Life-Threatening Massive Thromboembolism After Laparoscopic Splenectomy in an Idiopathic Thrombocytopenic Patient

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Abstract: A life-threatening massive thromboembolism is reported that developed after laparoscopic splenectomy in a 57-year-old female with idiopathic thrombocytopenic purpura (ITP). Although asymptomatic, she underwent splenectomy because of poor response to steroids and intravenous (IV) gamma globulin. Thirteen days after splenectomy, she suffered portal and mesenteric emboli, followed by pulmoner emboli and left popliteal thrombophlebitis. Extensive workup for hypercoagulable states was negative. Low molecular weight heparin (LMWH) was initiated at a suboptimal dose because of thrombocytopenia. During follow-up, her platelet count increased gradually. Whenever the platelet count had remained stable at > 50 x 10^9/L, she received full dose of LMWH treatment. Over the next 38 days, her pain resolved, she tolerated a full diet, and sent home. Follow-up imaging studies demonstrated a recanalized portal vein and totally resolved pulmonary arteries. We thought that, consumption of platelets in the massive thromboembolism sites including portal, mesenteric and pulmoner vascular beds had resulted in deep thrombocytopenia, which improved gradually with anticoagulant therapy.

Keywords: Portal vein thrombosis, Anticoagulation, Low molecular weight heparin.

INTRODUCTION

Splenectomy is performed for diagnosis or treatment of a variety of hematologic diseases including immune cytopenia, indolent non-Hodgkin's lymphoma, and, rarely, for myeloproliferative diseases [1]. Within the recent years, laparoscopic splenectomy has been increasingly performed and is associated with a decreased rate of intraoperative organ damage and quicker postoperative recuperation of the patient [2].

Postoperative complications most often comprise infections [2]. Additionally, patients with immune thrombocytopenia and low platelet counts may have an increased perioperative bleeding risk. Thromboembolic complications following splenectomy for hematologic diseases occur in up to 10% of patients and may range from portal vein thrombosis (PVT) to pulmonary embolism (PE), and deep vein thrombosis (DVT) [3]. Prompt diagnosis is critical and is typically achieved via color doppler ultrasonography [4] or computed tomography [5].

We present a mid-aged woman with the diagnosis of ITP who suffered from postsplenectomy massive venous thromboembolism (VTE) including portal, splenic, superior and inferior mesenteric, pulmonary veins. It was very surprising for us that, she had a deep thrombocytopenia at the time of the massive VTE, and the extensiveness of the thrombosis was widespread in comparison with those reported in the literature. Therefore, we decided to share our management of this rare and mortal complication, and finally, to review the current medical literature.

CASE PRESENTATION

A 57-year old female patient with deep thrombocytopenia (9 x 10^9/L) presented to our clinic because of prevalant petechiae and bruising on her legs. The differential diagnosis of thrombocytopenia was carefully evaluated. Endocrinologic and connective tissue diseases were excluded. Hepatitis, viral and bacterial tests were all negative. She underwent a bone marrow biopsy and histologic examination revealed normal findings in megacaryocyte morphology and a mild increase in myeloid hematopoietic cells. Afterwards, the patient was diagnosed as ITP. She had received 2 units of apheresis platelets and a short course of therapy with peroral methylprednisolone (1 mg/kg), and a complete remission was obtained with this treatment. However, only 2 months later, she presented with generalized bruising in her body and the platelet count was only 4 x 10^9/L. A second attempt

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including 1 unit of apheresis platelets and oral steroid was initiated, a second complete remission was achieved. A splenectomy was recommended. Because the patient had no history of previous abdominal surgery, a laparoscopic splenectomy was proposed and accepted.

