

Immune thrombocytopenic purpura healing after removing cardiac thrombosis

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Abstract

Because of the rareness of the reports about immune thrombocytopenic purpura patients undergoing cardiac surgery, there is no sufficient knowledge about perioperative strategies. In this article, we report a 42-year-old female patient with severe mitral regurgitation and large right atrial thrombus, in whom thrombocytopenia had disappeared after mitral valve replacement and thrombectomy from the right atrium. Large thrombi should be kept in mind as the cause of thrombocytopenia.

Key words: open heart surgery, immune thrombocytopenic purpura, thrombocytopenia, cardiac thrombus

Introduction

Publications related to open-heart surgeries in thrombocytopenic patients are rare. Therefore, there is no consensus on perioperative treatments in different situations that create thrombocytopenia. Large thromboses are also clinical problems that can cause thrombocytopenia. In this article, we present a patient whose platelet levels were low despite treatment for immune thrombocytopenia purpura (ITP) but whose platelet levels returned to normal after cardiac thrombosis surgery.

Case presentation

A 42-year-old female patient was taken into coronary intensive care after transesophageal echocardiography (TEE) was performed in a cardiology clinic due to complaints of dyspnea, fatigue and cough, advanced mitral regurgitation, and vegetation extending to the right atrium wall on a permanent catheter in the right atrium. The patient partially recovered with medical treatment and was subsequently admitted to the ward for surgical intervention. The patient had been treated for immune thrombocytopenia purpura (ITP) for 17 years. Splenectomy was performed eight years ago and during the last two years, she had been dialysis twice a week due to decreased renal function. The patient was using 50 mg of eltrombopag daily for ITP and 40 micrograms of darbepoetin alfa once a week for anemia due to chronic renal insufficiency. In TEE, transthoracic echocardiography revealed a vegetative mass of 38x23 mm

thrombus in the right atrium extending from the catheter tip to the posterior leaflet of the tricuspid valve in the control of the patient with advanced mitral insufficiency. Blood tests revealed a platelet count of 43,000/ μ L and hemoglobin of 8.3 mg/dL. In the hematology department, the patient underwent thrombocyte replacement, with a platelet count above 100,000/ μ L prior to surgery. The patient was taken into dialysis the evening before surgery.

After obtaining written informed consent, the patient was taken to the operating room. After standard median sternotomy and aorto-bicaval cannulation, one unit erythrocyte suspension was added to the reservoir to provide appropriate hematocrit levels. The right atrium was opened. The permanent dialysis catheter has an organizing thrombus approximately 1 cm thick surrounding the catheter on the part of the right atrium. After the catheter was cut from the highest possible level, the remaining part was removed from the entrance point in the neck. There was a mass in the right atrium of about 5x4 cm with irregularly restricted, dark gray-navy colored, right atrium infiltrated into the near wall of the diaphragm and extending to the tricuspid valve. The mass was removed. The infiltrated part of the right atrium wall was thoroughly scraped and cleaned. The mass was sent to the microbiology laboratory for culture purposes. Then the interatrial septum was opened. The mitral valve was rheumatoid. The subvalvular structures under the posterior annulus were attached to the leaflets and the annulus. The chordae tendineae (tendinous chords) were sharply shortened and fused. The anterior leaflet was excised, and

the posterior leaflet was preserved. With 16 pledget stitches, a 27-size mechanical prosthetic valve (St. Jude Medical, St. Paul, Minn., USA) was placed. After the closure of the septum and the right atrium and de-airing of heart chambers, the x-clamp was removed. The surgical procedure was terminated by continuing routinely.

Drainage during the first 24 hours was 400 cc. No platelet replacement was needed. The patient's pre-op and post-op thrombocyte and hemoglobin values are given in Table 1. 6,000 units of enoxaparin twice a day was started at the 12th hour postoperatively. The next morning, warfarin was added. The drainage of the patient on the third postoperative day was lowered to less than 100 cc/24h, and the chest tubes were removed. The patient was transferred to the cardiac surgery (or cardiology) ward. The patient was dialyzed on routine dialysis days during the hospitalization period. The patient was discharged eventless on the 10th postoperative day. Microbiological examination of the intraoperative material revealed a pseudomonas aeruginosa infection, and pathological examination revealed fibrinous tissue showing myxomatous changes.

