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ORIGINAL ARTICLE

Unusual Hematological Manifestations Associated with Primary Hypothyroidism in an Elderly

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ABSTRACT

Out of all the haematological aberrations associated with Primary hypothyroidism, anemia is found in 25% of hypothyroid patients. There are few reports of multiple autoimmune diseases co-existing with Hashimoto's thyroiditis and Grave's disease, however associations with primary hypothyroidism have not been reported yet. We report patient with primary hypothyroidism who presented with Immune thrombocytopenic purpura.

CASE

A 65 years old housewife presented with puffiness of face, progressive weight gain associated with hoarseness of voice, somnolence, constipation, hair loss for 7 months. She also gave history of progressive paleness of skin and easy fatigability for 5 months. She also had bleeding manifestations started in the form of development of purpuric sports all over the body, epistaxis, melaena and gum bleeding for one and a half months prior to admission. She presented with severe anemia and heart failure. Apart from a history of bronchial asthma for last two years for which she was on treatment with inhaled bronchodilators and intermittent oral steroids, the patient denied any history of hypertension, jaundice, chest pain, recurrent mucosal ulcerations or occurrence of purpuric spots, drug allergy, thyroid swelling, surgery or irradiation or blood transfusion in the past. She was diagnosed as a case of hypothyroidism by a local doctor who started her on 150 μg of L-thyroxin which she took only for 15 days.

Examination revealed an obese pale elderly women who was dyspneic with a respiratory rate of 30/minute with pulse of 74/minute and a blood pressure of 110/90 mmHg. General examination revealed marked pallor with the presence of purpuric spots in extremities and a subconjunctival hemorrhage in the left eye. Her skin was dry and coarse. Facial puffiness, hoarseness of voice, macroglossia and loss of lateral part of eyebrows were also present. JVP was raised. There was no icterus, cyanosis, clubbing or goiter. Cardiovascular examination revealed muffled heart sounds and a hemic murmur at the base of the heart. There were fine crepitations at right lung base without any evidence of exacerbation of asthma. She had tender hepatomegaly. Other systems were within normal limits.

At presentation the patient's hemoglobin was 2.0g/dl, total count 12,500 mm^3 with normal distribution, platelet count 46,000/ mm^3 and reticulocyte count 3.9%. Packed cell volume (PCV) was 9%, RBC count 1.27 million/ mm^3, mean corpuscular volume (MCV) 72.4 fl, mean corpuscular hemoglobin (MCH) 15.7pg, mean corpuscular hemoglobin concentration (MCHC) 21.7g/dl, serum iron 44μg/dl, total iron binding capacity (TIBC) 333.4μg/dl. Peripheral blood showed total leucocyte count with a normal

range and distribution, RBC's showed marked marked anisopoikilocytosis with microcytes and macrocytes and platelets were markedly reduced. Bone marrow examination revealed myeloid: erythroid (M:E) ratio of 3:1 with normal myeloid distribution, normoblastic erythropoiesis and normal megakaryocytes suggesting peripheral thrombocytopenia on account of increased destruction of platelets. Renal and liver function tests were normal. Stool for occult blood was positive. ECG showed low voltage complexes and Chest XRay was suggestive of cardiomegaly. 2D Echocardiography showed moderate pericardial effusion with normal LV function and no evidence of tamponade. Thyroid function test revealed a T_4 and TSH level of 3.7 μg/dl and 14.7 μU/ml respectively. Upper GI endoscopy showed multiple small sub mucosal hemorrhagic spots in whole of stomach. Immunological markers like ANA, DsDNA, LE cell phenomena were absent and CRP was positive. Anti thyroglobulin antibody and anti-thyroid peroxidase antibody were normal. Anti platelet antibodies could not be done due to lack of facilities.

The patient was diagnosed as a case of Primary Hypothyroidism with Immune Thrombocytopenic Purpura with pericardial effusion and severe anemia due to GI bleeding.

The patient was treated with eltroxin, steroids and multiple blood and platelet transfusions. Melaena stopped and her somnolence and facial puffiness improved. Pulse rate at the time of discharge was 94/ minute with a blood pressure of 140/80 mmHg. She was discharged with a hemoglobin level of 8.5 g/dl and platelet count of 56,000/mm^3.

One months later, the patient reviewed with hemoglobin of 13.7 g/dl and platelet count of 1.4 lacs/mm^3. Repeat 2-D ECHO showed minimal pericardial effusion. The patient continues to be on follow-up for more than one year after her admission presenting with acute exacerbations of asthma and her platelet count is maintained around 1,00,000/mm^3 without any bleeding episodes with 20mg of Prednisolone per day.

DISCUSSION

Immune thrombocytopenia is a diagnosis of exclusion where there is autoimmune-mediated destruction of platelets. It could be primary ITP, where it is idiopathic in origin, or secondary ITP, caused by variable conditions like viruses, drugs, infections, autoimmune disorders and malignancies. In ITP, the life span of platelets are reduced due to both antibody-mediated destruction and decreased production of platelets.[1] Majority of patients are asymptomatic with ITP. The presentation varies from mild bleed like petechiae and purpura to life-threatening hemorrhage in cases of severe thrombocytopenia.

The association between ITP and autoimmune thyroid disease has been described in medical literature in form of small retrospective studies and numerous care reports.[2] 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 ITP. Various reports have shown improvement in clinical outcome of ITP by treating hypo-/hyperthyroidism.[3,4] Platelet-associated IgG or specific platelet antibodies occurred in 83% and 86% of patients with ITP with and without autoimmune thyroid disease, respectively. Thyroid autoantibodies, on the other hand, were detected in 89% of patients with ITP and autoimmune thyroid disease[5].

Both graves disease and hashimoto thyroiditis has been found to be associated with ITP. It is postulated that the life span of platelet is reduced in graves disease by increase in reticuloendothelial phagocytic activity[6,7].

Howver, the association of primary hypothyroidism is a rare co-existing finding with ITP. The underlying mechanism of co-existence of these disorders and whether treatment of one improves the outcome of other need to be deciphered.

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