Laparoscopic splenectomy was carried out at a platelet count of 82 x 10^9/L with no bleeding complications, and after quick recovery she was discharged on day 3 after an uneventful operation with a platelet count of 232 x 10^9/L. On postoperative day 16, she presented with a 1-day history of acute abdominal pain. Her pain was periumbilical without radiation. Her vital signs were in normal ranges, and the abdominal examination was significant for mild periumblical tenderness without guarding or rebound tenderness. Laboratory studies revealed the following: white blood cell count (WBC), 21.9 x 10^9/L; hematocrit, 33.5%; platelets, 18 x 10^9/L. Blood chemistries were within normal limits except a mild increase in urea (57 mg/dL, normal range: 10-50) and LDH (314 U/L, normal range: 120-247) levels. An abdomen ultrasound (US) revealed wall thickness (8 mm) and edema of the left intestinal loops. A computed tomography (CT) scan of the abdomen and pelvis (Figures **1A, 1B, 1C**) revealed thrombosis involving the portal vein, extending into the branch veins, thrombus of the splenic (totally occluded), superior mesenteric, and inferior mesenteric veins. Free fluid (51 x 84 mm) at the operation site and a significant amount of pelvic free fluid, wall thickness and edema of the jejunal loops

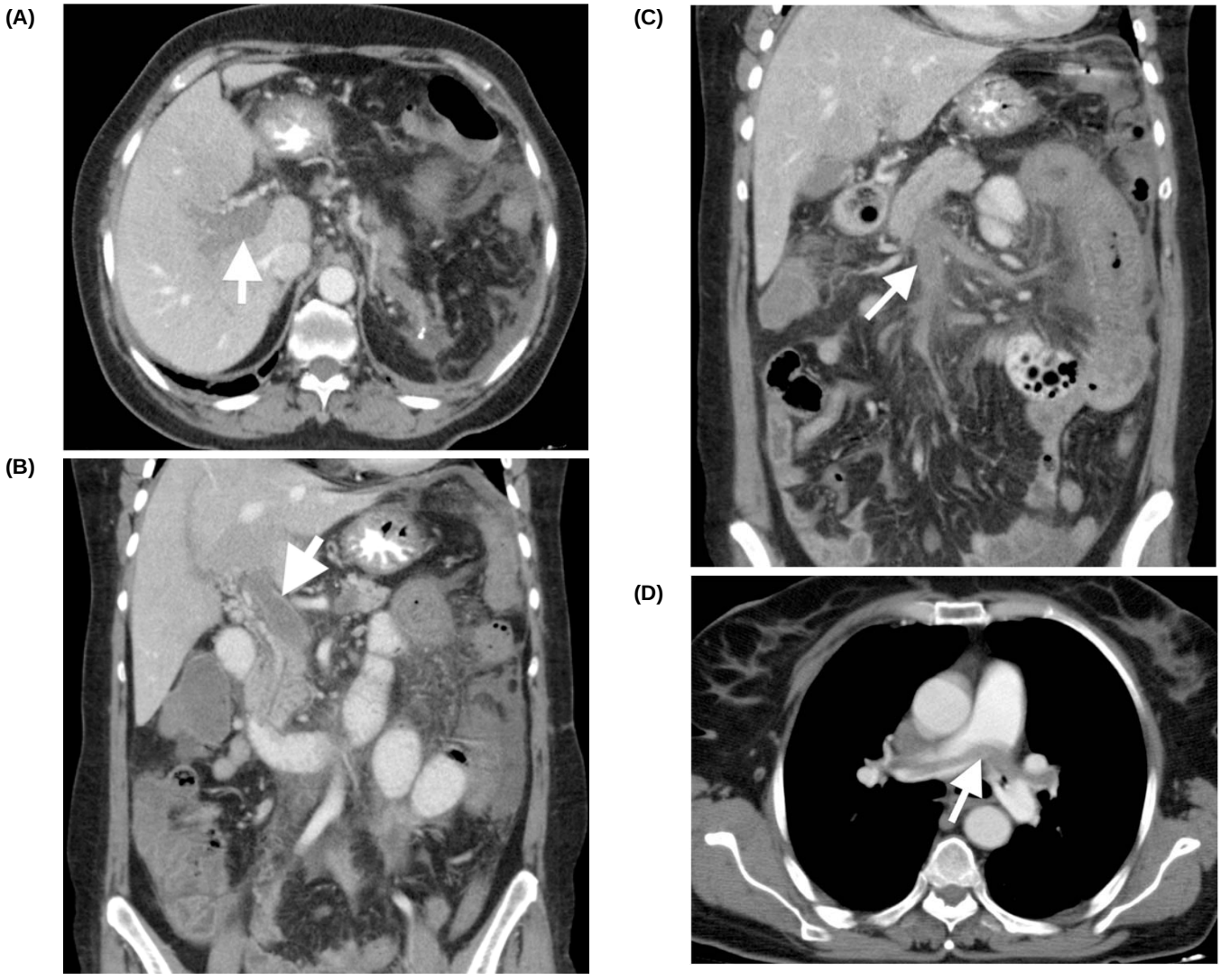

Figure 1. Thrombosis of portal vein and pulmonary artery. Axial **(A)** and coronal **(B)** contrast enhanced CT images demonstrate hypodense filling defects (arrows) in portal veins consistent with portal vein thrombosis. **(C)** Coronal contrast enhanced CT reveals thrombosis of superior mesenteric vein (arrow). **(D)** Axial contrast enhanced CT reveals hypodense filling defect (arrow) resulting in partial thrombus of pulmonary artery.

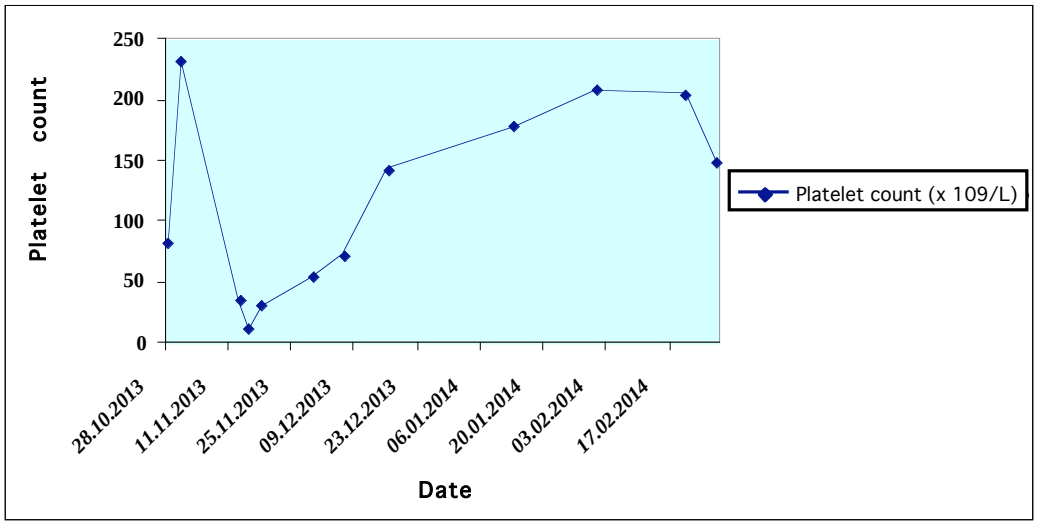

Figure 2. Platelet count (x 10^9/L) of the patient during follow-up.

located at the left upper quadrant were also reported (venous ischaemia). A thorax CT scan (Figure **1D**) showed filling defects compatible with pulmonary emboli in the main pulmonary artery, bilateral lobar arteries, and some segmented and subsegmented branches. In addition, a venous Doppler US of lower extremities revealed left popliteal thrombophlebitis.

