Letter to Editor

Hematologic Recovery of Pancytopenia after Treatment of Hashimoto's Thyroiditis and Primary Adrenal Insufficiency

Dear Editor,

Pancytopenia is not a common hematologic complication of endocrinopathies. The characteristic hematologic manifestation of hypothyroidism is normocytic normochromic anemia. The pathogenesis of this anemia is hypothesized to reflect either a lack of erythropoietin production or a physiologic adaptation to the decreased tissue oxygen requirements resulting from a decrease in the basal metabolic rate.[1] In most cases of pancytopenia, hypothyroidism was related to hypopituitarism.[2] This is to our knowledge the first case report of pancytopenia related to Hashimoto's thyroiditis and adrenal insufficiency that was completely treated with thyroid and steroid replacement therapy.

A 57-year-old woman presented to the emergency room with complaints of restlessness, increasing fatigue, and weight gain. The patient had also noticed that her face and extremities appeared puffier. She denied any medication or illicit drug use. She had no history of any prior pregnancies. Her menstrual period was regular and unremarkable. On physical examination, she was found to be obese with myxedema in extremities and her reflexes were sluggish. Her blood pressure was 85/60 mmHg. The patient did not have goiter and did not exhibit any hyperpigmentation or signs of dehydration.

The patient's laboratory tests revealed a pancytopenia with white blood cell 2.5×10^9/L (segmented neutrophil count was 0.8×10^9/L), Hemoglobin 8.4 g/dl, and platelet count 95×10^9/L. The reticulocyte count was 2.0%. The mean cell volume (MCV) was 95.6 fl, mean cell hemoglobin (MCH) 34.0 pg, and mean cell hemoglobin concentration (MCHC) 35.0 g/dl. She had normal iron studies, Vitamin B12, and folate levels. The results of rapid human immunodeficiency virus test, direct Coombs test, and anti-nuclear antibody titer were negative for her. She had normal sodium and potassium levels. Lactate dehydrogenase and bilirubin levels were normal, and hence, possibility of hemolysis was excluded. Serial stool occult blood test results were negative. Her thyroid stimulating hormone level was 422 mU/L (reference range, 0.4~5.0 mU/L), Total T3 0.33, free T4 0.22 (reference range, 5~12 mg/dl). Her baseline 8-am cortisol level was 5.4 µg/dl (reference range, 5~25 µg/dl). Computed tomography (CT) scans of the head, chest, and abdomen were unremarkable. Bone marrow biopsy revealed normal hematopoietic features lacking any atypical features. Serum anti-thyroid peroxidase antibodies were elevated at 250 IU/L (reference range, <100 IU/L), and this confirmed the diagnosis of Hashimoto's thyroiditis. Remainder of endocrinology workup including luteinizing hormone (17 IU/L), FSH (11 mIU/ml), and insulin-like growth factor-1 (210 ng/ml) were within normal limits.

The patient was subsequently started on levothyroxine 25 µg IV q12 hrs and levothyroxine 75 µg PO daily. An adrenocorticotropic hormone (ACTH) stimulation test was done; the baseline cortisol level was 5.4 µg/dl and the ACTH stimulation raised it to 25 µg/dl. The plasma ACTH level was 80 µg/dl. The results were consistent with primary adrenal insufficiency. Patient was given Dexamethasone 2 mg IV q12 hrs. After treatment for 1 week, she improved clinically, but what was surprising was that her WBC count improved to 5.2×10^9/L, Hemoglobin 10.0 g/dl, and platelets 200×10^9/L. Subsequently, the patient was discharged on levothyroxine 125 µg PO daily and prednisone 5 mg daily. At 3-month follow-up, outpatient laboratory tests revealed that she maintained her hematologic stability.

As this patient had both hypothyroidism and adrenal insufficiency, it may be difficult to specifically identify the precise role of each hormone deficit that contributed to this patient's pancytopenia. The bone marrow biopsy revealed no evidence of aplastic anemia, which is a common etiologic factor.[3] Nevertheless, recent data suggests that hypothyroidism is the major contributing factor in the pathogenesis of pancytopenia in patients with hypopituitarism. Song, *et al*. reported that the pancytopenia in patients with myxedema coma was secondary to marrow hypoplasia as documented by bone marrow biopsy.[4] Other authors have suggested that the normal and effective bone marrow hematopoeisis was affected by an autoimmune reaction, which was a plausible explanation in this case.[5] There was a prior report of pancytopenia in a patient with hypopituitarism and autoimmune hypothyroidism, which resolved following the initiation of corticosteroid and thyroid replacement therapy.[6] Unlike that fascinating case, our patient's

diagnostics revealed no evidence of hypopituitarism. In conclusion, in light of our experience, we recommend that a systematic endocrine work-up (thyroid function tests including anti-thyroid peroxidase antibodies, etc) should be part of the standard diagnostic work-up of unexplained pancytopenia so that effective management and treatment can be implemented.

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