

H. pylori and Alcohol-induced Immune Thrombocytopenic Purpura: A Case Report

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ABSTRACT

Immune thrombocytopenic purpura is a rare autoimmune disorder. ITP is a multicausal disease that can be mainly caused by infection (*H. pylori*, viral infection), excessive alcohol consumption, drugs like antibiotics, diuretics and can also be inherited genetically. In this case, a 40-year-old male came to the tertiary care hospital with complaints of high-grade fever, upper respiratory tract infection symptoms generalized weakness, and headache. On examination, petechial rashes over the abdomen were observed. Symptoms and blood reports were found to be similar with immune thrombocytopenic purpura. The patient was diagnosed with anemia of infection and inflammation. Immune thrombocytopenic purpura is not curable and only symptomatic treatment can be provided. This case report layout an insight of ITP, pathophysiology, and pharmacotherapy in

accordance with the hospital guidelines. Early detection of ITP will assist with further complications that are related to ITP.

Keywords: Immune thrombocytopenic purpura, Infection, Antibiotics, Diuretics, Anemia of infection and inflammation.

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INTRODUCTION

Immune thrombocytopenic purpura is a rare autoimmune bleeding disorder, in which platelets are destroyed by own immune system.¹ The disease can occur in both children and adults. Thrombocytopenia means decreased platelets count in blood and purpura indicates purple discoloring of the skin. ITP has mainly divided into two types which the first one is acute thrombocytopenic purpura (ATP) and the second one is chronic thrombocytopenic purpura (CTP), In which ATP mainly affects young children (age- 2 to 6 years old) and CTP mainly affects at any age.² There are many causes of ITP such as OTC medication which directly cross-reacts with platelets, infection (*H. pylori*, viral infection), excessive alcohol consumption, immune disorders such as rheumatoid arthritis (RA). Symptoms of ITP are easy or excessive bruising, bleeding from the gums and nasal bleeding, blood in urine or stool (haematuria, haematochezia).³ Epidemiology of ITP is reported as an incidence of 5.8/100,000 cases are reported whereas, the prevalence of 4.6/100,000 cases is reported in Europe. In North America, a slightly higher prevalence of 7.2/100,000 cases is reported in children aged (1-14 years). The annual incidence of 1.6/100,000 cases is reported in adults globally, higher in middle age with a prevalence rate of 1.9 for females to males. In India, 6.4/100,000 and 3.3/100,000 cases are reported in children and adults respectively annually. Seasonally, ITP shows a higher peak of incidence in winter and spring whereas, ITP can be also caused by vaccinations in a few cases (<8%).⁴⁻⁸ Diagnosis of ITP is CBC which serves the purpose to check the platelet levels in the blood. The patient's medical history, symptoms, physical examination, and laboratory tests are also taken into consideration by the doctor.³ The treatment regimen varies on basis of age, health condition, patient medical history, sensitivity and resistance to specific medications, the extent of disease, and tolerance for medical procedure of the patient. The treatment of ITP is corticosteroids, antibiotics, immunoglobulin, immunosuppressive agents.⁹

PATHOPHYSIOLOGY

The figure 1 represents schematic development of ITP and the figure shows, Platelet-associated autoantigen (solid-rectangle attached to platelet) is formed. As the platelet is intravascular, the initial response of the immune system occurs in the spleen and bone marrow.

After which, antigen-specific memory cells develop which, begin to circulate and initiate a more generalized immune response.



IgG antiplatelet antibodies are produced and cytotoxic T lymphocytes are produced.



Then, autoantibody binds to platelets which causes platelet destruction by the process of phagocytosis of complement(C) induced lysis.



Megakaryocytes result in decreased maturation and lead to cell death.



Cytotoxic T lymphocyte results in platelet and megakaryocytes lysis

Case Description

A 40-year-old male came to the tertiary care hospital with no significant past medical history. The patient experienced upper respiratory infection symptoms like chest pain, cough, and breathlessness on walking a distance of 200 meters along with high-grade fever and chills, nose bleed, back pain, for 5 days. The patient is a chronic alcoholic for 10 to 15 years.

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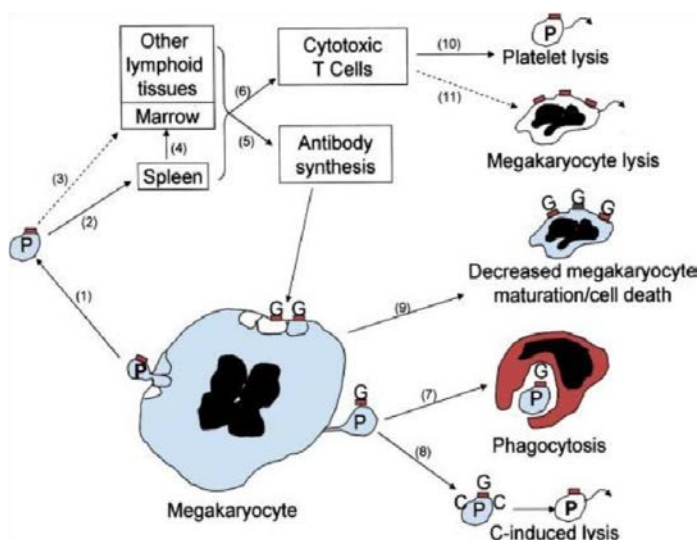