Oral intake was stopped after diagnosis of multiple thrombosis in vessels. Intravenous immunoglobulin 1 gr/kg, for 2 days was administered, and her platelet count rose to 28 x 10^9/L. Surgery department recommended conservative approaches, but not a re-operation. Low molecular weight heparin (enoxaparin, Clexane®, 4000 anti-Xa IU/0.4 ml) was initiated at a suboptimal dose because of thrombocytopenia. Unfortunately, an upper gastrointestinal hemorrhage presenting with melena occurred. Therefore, we had to stop LMWH. The patients' relatives did not accept the endoscopic examination. One day later, gastrointestinal bleeding was stopped and we started LMWH at a low dose again. This time gastrointestinal bleeding did not repeat. During follow-up, her platelet count increased gradually as shown in Figure **2**. Whenever the platelet count had remained stable at > 50 x 10^9/L, she received full dose of LMWH treatment. At discharge, her medical management included oral diet of normal foods, and anticoagulation with LMWH.

She had no prior history of thrombophilia, and exhibited no further obvious clinical risk factors for thromboemboly, such as prolonged immobilization, infection or heart failure. A workup for hypercoagulability states (factor V G1691A, factor II G20210A, MTHFR C677T mutations, protein C and S deficiency, anti-cardiolipin IgA, G, M) yielded normal results. However, she had heterozygous MTHFR A1298C, PAİ-I 4G/5G and β-fibrinogen gene 455 G>A mutations. Fluorescently labeled inactive toxin aerolysin (FLAER) panel was performed to exclude paroxysmal nocturnal hemoglobinuria (PNH), and did not reveal any PNH clones in granulocytes or monocytes. The cluster of differentiation (CD) 15 and CD 24 in granulocytes, CD 14 and CD 64 in monocytes, CD 59 and GpA in erytrocytes, and CD 45 were all found to be 100%.

Coronal contrast enhanced CT obtained three and a half months after initial CT demonstrated venous collaterals (Figure **3**) at portal hilus consistent with cavernous transformation. A follow-up thorax CT scan 3.8 months postsplenectomy showed the resolution of

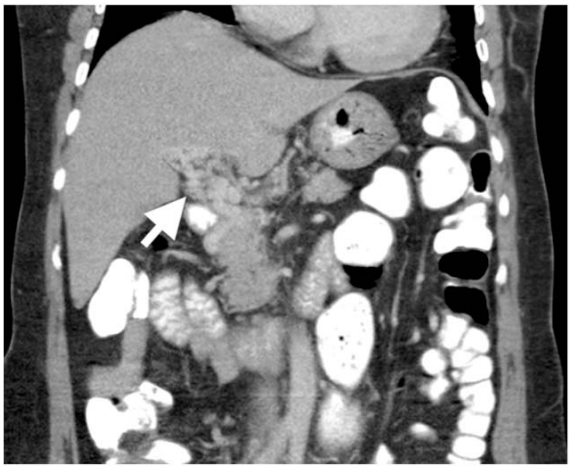

Figure 3. Recanalized portal vein. Coronal contrast enhanced CT obtained three and a half months after initial CT demonstrates venous collaterals (arrow) at portal hilus consistent with cavernous transformation.

the pulmonary emboli (Figure **4**). She is currently well with LMWH. To date, the patient remains clinically stable and asymptomatic.

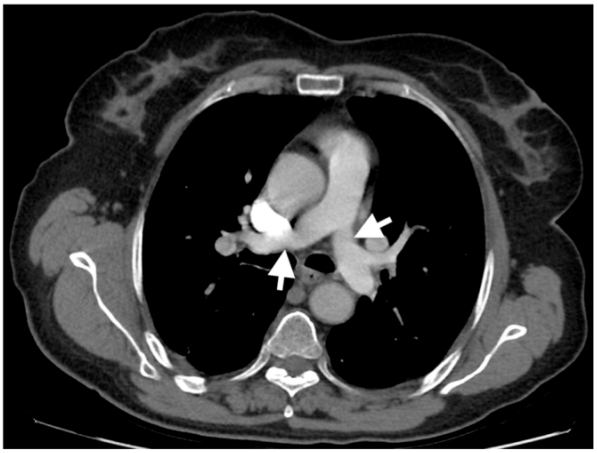

Figure 4. Contrast enhanced axial CT of patient after anticoagulant treatment demonstrates bilateral contrast material filled pulmonary arteries (arrows). No thrombus was observed in the pulmonary arteries.

