CLINICAL VIGNETTE

Normocytic Anemia in TPO-Positive Hypothyroidism

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Case Presentation

A 50-year old male presented for a routine health evaluation. He complained of a fatigue and a vague feeling of being unwell, not in his usual state of health. He was being treated for depression and decreased motivation with bupropion XL 150 mg daily.

A review of prior labs drawn several weeks before his initial visit by another provider revealed TSH of 20.88 and free T4 of 0.7.

His family history was significant for Hashimoto's thyroiditis in his mother, two sisters, and one brother.

In our office, thyroid studies were repeated, and demonstrated TPO of 186, TSH of 17.9, total T3 of 108, and free T4 of 0.9. He also had normocytic anemia, with Hgb of 12.6 and MCV of 94. Further evaluation included serum iron of 92 (80-180mcg/dL), with saturation 30%, ferritin of 136 (20-250ng/mL), RBC folate 491 (140-628 ng/mL), serum folate 14.7 (2-20ng/mL), vitamin B12 of 425 (200-900lg/mL), total testosterone of 526 (300ng/dL), and normal Hgb electrophoresis.

Since there was no obvious etiology for the normocytic anemia, a working diagnosis of TPO-positive hypothyroidism causing anemia was established. The patient was started on levothyroxine, which was gradually increased, guided by serial thyroid testing, with restoration of euthyroid status. His most recent thyroid studies included a TSH of 1.5 and normal free T4, and Hgb of 13.3 with a stable MCV of 94. Review of prior tests included a Hgb of 13.3 two years prior to his first visit to our office, without prior thyroid studies. Once the euthyroid state was achieved, the patient reported feeling much improved with energy and sense of well-being.

The patient was referred to Hematology, who agreed with the diagnosis of TPO-positive hypothyroidism as the likely cause, and the patient was advised to have yearly CBC's along with thyroid testing on replacement therapy.

Discussion

Anemia and TPO-positive hypothyroidism often occur concurrently and hypothyroidism should be considered in cases of anemia of uncertain etiology.[1] The mechanisms are unclear and may be multifactorial. Anemia in hypothyroidism is most often normocytic, normochromic, and hypoproliferative, but it may be microcytic or macrocytic.

A purposed mechanism is decreased potentiation of erythropoietin on bone marrow erythroid colonies due to insufficient thyroid hormone production.

Another possible mechanism includes anemia of chronic disease. The chronic inflammatory state induces interleukin-6, which potentiates hepcidin, ultimately reducing both intestinal iron absorption and release of iron from macrophages. One study demonstrated a decrease in hepcidin concentration in TPO-positive hypothyroidism following restoration of euthyroidism.[2]

Macrocytosis can be seen in hypothyroidism, despite normal levels of vitamin B12 and folate.[3] Brodsky reported 25% of patients with macrocytic anemia that resolved with levothyroxine.[3] Some patients had a decrease in MCV following treatment with levothyroxine. It should be noted that pernicious anemia is a common finding in TPO-positive hypothyroidism, which can lead to B12 deficiency and add to the macrocytosis. It is useful to measure a vitamin B12 level in such patients.

REFERENCES

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