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# RARE CASE OF HASHIMOTO THYROIDITIS ASSOCIATED WITH IMMUNE THROMBOCYTOPENIC PURPURA

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**ABSTRACT** Immune thrombocytopenic purpura (ITP) is a disorder characterized by a platelet count of less than 1.5 lakhs per cubic millimeter in the absence of other causes of thrombocytopenia, such as viral infections, rheumatic diseases, or drugs. ITP is one of the most common cause of thrombocytopenia and is a diagnosis of exclusion when no other etiology is present. ITP may rarely coexist with autoimmune thyroid disorders. However, ITP may be difficult to treat when associated with thyroid autoimmune disorders. In such cases, treating the underlying thyroid disorder may significantly improve platelet count and may either cause remission of disease or improve response to standard ITP therapy. Here we report a case of 37 year woman who was diagnosed as Hashimoto's thyroiditis and ITP. Treatment of hypothyroidism with levothyroxine and steroid therapy for short duration in our patient improved the platelet count significantly.

**KEYWORDS :** Hashimoto Thyroiditis, thrombocytopenia, ITP, hypothyroidism

## INTRODUCTION

Immune thrombocytopenia purpura (ITP) is an autoimmune disorder which is characterized by immune destruction of platelets leading to low platelet counts.[1] Majority of cases of ITP are idiopathic with no underlying cause, termed as primary ITP. Secondary ITP is caused by a variety of conditions like Hepatitis C virus, Human immunodeficiency virus (HIV), Systemic lupus erythematosus (SLE), certain drugs (eg: Quinine, Quinidine) and some malignancies (eg: breast cancer). Other causes of thrombocytopenia should be ruled out before diagnosing a patient with ITP as management strategy varies with etiology of thrombocytopenia. Clinical indicators of ITP include easy bruising of skin, petechiae, ecchymoses, epistaxis and other bleeding manifestations.[2] Prognosis of ITP is determined by low platelet count and the risk of spontaneous hemorrhage. Patients with ITP can be asymptomatic or present with life threatening spontaneous bleeding. Recent studies have shown the association of autoimmune thyroid disease with ITP and treatment of autoimmune thyroid disease improves the platelet count and overall outcome of ITP.[3] We report a case of 37 year old female who was admitted with severe ITP and was found to have subclinical Hashimoto's thyroiditis. Treatment of subclinical hypothyroidism with levothyroxine and oral steroid improved the platelet count significantly in our patient.

## CASE REPORT

A 37 year old woman married for 17 years presented to general medicine outpatient department at KVG Medical College, Sullia with history of regular menstrual cycles with menorrhagia but without dysmenorrhea for 1 year. She did not have mucocutaneous bleeding tendencies. There were no cardiac or respiratory symptoms. She was non-diabetic and non-hypertensive with no extra-dietary addictive habits. She had two uneventful pregnancies with normal vaginal delivery. She had a clear sclera and pink palpebral conjunctiva and was normothermic, acyanotic and normotensive (blood pressure = 120/70 mm of Hg). She had a normovolumic, regular pulse of 96 bpm and she was comfortably breathing with respiratory rate of 18 cycles of breaths per minute and had no signs of heart failure. She was well nourished (BMI = 26.6 kg per m2). She had no goiter and had no clinical signs of hypothyroidism except for dry and coarse skin. Cardiovascular, respiratory and neurological physical examination was unremarkable.

Hematologic investigations revealed platelet count of 26,000 per cubic millimeter with presence of occasional giant cells on peripheral smear and Platelet distribution width of 16.7. Red blood cell indices were within normal limits with erythrocyte sedimentation rate (ESR) of 6 mm at one hour. Leucocytes were normal in morphology and number. Direct and indirect Coomb's test was negative. Coagulation profile was within normal limits. Screening for chronic malaria by Quantitative Buffy Coat (QBC) method was negative. Anti-Nuclear Antibody (ANA) test was negative. There was no consumption of drugs causing thrombocytopenia.

Thyroid hormone study revealed features of primary hypothyroidism. The laboratory results are as follows:

| | Result | Normal values |
|---|---|---|
| Thyroid stimulating hormone (TSH) in milliunits per liter (mU/L) | 11.35 | 0.25-5 |
| Free (unbound) T3 (fT3) in picomoles per liter (pmol/L) | 3.91 | 4.6-9.7 |
| Free (unbound) T4 (fT4) in picomoles per liter (pmol/L) | 8.8 | 12-30 |
| Anti TPO antibody (IU/ml) | 249.4 | Upto 34 |

Ultrasound scan of thyroid gland revealed features of Hashimoto's thyroiditis. On enquiry, she did not give history suggestive of hypothyroidism except fatiguability.