DISCUSSION

The pathogenesis of thromboembolism after splenectomy is poorly understood. In several reports, thrombocytosis, large spleen size, and a myeloproliferative disease (MPD) as the underlying disease were described as risk factors [6, 7]. Patients with a large spleen (>3,000 g) and MPD had a 75% incidence of portal vein thrombosis (PVT) [6].

Postoperative PVT was more likely to occur in patients with hematological diseases (2% vs 10%) in a large Dutch study [8]. Postsplenectomy thrombocytosis in myeloid metaplasia patients was associated with postoperative thrombosis [9]. However, the association of postsplenectomy thrombocytosis and PVT is unclear, particularly in patients without MPD [6]. Although quantitative changes in platelet levels have been shown not to correlate with the development of postsplenectomy splanchnic venous thrombosis [10], qualitative changes in platelet function have been suggested to play a role in thrombotic tendencies [11].

In this patient, laparoscopic surgery, postoperative period and lack of anticoagulant prophylaxis after surgery seem to be probable responsibles of the thromboembolic state. We thought that, consumption of platelets in the massive theromboembolism sites including portal, mesenteric and pulmoner vascular beds had resulted in deep thrombocytopenia, which improved gradually with anticoagulant therapy.

Portal vein thrombosis may result from local factors, such as intraoperative manipulation of visceral vessels, the splenic vein in particular [12], and may account for the particular risk in patients with massively enlarged organs. For this reason, appropriate surgical technique for splenectomy should also be carefully considered in patients with hematological diseases.

Laparoscopic splenectomy has become an important option in early 1990s [13-21]; because it offers the advantages of minimally invasive surgery to these anemic, trombocytopenic or immune suppressed patients. There are many reports in the literature, which compare open and laparoscopic splenectomy [22-28]. They show comparable mortality and lower morbidity with longer operation time, shorter duration of hospitalization, lesser postoperative complications, greater patient comfort and earlier return to normal daily life with laparoscopic procedure. Ozdemir *et al*. performed laparoscopic splenectomy for ten patients with hematological diseases and found it safe and efficaeus in these patients. Postoperative PVT was reported in one patient (10%) with thalassemia minor. She received anticoagulation therapy for 3 months, and she is still using acetylsalicilic acid [29]. Similarly, Harris *et al*. reported portal vein thrombosis after laparoscopic splenectomy in 2 (14%) of 14 patients [30]. Both patients had ITP. Preoperatively, all patients received a subcutaneous injection of heparin. These two studies suggest that, the risk of PVT after laparoscopic splenectomy is at least as for open splenectomy. However, in a retrospective analysis by Svensson *et al*., PVT was diagnosed in five of 37 (13.5%) patients with hematological malignancies, four after open splenectomy and only one after laparoscopic splenectomy. The authors recommended careful observation and prolonged thromboprophylaxis for these patients that seem to have a high risk of developing PVT after splenectomy [31].

The elevated intraabdominal pressure associated with the creation of the pneumoperitoneum necessary for a laparoscopic procedure can lead to blood stasis in the portal system. Normal portal venous pressure is in the range of 10–12 mmHg. Significant hemodynamic changes have not been seen, however, when a pneumoperitoneum of ≤12 mmHg is employed [32].

Hypercoagulability also places the patient at high risk for PVT. However, there exists little data on activation of plasmatic coagulation and/or lack of coagulation inhibitors, such as protein C, S, or antithrombin III deficiency after splenectomy or the

presence of the factor V Leiden mutation or antiphospholipid antibodies. Few reports demonstrated a systemic hypercoaguable state in patients with PVT [12, 33]. Our patient had heterozygous MTHFR A1298C, PAİ-I 4G/5G and β-fibrinogen gene 455 G>A mutations. Folic acid replacement two days a week was started.

