Thyroid

THYROID DISORDERS CASE REPORT

"Fueling the Fire" - Irish Sea-Moss Resulting in Jod-Basedow Phenomenon in a Patient With Grave's Disease

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Background: Jod-Basedow phenomenon is a rare cause of thyrotoxicosis due to excess iodine intake. Herbal supplements containing sea-moss have high iodine amount which may precipitate thyrotoxicosis in patients with underlying Grave's disease or autonomous thyroid nodules. **Clinical Case:** A seemingly healthy 28-year-old female presented to the ED with chief complaint of fatigue with associated anxiety, palpitations and weight loss. On admission her temperature was 100.4 F, pulse 126 bpm and blood pressure 116/56 mmHg. Exam was unremarkable for thyroid goiter or orbitopathy. Labs revealed WBC count 3.4 $x10^3/\mu L$ (ref range 4.0-11.0) with neutropenia, hemoglobin 4.3 g/dL (11.7-15.7), platelet 49 $x10^3/\mu L$ (150-450). Liver transaminases (AST, ALT, and alkaline phosphatase) were elevated with levels up to 4 times the upper limit of normal. She was diagnosed with hemolytic anemia secondary to severe Vitamin B12 deficiency due to pernicious anemia. TSH was <0.01 mIU/L (0.27-4.20), free T4 2.46 ng/dL (0.8-1.9) and total T3 139 ng/dL (76-181). The patient subsequently endorsed remote history of hyperthyroidism diagnosed 7 years ago however she could not recall the underlying etiology or the name of medication she was treated with. She reportedly stopped this medication after 1 month due to developing goiter. She also endorsed intermittent use of store-bought supplement of Irish sea moss and bladderwrack in last 2 years. Further workup revealed elevated TSI and TBII antibody titers establishing diagnosis of Grave's disease. Thyroid ultrasound showed normal sized heterogeneous hypervascular gland with no nodules. I-123 thyroid uptake and scan showed diffuse moderately elevated radioiodine uptake of 16.8% and 40.8% at 4 and 24 hours, respectively. Thionamide therapy was withheld due to concern of neutropenia and transaminitis. She was treated with beta-blocker after which her vital signs normalized. Labs 1 week after stopping sea moss showed TSH 0.01 mIU/L and free T4 1.4 ng/dL. **Conclusion:** Irish sea moss is a readily available herbal supplement with high, variable amounts of iodine. Despite little scientific evidence, it is often marketed to improve goiter amongst other health benefits. The recommended daily iodine intake per the FDA is 150 mcg. Higher amounts are expected to initially cause a short-lived suppression of thyroid function; the Wolff-Chaikoff effect, followed by "escape" and accelerated production of thyroid hormone in abnormal thyroid gland, known as Jod-Basedow phenomenon. In our case, the patient unknowingly worsened her underlying Grave's disease due to the Jod-Basedow effect. Of note, apparantly she had a longer than expected course of Wolff-Chaikoff effect preceding the thyrotoxic state due to sporadic irregular intake of sea moss. Discontinuing sea moss led to clinical and biochemical improvement of hyperthyroidism without requiring thionamide therapy.

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Graves' Disease, a Late Complication of Chronic Graft vs Host Disease

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Introduction: Graft-versus-host disease (GVHD), a complication of bone marrow transplant (BMT), occurs when the donor white blood cells attack the recipient's host cells and can occur acutely or chronically several years post-transplant. It is very rare for chronic GVHD to cause hyperthyroidism due to Grave's disease and thyrotoxicosis years after BMT. Hyperthyroidism after BMT is thought to occur by GVHD or by donor's auto-reactive lymphocytes transferred to recipient, with majority of cases occurring with donors that have an underlying autoimmune thyroid condition.
Case Report: We report a 62 year old male who presented with palpitations, tremors, sweating, weight loss, and anxiety for 3 weeks. Past medical history was significant for allogenic BMT due to AML 14 years ago, which was further complicated by GVHD of eye, liver and skin. Physical exam was negative for thyromegaly or thyroid tenderness. Initial work up showed TSH 0.03 (normal 0.4-5.0 MUI/l), free T4 3.32 (normal 0.6-1.2 ng/dl), TSI 194 % (normal <140 %). Ultrasound revealed a diffuse hypervascular thyroid gland with numerous avascular cystic areas in both lobes and increased homogenous, symmetrical uptake consistent with Graves' disease was noted on radioactive iodine uptake scan. With clinical suspicion of GVHD leading to Graves' disease with subsequent thyrotoxicosis, he was started on methimazole. However, due to persistent increase in transaminases, concern for agranulocytosis with methimazole, and risk of developing worsening GVHD of the skin or eye with radioactive iodine therapy, he underwent total thyroidectomy. His pathology showed bilateral lymphoepithelial lesions with florid follicular hyperplasia, marked monocytoid hyperplasia, and Germinal B cells positive for CD10 and negative for BCL2. Lymphoepithelial lesions can be seen in chronic lymphocytic thyroiditis due to persistent inflammation.
Conclusion: This case raises the question on the etiology of thyroid nodules and Graves' disease occurring after BMT. We believe that his pathology findings may be due to chronic GVHD. Hyperthyroidism after BMT is extremely rare, with only a few case reports documenting this phenomenon. We believe that our patient developed hyperthyroidism 14 years after BMT most likely as a sequela of chronic GVHD with underlying immune dysregulation leading to formation of TSI and thyroiditis due to cellular invasion. This suggest a multifactorial etiology of hyperthyroidism in patients with GVHD. However, more prospective studies are required to the explain etiology of hyperthyroidism in GVHD. Moreover, such patients should be closely monitored for development of thyroid dysfunction or thyroid nodules.

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