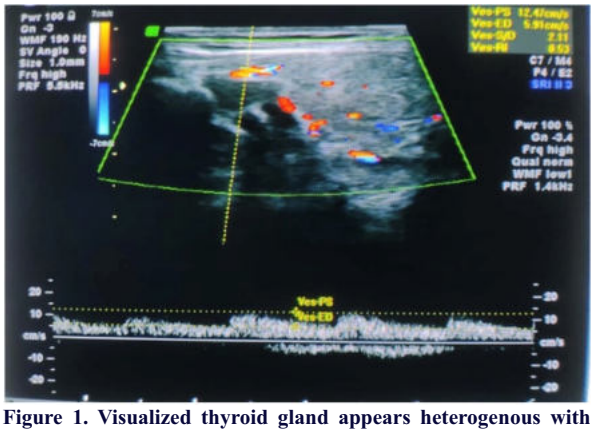

**Figure 1. Visualized thyroid gland appears heterogenous with multiple small hypoechoic nodules seen. There is mild increase in the vascularity.**

Renal function tests, 12 lead electrocardiogram and chest x-ray were within normal limits. Ultrasonographic imaging of abdomen was unremarkable except for a small intramural fibroid in the uterus.

In view of possibility of ITP, intra-venous steroid (Dexamethasone) was started at a dose of 4mg 8th hourly and platelet count improved to 77,000 cells per cubic millimeter on day four. Patient was initiated on oral steroid (prednisolone 40mg per day) from day six. Platelet count improved to 2,31,000 per cubic millimeter over 10 days. Patients was also started on levothyroxine supplementation (100 mcg once daily equivalent to 1.6 mcg per kg body weight) and subsequently followed up after 1 month. Steroid was tapered and stopped after 3 months. Currently patient has a well controlled TSH (TSH level: 4.98 milliunits per liter (mU/L)) on 100 mcg of levothyroxine and off steroid therapy since 3 months.

In-spite of not being on steroid therapy, platelet count has been over 1.5 lakh per cu mm. We can presume that the platelet count is maintained at normal level after achieving euthyroid state. Hence, we propose that the improvement of hypothyroidism may improve platelet count in patients with ITP and this may establish an association between autoimmune hypothyroidism and ITP.

## DISCUSSION

Immune thrombocytopenic purpura (ITP) is an acquired autoimmune disorder characterized by immune mediated destruction of platelets and possibly inhibition of platelet release from the megakaryocyte. ITP can be classified as primary ITP which is idiopathic and secondary ITP which is caused by variety of conditions like viruses, autoimmune diseases, drugs, infections and malignancies. In adults, it is a more chronic disease, and in some patients spontaneous remission occurs usually within few months of diagnosis. Literature data shows that autoimmune thyroiditis is one of the most commonly diagnosed immune disorders in ITP patients.[4]

The combination of autoimmune thyroid disease and ITP will reflect a significant defect in the immune self-tolerance of these patients compared with those who have primary ITP alone.[5] Such immune defects are refractory to standard ITP therapy. Screening patients for hypothyroidism with thyroid stimulating hormone assay helps us to detect subclinical thyroid disease.

Autoimmune thyroid diseases and ITP may present simultaneously, although the time between the onset of both the diseases vary from months to years. Hashimoto's thyroiditis is the most common thyroid disorder, however the association between ITP and other autoimmune diseases has been described in a small number of studies.[6]

Clinical presentation of patients with ITP vary from asymptomatic disease to life threatening spontaneous bleeding. Mild bleeding manifestations like petechiae and purpura are usually common, but life threatening complications like hemorrhage is very rare and is usually seen with severe thrombocytopenia with platelet count of less than 10,000 to 20,000 cells per cu mm. In patients with no risk of bleeding manifestations, literature recommends an observe and wait approach.[7] Prognosis of ITP is determined by low platelet count and the risk of spontaneous hemorrhage.

For diagnosis of ITP, antiplatelet antibody testing is not routinely recommended by American Society of Hematology due to its low sensitivity and specificity. After diagnosing ITP, other secondary causes of ITP should be ruled out and peripheral blood smear, HCV and HIV testing should be done.[8]

ITP should be treated only if there is significant bleeding or risk of bleeding with platelet transfusions, IVIG and glucocorticoids.[9] However ITP specific therapy is recommended in patients with platelet count less than 30,000 cells per cu mm without significant bleeding.[10] In patients with ITP and hypothyroidism, levothyroxine supplementation improves the platelet count significantly as in our patient.

Here we present a case of 37 year old female who presented with history of menorrhagia of 1 year duration. On evaluation, she was found to have thrombocytopenia of unknown etiology and primary hypothyroidism (Hashimoto's thyroiditis). Platelet count improved with steroids which was gradually tapered and stopped. The platelet count is sustained at normal level with levothyroxine alone with achievement of euthyroid state, suggesting a possible association between autoimmune hypothyroidism and ITP.

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