressive drugs were not suitable in this patient with mitral insufficiency caused by IE, since it has been reported that they might increase the risk of infection. In some reports, splenectomy was performed prior to or combined with cardiac surgery. Response to splenectomy typically occurs within several days and refractoriness to splenectomy is reported to be approximately 30%. Splenectomy was thought to be overly invasive for cardiac function and had a harmful effect on IE. The mechanism of intravenous high-dose gamma-globulin therapy remains to be elucidated. The major effect is thought to be macrophage Fc receptor blockade, decrease in autoantibody synthesis, protection of platelets or megakaryocytes from platelet antibody, and clearance of persistent viral infection by infusion of specific antibodies. Platelet counts usually increase 5 to 7 days after administration and then are restored to the previous level within 1 month. The increased platelet production is unopposed and large, young platelets, which are highly effective in hemostasis, are released into the circulation. In our case, preoperative high-dose gamma-globulin therapy induced an increased platelet count immediately but it decreased two days before the operation. By platelet transfusion during and just after the operation and perioperative high-dose gamma-globulin therapy, the platelet count was increased and maintained over 50,000 cells/mm² in spite of a gradual elevation in platelet associated IgG (PAIgG) after the operation (Fig. 1). Heparin coating improves biocompatibility by reducing both component activation and platelet loss on the surface of the ECC. The effect of platelet transfusion is usually transient and multiple platelet transfusions result in refractoriness to subsequent platelet transfusion. Perioperative high-dose gammaglobulin therapy was thought to be effective to minimize the need for platelet transfusions and to prevent the fall in the number of the platelets.

In conclusion, perioperative intravenous high-dose gamma-globulin therapy and platelet transfusion were effective for cardiac surgery in a patient with ITP, in whom performance of steroids therapy, immunosuppressive drugs and splenectomy are associated with an increased risk of perioperative infectious complication and cardiac morbidity.

References