Mesenteric thrombosis, as was also seen in our patient, is associated with a poor prognosis due to subsequent bowel ischemia and multiorgan failure [6, 33, 34]. In cases of acute mesenteric thrombosis, most authors agree that prompt intravenous anticoagulation is warranted [35-37]. Early anticoagulation promotes recanalization of the portal vein and reduces the risk of splanchnic venous infarction without increasing the risk of variceal bleeding [38, 39]. The probability of recanalization is related to the extent of thrombosis [39]. Our patient suffered from both superior and inferior mesenteric vein thrombosis which could be resulted with bowel ischaemia and massive gastrointestinal bleeding. At the follow-up, under anticoagulant therapy, a gastrointestinal bleeding occurred and controlled after withdrawal of LMWH.

Acute abdomen caused by thrombosis of the portal and superior mesenteric veins in a splenectomized female ITP patient was reported by Spanish investigators. They performed local fibrinolysis successfully through a percutaneous transhepatic catheter [40]. More recently, 2 patients who underwent splenectomy for ITP developed portal and mesenteric vein thromboses. Both were treated successfully with anticoagulants for 3 months until the thrombi regressed, as shown by CT scan [41].

Portal vein thrombosis is usually diagnosed in symptomatic patients. Because screening for clinically occult portal vein thrombosis is not routine, it has been suggested that its true incidence is underestimated [42-44]. The recommendation to perform routine screening remains controversial [45]. Petit and colleagues [44] suggested that screening 2 weeks postoperatively may be preferable. Currently, color-flow Doppler ultrasound and contrast-enhanced CT scans are the best methods for the nonoperative diagnosis of PVT. Doppler ultrasound has a higher sensitivity than CT and is relatively inexpensive to perform [44]. However, CT evaluation may be more accurate during the 1st postoperative week because bowel distention and ileus may limit ultrasound examination. The flexibility of Doppler ultrasound in allowing the visualization of vessels along their long axes is a clear advantage [46].

Venous thromboembolism occurs in approximately 10% of splenectomized patients, and prophylactic anticoagulation is warranted in patients undergoing splenectomy for hematologic diseases [3]. The most appropriate duration of anticoagulant therapy is unknown, but some authors have demonstrated success with oral anticoagulation for 3 months, [47] and a general guideline of 6 months to 1 year has also been suggested [48]. To the best of our knowledge, duration and intensity of mandatory anticoagulation has yet to be established.

It was felt that ITP patients with a bleeding diathesis were likely at low risk of thrombosis. However, this assumption is likely not valid for hematologic malignant conditions. A retrospective analysis of 49 patients underwent splenectomy for primary/relapsing refractory ITP were analyzed in the absence of anticoagulant prophylaxis. The VTE rates of laparoscopic and open surgery were similar (9.5 vs 10.7%), and patients at risk were those who have had an exponential rise of the platelet counts [49]. More recently, among 9976 patients with ITP, 1762 of whom underwent splenectomy, the cumulative incidence of abdominal VTE was 1.6% compared with 1% in patients who did not undergo splenectomy. As expected, the increased risk of abdominal VTE was more pronounced <90 days after splenectomy, but not late [50]. These 2 studies had clearly stated that, postsplenectomy, ITP should be considered as a thrombophilic condition.

CONCLUSIONS

Splenectomy for hematological disorders (even with low platelet counts), and laparoscopic splenectomy are associated with increased risk of venous thrombosis, especially for portal and mesenteric vein thrombosis. Anticoagulation with LMWH for an extented period may be a beneficial prophylaxis regimen in patients undergoing general surcical procedures. Routine surveillance for portal vein thrombosis may be advisable after laparoscopic splenectomy, especially in patients with additional risk factors for thromboembolic disease. Prompt diagnosis and mandatory therapy should be based on each patient's clinical circumstances and the abilities of individual institutions.

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