**Scholars Journal of Medical Case Reports (SJMCR)** **ISSN 2347-6559 (Online)**
***Abbreviated Key Title: Sch. J. Med. Case Rep.*** **ISSN 2347-9507 (Print)**

A United of Scholars Academic and Scientific Society, India

# Pulmonary Embolism after Splenectomy in Chronic Immune Thrombocytopenic Purpura Patient

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**Article History**
*Received: 23.02.2018*
*Accepted: 07.03.2018*
*Published:30.03.2018*

**DOI:**
10.36347/sjmcr.2018.v06i03.003

**Abstract:** Chronic Immune Thrombocytopenic Purpura (ITP) is a disease characterized by thrombocytopenia. Thrombocytes become antigenic and being disrupted by the reticuloendothelial system. Splenectomy is performed in patients with ITP who do not respond to medical treatment. Our patient had splenectomy and pulmonary emboli developed afterwards. After splenectomy in ITP patients; it should not be forgotten that thrombocytopenic patients may develop thrombotic complications that could pose a life threatening effect.
**Keywords:** Immune thrombocytopenic purpura, splenectomy, pulmonary embolism.

## INTRODUCTION

Chronic Immune Thrombocytopenic Purpura (ITP) is a disease characterized by thrombocytopenia, in which IgG-binding thrombocytes become antigenic and are cleaved by the reticuloendothelial system (RES), particularly via the spleen. Splenectomy is performed in patients with ITP who do not respond to medical treatment and the risk of thrombosis after this operation is remarkable [1,2].

Pulmonary embolism (PE) is a life-threatening emergent that can cause severe right ventricular failure due to frequent and acute pulmonary artery occlusion. Deep venous thrombosis (DVT) is the first cause [3].

In our case, splenectomy was performed because of failure to respond to medical treatment, and PE developed later. Although thrombotic complications have been observed after splenectomy in cases with ITP, no case of PE has been reported previously in the literature.

## CASE REPORT

Our case was a 57 years old female patient who had rashes on her legs and had complaints of easy bruising about 9 months ago and admitted to our clinic with these complaints. Her labaratory findings were as follows; Hb: 12.7 g/ dl, Htc: 38.5 %, MCV: 88 fl, WBC: 5470/ $mm^3$ and platelet (PLT) count was 35.000/ $mm^3$ and was compatible with thrombocytopenia. Physical examination revealed no pathology other than ecchymosis in the legs. Ultrasonographic examination was normal. Hepatitis markers, rheumatological parameters, bacterial and viral causes thrombocytopenia (TORCH, EBV, Herpes, etc.) were investigated to exclude secondary causes of thrombocytopenia. Bone marrow biopsy result was also normal. Treatment with prednisolone at a dose of 1 mg/ kg for 4 weeks was reduced gradually and discontinued at the end of 2 months. After 1 month of treatment, the platelet count was increased to 117.000/ $mm^3$ in the complete blood count (CBC), however after the cessation of therapy platelet count decreased to 4.000/ $mm^3$ levels. Because of the sudden drops in platelet count, the patient was given thrombocyte suspension and prednisolone therapy was started again. The patient underwent steroid treatment for 6 months and PLT values followed between 30.000 to 110.000/ $mm^3$. The patient was diagnosed with chronic ITP and treated with 1g/ kg intravenous immunglobuline treatment for 1 month; however, there was no significant increase in PLT values and splenectomy was executed. 16 days after splenectomy operation, she admitted to the emergency department with shortness of breath, abdominal pain and swelling on the feet. In abdominal tomography; intrahepatic portal vein, splenic vein, main portal vein, superior and inferior mesenteric veins were all obliterated with thrombus. In thorax tomography; filling defects were observed in bilateral main pulmonary arteries, pulmonary lobe arteries and some segment branches. In doppler ultrasonography of lower