Figure 1: Pathophysiology of ITP.¹⁰

The patient has a history of blood transfusion. He was not taking any home medication. His family history was not significant. His vital signs were stable. On physical examination, it was notable that pinpoint-sized reddish-purple spots generally looked like a rash (petechial rashes) over the abdomen. He was found to have a platelet count of $0.4 \times 10^9/L$ on the day of admission which was lower to $0.3 \times 10^9/L$ on the next day the normal value of the platelet is $1.5-4.5 \times 10^9/L$. TSH, Hepatitis C antibody, HIV antibody, a sickling test, DCT, and dengue reports were all negative but *H. Pylori* stool antigen test was positive. The complete blood count report sighted low Haemoglobin, packed cell volume, mean cell volume, mean cell hemoglobin concentration, red blood cell, serum iron, ferritin, total iron-binding capacity. The USG abdomen and pelvis test confirmed that the patient was suffering from fatty liver and thickened gall bladder. He was diagnosed with o ITP which seems to be associated with *H. pylori* infection and long-term alcohol consumption.

After admission in male medicine, the ward patient was immediately started on Inj.Paracetamol (Inj. Febrinil) 1 amp q12h. On day 2 it was confirmed that patient had a positive *H. pylori* antigen test along with low platelet count for which steroid and antibiotic were initiated. The physician advised taking plenty of oral fluids and green leafy vegetables. After 5 days of treatment, the condition of the patient was improved and platelet count was stable. After which patient was discharged with the following medicine as follow tablet ciprofloxacin (Tab. Cipro) 500mg 1-0-1 for 5 days, tablet domperidone+pantoprazole(Tab. Dan P) 1-0-0 for 10 days, tablet paracetamol(Tab. Dolo) 650 mg 1-0-1 for 3 days, tablet folic acid-pyridoxine hydrochloride-methylcobalamin(Tab. FDSON MP forte), capsule cyanocobalamin-ferrous fumarate-folic acid (Cap. Autrin), tablet vitamin-C (Tab. Limcee) for 15 days were given as discharge medication to the patient. Follow-up was advised after 15 days along with a blood culture report.

DISCUSSION

H. pylori is a gram-negative bacteria that contributes as a main cause of ITP. Some observations show a positive association between *H. pylori* infection and ITP and that eradication of the bacterium was accompanied by a significant increase in platelet count in most cases were made by Gasbarrini *et al.* in 1998.¹¹ Analyzing platelet response after bacterial eradication shows that the majority of patients in whom *H. pylori* infection was eradicated had a platelet increase. Patients

who became *H. pylori* negative obtained a complete or partial platelet response. Another finding that confirms the etiologic role of *H. pylori* in ITP patients is that, in the majority of studies reported, platelet count did not differ before and after treatment in those patients in whom bacteria was not eradicated.¹² Alcohol is an important triggering factor in the causation of ITP. Analysis of occurrence of ITP in chronic alcohol drinkers was studied where it was interpreted that alcohol acts as a factor that suppresses the formation of morphotic components of blood. ITP caused due to excessive alcohol intake usually showed normal platelet ranges once excess consumption of alcohol was halted. The pathological mechanism involved in the correlation of excessive alcohol consumption and ITP is that alcohol directly affects thrombocytes production and it also accelerates degradation and apoptosis of thrombocytes.¹³

CONCLUSION

In this case report, we concluded that the eradication of *H. pylori* shows a beneficial effect on platelet count in adults suffering from ITP that are recently diagnosed. The occurrence of ITP is associated with *H. pylori* infection present within the patient. The pathogenesis of ITP associated with *H. pylori* is not well defined. Presenting pathogenic mechanism involves cross-reactivity between platelet-associated immunoglobulin and *H. pylori* Cag-A protein. Further, we would conclude that excessive alcohol consumption plays a triggering role in ITP due to alcohol's platelet lowering capabilities. Long-term alcohol consumption affects the functioning efficacy of liver and bone marrow that leads to bone marrow suppression, defective platelet formation, decreased platelet lifespan, and impaired platelet function. Early detection of ITP will assist with further consequences that are related to ITP.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

ITP: Immune Thrombocytopenic Purpura; **ATP:** Acute Thrombocytopenic Purpura; **CTP:** Chronic Thrombocytopenic Purpura; **OTC:** Over The Counter; **IV:** Intravenous; **TSH:** Thyroid Stimulating Hormone; **HIV:** Human Immunodeficiency virus; **DCT:** Direct Coombs test; **q12h:** every 12 hr.

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