

Therapeutic platelet reduction: Use in postsplenectomy thrombocytosis

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Abstract:

Therapeutic platelet reduction is an effective modality for the reduction of platelet count in patients with treatment of extreme thrombocytosis resulting from a variety of primary and secondary causes of thrombocytosis, which may be associated with thrombotic or hemorrhagic complications of varying degrees. These cases when symptomatic fall into the ASFA Category II indication for therapeutic platelet apheresis procedure. Here, we report a case of postsplenectomy secondary thrombocytosis presenting with extremely high platelet counts and subsequent thrombosis in the shunt and successful treatment after therapeutic platelet reduction. The case is being presented to bring forth the fact that therapeutic platelet reduction is an easy procedure that gives quick and good results and also to bring to the attention of transfusion specialists an associated but as yet unreported procedural finding.

Key words:

Postsplenectomy, therapeutic platelet reduction, thrombocytosis

Introduction

Extreme thrombocytosis may result from a variety of primary and secondary causes of thrombocytosis that may be associated with thrombotic or hemorrhagic complications of varying degrees, and therapeutic platelet reduction is an effective modality for the treatment of symptomatic patients. The common causes of reactive thrombocytosis are infection, trauma, surgery, drugs and occult malignancy.^[1] Markedly increased platelet counts in myeloproliferative disorders or postsplenectomy patients may lead to life-threatening thrombotic and/or hemorrhagic complications.^[1] If started timely, the therapeutic apheresis procedure can contribute to reduction in the morbidity associated with the hemorrhagic or thrombotic manifestations of thrombocytosis.

Case Report

A 42-year-old male presented to the surgery ward with pain, vomiting and fever for 5 days. The patient had a history of prior hospitalization as he was a known case of portal hypertension due to extrahepatic portal vein obstruction being managed conservatively in the Gastroenterology Department. The patient was found to have tender splenomegaly. Ultrasound and magnetic resonance imaging revealed portal thrombosis and formation of many collaterals along with splenomegaly, which was approximately 17.7 cm in size. The patient was put on Ecospirin 75 mg. Endoscopy revealed Grade II esophageal varices. Complete blood counts were within normal limits.

Splenectomy was performed with mesocaval shunt using Dacron graft. Histopathology revealed congestive splenomegaly. Postoperatively, the patient developed paralytic ileus and was managed conservatively. Hemogram performed on the second postoperative day revealed markedly increased total leukocytes (31,600/cumm) and platelets (15.8 Lakh/cumm) with hemoglobin value of 11.4 gm/dL. The patient also developed thrombosis in the shunt, which was worsening the condition further as the patient was having fluid collection in the peritoneal cavity. After ruling out, clinically, any possibility of concurrent infections and all causes of secondary thrombocytosis based on history and relevant hematological and other investigations, the patient was started on hydroxyurea and, as the counts were persistently high (>15 Lakhs/cumm), the "Therapeutic Platelet Reduction" procedure was performed using an MCS + Hemonetics blood cell separator. The procedure was performed 1 month after the splenectomy when the patient developed obstruction of the mesocaval shunt due to thrombosis. The apheresis was performed on three sittings performed sequentially on three consecutive days. An unusual occurrence in the procedure was recurrent blocking in the platelet tubes and centrifuge bowl at the time when platelets were sedimented and being diverted to the platelet bag. It was happening probably owing to high stickiness that was managed by modification of the machine parameters. At this time, the return cycle had to be started manually earlier than scheduled and, as a result, less platelets could be removed than expected on the first procedure. As the patients counts came down, the stickiness was lesser in the

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subsequent procedures and the apheresis could later be performed more effectively. Finally, the platelet count reduced to 9.5 Lakhs/cumm and, as the patient was symptomatically relieved as well and comfortable, he was discharged. Later, the patient continued with medical treatment and the platelet count reduced to 5 Lakh/cumm after 5 months of follow-up.

Discussion

Postsplenectomy thrombocytosis is a known postoperative complication that has been reported by many authors.^[1-3] An unusual case has been described of a very young patient having primary thrombocythemia with gross elevation of the platelet count following splenectomy as well thus showing diagnostic and therapeutic difficulties as thrombocytosis was an expected postsplenectomy finding in this rare patient who also had an underlying myeloproliferative disorder.^[3]

In one study, splenectomy was found to be one of the main causes of thrombocytosis, and the probability of thrombocytosis in patients who have had splenectomy is about 75-82%; about 9% of all reactive thrombocytosis occurrences are caused by this procedure.^[2]

Many studies have revealed thrombotic and/or hemorrhagic complications in patients with such high platelet counts.^[4]

Although the exact mechanisms of thrombosis after splenectomy remain unclear, altered platelet function as well as transient thrombocytosis after splenectomy, a decrease in portal blood flow and pressure and stasis of blood in the stump of the splenic vein appear to predispose to Portal Vein-Superior Mesenteric Vein thrombosis.^[5] Less commonly, postsplenectomy thrombocytosis results in arterial thrombosis that leads to stroke or myocardial infarct.^[2]

Many studies have reported that such patients when treated urgently by plateletpheresis in combination with the myelosuppressive therapy procedure experience less morbidity associated with hemorrhagic or thrombotic manifestations of thrombocytosis.^[4,6]

Three patients with marked thrombocythemia secondary to myeloproliferative disorders were treated with plateletpheresis using an Aminco cell separator and myelosuppressive agents. Immediate control of the raised platelet count was achieved by plateletpheresis and long-term control by the concomitant cytotoxic therapy.^[7]

In another study where reduction of platelet counts was achieved rapidly, abnormal platelet aggregation testing was present before pheresis, but improved immediately after pheresis. Platelet-sizing data obtained in one case suggested that during the pheresis procedure, a population of larger volume platelets was selectively removed. The efficacy of plateletpheresis in these clinical situations may be related to the selective removal of a large abnormal platelet population.^[8] Two or more procedures — either on consecutive or on alternate days — are usually required to achieve adequate reduction in the platelet count. Asymptomatic thrombocytosis does not require removal of platelets unless the patient has a prolonged

bleeding time and is about to undergo an operation. Long-term thrombocytapheresis in chronic diseases is not effective.^[9]

The unusual occurrence in the procedure, i.e. recurrent blocking in the platelet tubes, decreased with the subsequent procedures as the counts improved. Such instances have been experienced but not reported in the literature and therefore it becomes an important issue that needs detailed study in bigger groups of patients to understand the mechanism involved and the ways to prevent it.

These patients who are symptomatic due to extreme thrombocytosis fall under the Category II indication for therapeutic apheresis according to the American Society for Apheresis guidelines.^[10] Very few case reports are found in the literature about such an effective modality in the management of thrombosis following severe thrombocytosis. More such cases need to be published to enable sharing of data about this highly effective modality.

Conclusion

In the case reported, it was found that splenectomy was the prime cause of severe thrombocytosis and the debilitating complication of thrombosis in the mesocaval shunt was causing further morbidity that could be successfully taken care of by the therapeutic apheresis procedure.

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