The patient was seen on the seventh day, third week, and sixth week after discharge. The results of the tests performed here are given in Table 2. The patient was not given any medication to stimulate thrombocyte suspension or platelet production in her follow-up visits. The patient is still followed up routinely by monthly controls, and six months after the operation, her platelet levels are normal. The patient does not use any medication for thrombocytes.

Discussion

ITP is an autoimmune disease caused by autoantibodies to thrombocytes. These autoantibodies generally occur against the glycoproteins IIb/IIIa or Ib/IX present in the platelet membrane. The autoantibody-bound thrombocyte is removed from the circulation by phagocytosis through the macrophages, but not through the life cycle, thus resulting in thrombocytopenia [1]. However, platelet production is also suppressed. The incidence in adults is approximately 1.6 to 2.68/100,000 and is more common in women [2].

Reports about open-heart surgeries on ITP patients are very rare. Only 0.2% of open-heart surgery candidates consist of ITP patients [3]. Therefore, there is no consensus on the treatment of ITP patients during open-heart surgeries. The first choice in ITP treatment are glucocorticoids and intravenous immunoglobulin (IVIG), which usually results in an effective and transient increase in platelet counts. However, if effective results cannot be obtained as a result of these treatments, rituximab, splenectomy, thrombopoietin receptor agonists (TPO-A), and immunosuppressants can be used [4]. The platelet increase provided by these treatments is usually longer. One of the most important problems of IVIG treatment is that it can cause renal failure. In our case, IVIG could not be used due to the affected renal functions. Splenectomy was already done eight years ago, and steroid was already in use. For this reason, a direct platelet replacement was performed to increase the platelet count before the operation, and the platelet count was increased to 100,000/ μ L. Platelet values during the hematology follow-up and diagnosis of intracardiac thrombus are shown in Table 1.

Table 1 Early Postoperative Results

Date	Haemoglobin (g/dL)	Thrombocyte ($10^3/\mu$ L)
Preoperative	8,3	43
Postoperative	9,1	79
Postoperative first day	8,9	96
Postoperative second day	9,0	103
Postoperative third day	9,2	82
Postoperative fourth day	9,1	87
Postoperative fifth day	9,3	82
Postoperative sixth day	9,6	102
Postoperative seventh day	9,4	116
Postoperative eighth day	9,4	181
Postoperative ninth day	8,8	237
Postoperative tenth day	9,1	254

Table 2 Outcome of Outpatient Clinic

	Haemoglobin (g/dL)	Thrombocyte ($10^3/\mu$ L)
First Check	9,4	275
Second Check	10,4	212
Third Check	8,5	295

It is interesting that the patient's thrombocyte levels steadily dropped before surgery and her platelet counts were steady at normal levels even one year after the operation. The patient has not received any medication for ITP for the past year despite excessive menstrual bleeding. Although the excess in menstrual bleeding is due to ITP, it has been attributed to the use of warfarin by physicians. We think that the reason for the patient's low platelet level in the preoperative period is the large thrombus in the right atrium despite the treatment. We believe that watching platelets at normal levels without treatment after removal of the thrombus during the operation also supports this view. Studies on ITP patients have shown an increased

risk of thrombosis in these patients. Aledort and colleagues detected 18 thromboembolic events in 186 chronic ITP adult patients [5]. These events were thought to be associated with antiphospholipid antibodies (APLAs), although the cause of thrombus development was not fully elucidated [6]. In APLA-positive patients, thrombosis is more common. It has been previously shown that large thromboses in the body may cause thrombocytopenia by agglutinating platelets. Kitchens published three case reports of thrombocytopenia in deep vein thrombosis, indicating that thrombocytopenia may interfere with heparin-induced thrombocytopenia [7].

Conclusion

If thrombocytopenia exists in an open-heart surgery candidate, its cause should be diagnosed correctly, and preparations should be made accordingly. However, it should be kept in mind that large thrombi formations may be the cause of thrombocytopenia even though the patient has already been diagnosed. While publications on open-heart surgeries in ITP patients are very rare, open-heart surgeries can be safely

performed in these patients after necessary precautions have been taken.

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