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IMMUNE THROMBOCYTOPENIA (ITP) SECONDARY TO OVERT HASHIMOTO'S THYROIDITIS :ROLE OF STEROIDS AND LEVOTHYROXINE IN IMPROVING CLINICAL OUTCOME

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ABSTRACT ITP is a disorder of immune mediated destruction of thrombocytes leading to platelet count <1,50,000 in the absence of other causes of thrombocytopenia such as viral infections, drugs. ITP is one of the most common cause of thrombocytopenia and it 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 establish a remission or improve response to ITP standard therapy. Here we report a case of a 30 year old female diagnosed with Immune thrombocytopenia. When her evaluation was not in favour of primary ITP, she was screened and proved to have a coexisting Hashimoto's thyroiditis. Our patient has uncontrolled hypothyroidism as she is not compliant to medication. Thrombocytopenia improved with steroids and levothyroxine for 4 weeks.

KEYWORDS :

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 haemorrhage. 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 30 year old female who was admitted with severe ITP and was found to have coexisting Hashimoto's thyroiditis. Treatment of hypothyroidism with levothyroxine and oral steroid improved the platelet count significantly in our patient.

CASE REPORT

30yr old female married for 10 years presented to emergency department with complaints of generalised weakness, giddiness. she denied history of fever, rash, heavy/excessive menstrual bleeding, sore throat, joint pain and swelling. There were no cardiac or respiratory symptoms. past medical history was significant for hypothyroidism since 2yrs for which she was on thyroid hormone replacement (levothyroxine 50 mcg) non complaint. she had history of thrombocytopenia and one unit of blood transfusion during her first pregnancy. She was non-diabetic and non-hypertensive with no extra-dietary addictive habits. Family history is insignificant for hypothyroidism.

On examination vitals were stable. patient is moderately built and well nourished but there were no signs of icterus, pallor, clubbing, pedal edema, lymphadenopathy or hepatosplenomegaly. she had no goiter and no clincal signs of hypothyroidism except for dry and coarse skin. Rest of the physicalexamination was unremarkable. Hematologic investigations revealed platelet count of 9,000 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. Hepatitis panel, and HIV screening test were negative There was no consumption of drugs causing thrombocytopenia. Thyroid antibodies were also ordered to screen for concurrent autoimmune thyroid disease in ITP, which came back positive for anti-TPO antibodies (462 IU/mL). Thyroid-stimulating hormone (TSH) was done subsequently, which was higher normal (>100 μIU/mL), and total T4 and T3 were decreased. Isolated thrombocytopenia and normal peripheral blood film in the presence of unremarkable physical examination led to the presumptive diagnosis of ITP. As platelet counts were low (9000/μL), it was considered a medical emergency and the patient was treated immediately with ITP standard therapy, that is steroids. Patient was started on 1mg/kg body weight oral prednisolone and oral levothyroxine 100 mcg followed up on week platelets increased to safe level but not normal, patient did not have bleeding manifestations during the course. Patient was kept on 40mg oral prednisolone and followed up after 4 weeks platelets were 2.15 lakhs and TSH is 12. Steroid was tapered and stopped after 3 months. Currently patient has a well controlled TSH (TSH level: 4.5 μIU/mL) on 100 mcg of levothyroxine and off steroid therapy since 3 months.

DISCUSSION

Immune thrombocytopenia is a destructive platelet disorder that can be classified as primary ITP, which is idiopathic in origin, or secondary ITP, due to variety of conditions like viruses, drugs, autoimmune disorders, infections, and malignancies. In ITP, there is autoimmune-mediated destruction of platelets directed against surface antigens,

resulting in opsonization and destruction of platelets by reticuloendothelial system, particularly in spleen[4].Both antibody-mediated destruction and suppressed platelet production leads to reduced platelet life span[5]. ITP is the most common cause of isolated thrombocytopenia, with majority of patients being asymptomatic. Mild variety bleed like petechiae and purpura are also common, but life-threatening haemorrhage is rare and is usually seen with platelet counts of less than 10 000 to 20 000. Contrary to other causes of thrombocytopenia, ITP is the diagnosis of exclusion. The most important diagnostic approach to ITP is excluding the important causes. Antiplatelet antibody testing for the diagnosis of ITP is not routinely recommended by the American Society of Haematology guidelines due to its low sensitivity and specificity and lack of correlation of antibodies with clinical outcomes[6].

After diagnosing ITP, every effort should be made to rule out any secondary cause of ITP as treatment of underlying cause may improve platelet count. All patients should have peripheral blood smear, HCV, and HIV testing. Addition testing like bone marrow biopsy, thyroid profile, and coagulation and immunological studies are reserved for selected patients only.

The patient may have features of hypothyroidism such as constipation, dry skin, weight gain, cold intolerance, and fatigue. However, in some patients, presentation may be subclinical, that is, without any symptoms and diagnosis is made by routine thyroid function testing. Hashimoto's disease is usually diagnosed by high TSH and elevated antithyroid peroxidase (anti-TPO) and/or anti thyroglobin (anti-TG) antibodies .

Literature data shows that autoimmune thyroiditis is one of the most commonly diagnosed immune disorders in ITP patients[7]. 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. Such immune defects are refractory to standard ITP therapy. Screening patientsfor hypothyroidism with thyroid stimulating hormone assay helps us to detect subclinical thyroid disease. ITP should be treated only if there is significant bleeding or risk of bleeding with platelet transfusions, IVIG and glucocorticoids. However ITP specific therapy is recommended in patients with platelet count less than 30,000 cells per cu mm without significant bleeding [8]. In patients with ITP and hypothyroidism, levothyroxine supplementation improves the platelet count significantly as in our patient.

Treatment goal for ITP is not to bring platelet counts to normal but to maintain the platelet count at a level that successfully prevents spontaneous bleeding. It has been reported that treatment of coexisting thyroid disease in ITP patients results in either remission of autoimmune thrombocytopenia or enhanced response to standard therapy of AITP.

Here we present a case of 30 year old female who presented with generalised weakness and giddiness since 2 months .on evaluation, she was found to have thrombocytopenia of unknown etiology and 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, possibly an association between autoimmune hypothyroidism and ITP.

CONCLUSION

Immune thrombocytopenia can rarely coexist with Hashimoto's thyroiditis. ITP in such cases might be refractory to standard first-line and second-line therapies due to much more significant defect in immune tolerance. Treating coexisting autoimmune disorder can improve the platelet count and therefore should be considered in such patients.

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