extremities, the appearance of thrombus in both leg's popliteal vein was observed. Low dose (0.4 cc) enoxaparin sodium was given once daily as a treatment because of the the risk of bleeding and PLT value was 26.000/ $mm^3$. Because of widespread thrombi in main body veins despite low platelet count after splenectomy; patients was investigated in terms of bleeding disorders such as Paroxysmal Nocturnal Haemoglobinuria and inherited thrombophilia disorders (factor II, Factor V G1691A, MTHFR C677T, MTHFR A1298C, beta fibrin 455 G> A, Factor XIII V34L, GPIIb L33P ), and the results were normal. The patient was followed up for 2 weeks with enoxaparin sodium therapy with daily CBC values. At the end of the 2-week period, PLT value increased to 143.000/ $mm^3$.

## DISCUSSION

Chronic Immune Thrombocytopenic Purpura (ITP) is a disease characterized by thrombocytopenia. In this disease, IgG-binding thrombocytes become antigenic and are cleaved by the reticuloendothelial system (RES), particularly via the spleen. The factor that triggers the formation of antibodies in the body is stil unclear [4-6]. These antibodies (usually IgG, sometimes IgM or A) bind to the platelet surface antigen GPIIb-IIIa, and are cleared rapidly from the circulation by macrophage and dendritic cells in the liver and spleen by RES. These antibodies not only trigger platelet disruption, but also inhibit megakaryocyte maturation which plays a key role in platelet production and by this way accelerate PLT apoptosis. ITP is usually diagnosed by excluding the other causes of thrombocytopenia [7-9]. Clinical findings in ITP are compatible with the platelet count and usually occur with petechiae, purpura, nasal and gingival bleeding. Drug use, HIV infection, hepatitis C virus infection, lymphoproliferative diseases, systemic lupus erythematosus and autoimmune diseases are frequent causes of thrombocytopenia [9-13].

1 mg/ kg/ day prednisone therapy is recommended for 4 to 6 weeks in ITP patientsis and dose is gradually reduced [14]. After discontinuation of steroid therapy, only 10% to 30% of patients remain in remission and treatment can not be extended due to steroid side effects such as infection, osteoporosis, emotional disorder [15,16]. Intra venous immunoglobulin is another treatment option in patients who do not respond to steroid treatment and rapidly increases the platelet count in 65-80% of patients [17].

Currently, laparoscopic splenectomy treatment is applied in cases who do not respond medical treatment, and after this operation, about 10% complication develops and the increased risk of thrombosis is noteworthy [1,2].

Pulmonary embolism (PE) is a life-threatening emergent with acute pulmonary artery occlusion that can cause severe right ventricular failure. The incidence of PE was found to be 70 in 100.000 on average [18] and deep vein thrombosis (DVT) is the first of causes in order 33% (3). When investigating the causes of PE, the etiologic factors related to the patient such as cancer, neurological diseases etc. should be considered and the important causes of thrombophilia should not be overlooked (19). Early treatment in PE cases is life-saving and reinstating the flow of pulmonary artery is the priority [18]. Current treatment of PE has been described as low molecular weight heparin, fondaparinux, intravenous or subcutaneous unfractionated heparin in the guideline of the American College of Chest Physicians [20-22].

We report a patient with PE and splenic, mesenteric vein thrombosis on the 16th day after laparoscopic splenectomy, whose thrombocytopenia improved under close follow-up with anticoagulant therapy in our clinic. Despite publications about portal vein thrombosis in patients with ITP after splenectomy in the literature, patient with DVT, PE, and widespread thrombosis of intraabdominal organs in the postoperative thrombocytopenic state was remarkable as a rare case.

## CONCLUSION

In conclusion, after splenectomy in chronic ITP patients; we think that complications such as life threatening PE, splenic thrombosis and mesenteric venous thrombosis may develop, even if the patient is thrombocytopenic, and we think that it will be beneficial to start anticoagulant treatment when necessary.

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