Case Report

Thyroid disease in patients with idiopathic thrombocytopenic purpura

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Abstract

Idiopathic thrombocytopenia (ITP) is an autoimmune disorder characterized by a reduced platelet count with otherwise normal blood cell counts. Autoimmune thyroid disease is not considered a cause of thrombocytopenia. We report a case of autoimmune thrombocytopenic purpura associated with hyperthyroidism in which the patient's thrombocytopenia and thyrotoxicosis resolved concomitantly. We recommend testing for hyperthyroidism in all patients with unexplained thrombocytopenia and that family members of patients be evaluated, screened, and observed for thrombocytopenia and hyperthyroidism.

Keywords: Idiopathic thrombocytopenic purpura, Hyperthyroidism, Autoimmune

Introduction

Thyrotoxicosis and thrombocytopenia both are common diseases. Previous case reports and small studies have shown that hyperthyroidism sometimes is present in patients with ITP. Autoimmune thrombocytopenic purpura is frequently referred to as idiopathic thrombocytopenic purpura.1 ITP has been shown to be associated with systemic lupus erythematosus and some other autoimmune diseases 2, but ITP presenting as symptom of hyperthyroidism or Graves’ disease is rarely encountered. The first case report of association of acute hemorrhagic purpura with exophthalmic goiter was reported by Jackson in 19213: following this there has been series of case reports published describing similar association. Here we report a case of Idiopathic thrombocytopenic purpura developed together with autoimmune thyroiditis and overt hyperthyroidism in a 26-yearold woman, probably first case report in Nepal.

Case report

A 26-year old young female, presented on medical OPD with bleeding from gums on and off for 3 days and bluish discoloration over some parts of body for 20 days. She was very well before this. She suddenly has bleeding from gums and nose along with petechiae over the body. There was no history of recent illness, drug ingestion. There was no significant family history and similar episodes in past. She doesn’t have classical thyrotoxicosis symptoms.

On examination, her BMI was 23.5/kgm2. She had a pallor, widespread petechiae and bruises all over the body and extremities. The spleen and liver was not palpable, her resting pulse rate was 84 per minute, regular and blood pressure was 130/80 mm of Hg. There was no fine tremors of both hands but palms was abnormally moist. Further examination showed diffusely enlarged thyroid gland without thyroid bruit. Her nervous system and mental status was normal. Investigations done included a complete blood count in which hemoglobin was 11.4% without leucocytosis, normal reticulocyte count and platelet count on admission was 10,000/mm3 only. Bone marrow aspirates showed megakaryocytic thrombocytopenia. Thyroid function tests confirmed the diagnosis of hyperthyroidism (serum FT3 =7.49; normal range / serum FT4 =4.46; normal range / TSH=0.16; normal range). Her Anti-thyroidperoxidase antibody was also positive. Ultrasound thyroid showed features suggestive of thyroiditis and her PT/ aPTT, antinuclear antibody, routine urine examination were normal.
The patient was started on pulse methylprednisolone at 1gm intravenously for 3 days followed by oral prednisolone 1mg/kg body weight and also platelet rich plasma transfusion around 6 pints and with this treatment her platelet count rose from 10,000 to 55,000 with hemoglobin of 13.2% at discharge. Carbimazole was started 5mg thrice daily with propanolol 40mg twice daily on laboratory confirmation of thyrotoxicosis. There was symptomatic improvement and the patient was discharged after 12 days of hospital admission. There was no clinical evidence of thrombocytopenia. Medication on discharge was prednisolone 40 mg once daily and carbimazole 5mg thrice daily. After two weeks on follow up in OPD she was symptomatically improved and her platelet count was 1.25 lakhs/mm$^3$ and so she was advised to gradually taper the dose of steroid and was continued on carbimazole.

**Discussion**

The association of thrombocytopenic purpura with certain endocrine disturbances is not uncommon. Marshall et al. reported that 4 of 42 patients with ITP had clinically overt hyperthyroidism, and that in 2 other patient’s laboratory tests showed evidence of hyperthyroidism. Since both conditions are thought to have an underlying auto-immune mechanism it is surprising that the incidence is not higher, as in systemic lupus erythematosus (SLE). The incidence of the combined diseases seems to be about equal between male and female. No case has occurred below the age of 28. Our patient was 26 year old female. This case specially highlights the incidental findings of autoimmune hyperthyroidism of in patients with ITP which probably has the same immunological cause and genetic predisposition of underlying autoimmune disease or there may be possible non immune mechanisms involved in the causation of thrombocytopenia during thyrotoxicosis and the need for testing thyroid dysfunction in cases of autoimmune diseases like idiopathic thrombocytopenic purpura (ITP), especially when response to conventional treatment is poor. Woodruff et al., reported platelet numbers to be significantly lower in thyrotoxic patients as compared with normal adults. Kurata et al. in 1980 produced evidence showing that 43% of patients with untreated hyperthyroidism had platelet counts less than 150,000/mm$^3$ but in our case platelet count was 10,000/m$^3$. So it seems thrombocytopenia is likely to occur in severely hyperthyroid patients but the prognosis being good owing to fact that megakaryocytes are present in the bone marrow. There are numerous case series that have shown normal or increased number of megakaryocytes in the bone marrow in hyperthyroidism, independent the number of platelets. Majority of patients with ITP improve when the thyroid disease is under control by medical, surgical or radio-iodine ablation. I think in this particular case thrombocytopenia presented after the onset of thyroid disease and the patient was treated with anti thyroid medications as compared to Radio Iodine ablation in the case described by S Kannan et al.

**Conclusion**

In conclusion I would like to suggest early evaluation of thyroid function test is needed in every patient with unexplained thrombocytopenia or thrombocytopenia refractory to usual treatment.

**References:**