

Pancytopenia with Hyperthyroidism

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Abstract: Pancytopenia is a rare complication of hyperthyroidism. Several cases had been reported before in the literature, the pathogenesis is still unclear. Immunological mechanisms have been suggested to be involved in the reduction of the life-span of blood cells and platelets. Hyperthyroidism can be associated with various hematological disorders, Especially single lineage abnormalities such as anemia (34%), leukopenia (5.8%), thrombocytopenia (3.3%) are reported, but pancytopenia is a rare presentation of hyperthyroidism. The suspected etiologic mechanisms include ineffective hematopoiesis, reduction in blood cell life span, autoimmune process, toxicity of thyroid hormone. We report a case describes a 47 yr old, female with pancytopenia. On further investigations the patient was found to have hyperthyroidism.

Keywords: Pancytopenia, Hyperthyroidism, thyrotoxicosis, hematopoiesis.

I. INTRODUCTION

Hyperthyroidism can be associated with various hematological disorders. The prevalence of anemia among patients with hyperthyroidism ranged from 10 to 15%. Slight leucopenia, neutropenia, and thrombocytopenia are common events in thyrotoxicosis and are usually of an autoimmune origin. Pancytopenia is a rare complication of hyperthyroidism. Thyroid hormone stimulates the production of erythrocytes via erythropoietin and by stimulating hemopoietic stem cells directly. Patients with hypothyroidism demonstrate anemia, but there are few reports of hematologic disorders, such as iron deficiency anemia, pernicious anemia, or idiopathic thrombocytopenic purpura, in patients with hyperthyroidism. We report here an interesting case of hyperthyroidism with severe anemia, leukocytopenia, and thrombocytopenia and the Successful treatment of hyperthyroidism caused resolution of the pancytopenia.

II. CASE REPORT

A 47-year-old woman, complaint of progressive weight loss, palpitation, and dyspnea on exertion for one month. The symptoms worsen recently that patient felt breathlessness even at rest.

On examination, patient was tachypnea, with blood pressure of 160/60 mmHg, heart rate 110/min, She does not smoke nor drink alcohol. No fever or signs of gastrointestinal bleeding. respiratory rate 25/min and body temperature 37°C. There was pallor with anemic conjunctiva, no jaundice or icteric sclera. The thyroid gland was mildly enlarged with warmth, firm in consistency, non-tender, no palpable nodule and no thyroid bruit. There was fine tremor on both hands but no exophthalmos. The heart rate was regular without murmur, fine basal rales heard over both lungs. Liver and spleen were impalpable, no pretibial myxedema, instead mild pitting edema found on both feet. The stool occult blood examination was negative A complete blood count analysis was done at clinic and the result showed pancytopenia with WBC count of 2050/ μ l (normal: 4000– 10000/ μ l), hemoglobin of 5.7 g/dl (normal: 13–15 g/dl) and platelet count of 59 x 103/ μ l (normal: 130–400 x 103/ μ l). The Patient did not take any medication before admission. Hypoalbuminemia and mild hyperbilirubinemia were found from blood biochemistry examination. Peripheral blood smear showed normocytic RBCs with anisocytosis and poikilocytosis. The Coombs tests were negative. Reticulocyte was 5.8%, while the reticulocyte production index was 1.3%. Bone marrow cytology was hypercellularity with adequate megakaryocyte, both myeloid and erythroid series were hyperplastic. The folic acid level was normal (13.4 ng/ml), iron reserve was mildly decreased (ferritin: 48 ng/ml) and the vitamin B12 level was low (149 pg/ml) .Abdominal ultrasound showed coarse echogenicity of liver parenchyma, suspect chronic liver disease. There was mild splenomegaly with spleen 12 cm in

diameter. Hormonal studies revealed hyperthyroidism with T4: 16.1 µg/dl (normal: 5.1–13.5 µg/dl), T3: 3.65 ng/ml (normal: 0.8–2.0 ng/ml), and TSH: 0.005 µU/ml (normal: 0.3–6.5 µU/ml). The anti-TSH receptor antibody titer was 59% (normal: < 10%), and anti-microsomal antibody was positive on 1600X. Mild enlargement of thyroid gland with increased vascularity was found from ultrasound examination. Tc99M-pertechnetate thyroid scan showed diffuse increased uptake of tracer over the thyroid. The patient was admitted for conservative management and further workup.

III. DISCUSSION

An association of pancytopenia and hyperthyroidism is of rare occurrence. This patient presented with hyperthyroidism and pancytopenia. Cell counts fluctuated with thyroid function. She had received no drugs before admission. Bone marrow showed arrested hematopoiesis and no signs of Myelodysplastic syndromes (MDS). These results suggest that pancytopenia was caused by hyperthyroidism. The prevalence of anemia among patients with hyperthyroidism ranged from 10 to 15%. In association with hyperthyroidism, iron deficiency anemia, pernicious anemia and hemolytic anemia have been reported. The pathogenesis of pancytopenia in hyperthyroidism is still poorly understood. Immunological mechanisms have been suggested to be involved in reduction of life-span of blood cells and platelets. Leukopenia may complicate hyperthyroidism (before starting antithyroid treatment) but is usually associated with pancytopenia. The case demonstrated an improvement in cell counts with improvement of thyroid function, similar to the clinical course in the present case. These findings support the thyrotoxic state itself causing the arrested hematopoiesis which leads to pancytopenia. The coexistence of pancytopenia and hyperthyroidism is uncommon, and no consensus exists as to the most appropriate treatment. Moreover, the delivery of standard anti-thyroid medication may be delayed based on the concern about drug-induced pancytopenia. In fact, pancytopenia resolved in most of the patients after they achieved the euthyroid state with the initiation of anti-thyroid treatment.

IV. CONCLUSION

This case describes an important though rare clinical association. Any patient with unexplained pancytopenia should be investigated for thyroid dysfunction. and the Successful treatment of hyperthyroidism caused resolution of the pancytopenia. The pathogenesis of pancytopenia in hyperthyroidism is still poorly understood.

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