

Thyrotoxicosis and Leukopenia – A Rare Association

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Abstract

A 62 year old male presented with complaints of high grade fever and weight loss of one month duration. He was evaluated for systemic infections, autoimmune diseases and hematological malignancies. In the presence of tremors of the hand, tachycardia, thyromegaly with abnormal thyroid profile, a diagnosis of hyperthyroidism was made. Total leukocyte count was low (2600/cmm) and the relevant investigations were negative. The patient responded well within 4 days of start of anti-thyroid drugs and is now on regular follow up.

Key Words: Thyrotoxicosis; leukopenia.

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Introduction

Thyrotoxicosis has many systemic manifestations. Hematological changes occur in leukocytes, red blood cells and platelets. The prevalence of anemia among patients with hyperthyroidism ranged from 10-15%.¹ Slight leukopenia, neutropenia and thrombocytopenia are common manifestations occurring in thyrotoxicosis and are usually of an autoimmune origin.^{2,3} One of the serious side effects of thionamides is the development of agranulocytosis.⁴ It occurs in less than 1% of the patients usually within first few weeks or months of therapy. But severe leukopenia prior to initiation of thionamides is a rare manifestation of thyrotoxicosis and paradoxically it responds well to thionamides (carbimazole) medications.

Case report

A 62 year old male was admitted to hospital with complaints of fever and weight loss one month duration. He also gave history of generalised weakness, palpitation, increased fatigability, loss of appetite, insomnia and getting angry with family members over minor issues. There was no history of cough, rash, joint pains, dysphagia, headache, vomiting and change in bowel habits. He was non-diabetic, non-hypertensive and non-smoker. On examination, he was found anxious and agitated. Pulse rate was high with 120/min, regular; BP 140/80 mmHg; respiratory rate 18/min; temperature 102°F and no anemia. Examination of hands revealed fine tremors with warm moist hands. There was no hepatomegaly, lymphadenopathy, bony tenderness, cyanosis, jaundice, petechiae or purpura. Sputum examination for tubercle bacilli was negative. He was empirically treated with ceftriaxone and artemether (antimalarial) parenterally before admission to the hospital but fever did not subside. Examination of neck revealed diffuse goiter without eye signs of ophthalmopathy. Laboratory investigations revealed Hb 10.9 g%; TLC 2600/mm³; DLC P64,L36,E0,B0; ESR 26 mm/first hour; Platelet count 140000/mm³; and fasting blood sugar 75 mg/dl. Blood and urine culture

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sterile; Widal test negative; RA factor and ANA levels normal; X ray chest normal; Bone marrow aspiration revealed hypocellularity with M:E ratio of 3:4 and normoblastic cells. Thyroid profile revealed T₃ (180 ng/dl), T₄(15 µg/dl), and TSH (0.01µ/L). Thyroid scan (Tc99m pertechnate) showed low uptake. Thyroid microsomal antibody was 80.2 IU/ml (N<34 IU/ml). In view of clinical presentation and abnormal thyroid profile report, a diagnosis of hyperthyroidism was made. He was treated with 3rd generation cephalosporins and antithyroid medications (carbimazole 20 mg twice daily). After 4 days of treatment, patient was afebrile, his appetite and weight started improving with increase in TLC to 4800/mm³. He was discharged in a satisfactory condition with now regular follow up.

Discussion

Thyroid hormones play an important role in maintaining the normal cellular functions in all organ systems. They directly stimulate erythropoiesis thus causing increased tissue oxygen demand⁵. 1-3% cases of Grave's disease may develop pernicious anemia and 15-20% may develop antiparietal cell antibodies⁶. Leukocyte count in thyrotoxicosis is usually normal with slight increase of lymphocytes³. The number of monocytes and eosinophils may also be increased. Spleen may be enlarged in 10% cases while thymic and lymph node enlargement is common⁷. Various workers⁸ have reported neutrophil count less than 2000/mm³ in 27% cases and between 2000-4000/mm³ in 57% subjects in their study. Granulocytopenia may occur due to immune mechanism in the form of anti-neutrophil antibodies although the exact cause is unknown⁹. Kurata Y¹⁰ in their study have demonstrated platelet count less than 150000/mm³ although the count mostly remains unaltered. Our patient had marginally low platelet count with no clinical evidence of petechiae or purpura. Some workers² have documented cause of thrombocytopenia due to presence of antiplatelet antibodies or binding of TSH receptor antibodies to the actin

binding protein on the platelets. Only one case of thyrotoxicosis with leukopenia has been reported in the literature¹¹ where the patient was 19 years female with TLC of 2400/ mm³ and platelet count of 150000/ mm³.

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