Clinical Case: A 21-year-old male with recently diagnosed metastatic non-seminomatous germ cell choriocarcinoma presented with persistent tachycardia and anxiety. At diagnosis, his β- human chorionic gonadotrophin (β-HCG) was elevated to 6,435 mIU/mL (normal <1 in male) and thyroid-stimulating hormone (TSH) was within normal limits. At presentation, however, his β-HCG increased to 103,229 mIU/mL, TSH was suppressed <0.02 mcIU/mL (normal 0.3-4.7), and free thyroxine was elevated (FT4) 2.6 ng/dL (normal 0.8-1.7). His thyrotoxicosis improved with initiation of methimazole; however, his TSH remained undetectable due to persistently elevated β-HCG levels. His course was complicated by hemorrhagic shock and acute liver injury in the setting of a presumed intraluminal gastric metastasis, necessitating the discontinuation of methimazole. He was continued on steroids to try to minimize T4 to T3 conversion, but ultimately his thyroid hormones uptrended. He became stable enough to tolerate 5 days of chemotherapy, after which his FT4 quickly normalized. Unfortunately, he continued to suffer from vasodilatory shock and ultimately passed away.
Discussion: It has been demonstrated that HCG can bind to the TSH receptor and has thyrotropic activity. The development of hyperthyroidism requires HCG levels >200,000 mIU/mL that are sustained for several weeks (1). It is unknown what the prevalence of hyperthyroidism is in choriocarcinoma, but it has been shown to greatly increase when serum HCG levels are greater than >50,000 mIU/mL (2).
Conclusion: Hyperthyroidism can be difficult to recognize in patients suffering from cancer as many of the typical symptoms can also be seen with active malignancy. Patients with HCG-secreting tumors should be evaluated for hyperthyroidism and may benefit from treatment until the underlying cause can be managed.
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Thyroid

THYROID DISORDERS CASE REPORT

A Case of Parathyroid Adenoma Three Decades Post Radioactive Iodine Therapy: Is It Just a Coincidence or Real Risk?

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Background: External radiation to the head and neck is a known risk factor for the development of parathyroid adenoma. But the incidence is very rare in internal radiation recipients. Here we describe a case of parathyroid adenoma after 30 years of radioactive iodine therapy for Graves' disease.

Clinical Case: A 50-year-old female presented during an annual visit with progressive fatigue, polyuria, polydipsia, nocturia, muscle weakness, and some memory impairment. She had a past medical history of Graves' disease treated with radioactive iodine therapy 30 years ago with subsequent hypothyroidism, controlled essential hypertension, asthma, and obesity BMI 32.5 kg/m^2. There was no history of nephrolithiasis, fractures, pituitary tumor, or acid reflux. Family history was noted for thyroid diseases, no calcium issues or hyperparathyroidism. Neck examination did not reveal any thyromegaly or palpable nodule. Laboratory tests showed calcium 11.4 mg/dL (8.6-10.3), ionized calcium 6.1 mg/dL (4.2-5.4), parathyroid hormone 213.9 pg/mL (12-88), total vitamin D 22 ng/mL (31-100), TSH 0.47 uIU/mL (0.34-3.00), Free T4 1.1 ng/dL (0.6-1.6), creatinine 0.7 mg/dL (0.6-1.2), eGFR >60 ml/min/1.73 m^2, 24-hr urine calcium 405 mg (40-350). Bone density revealed T-score -4.9 at the lumbar spine, -2.8 at the total hip, -3.1 at the femoral neck, and -2.9 at distal 1/3 radius. Neck ultrasound showed atrophy of the thyroid gland with a lobulated hypoechoic area measuring up to 1.3 x 0.9 x 0.9 cm without internal blood flow at posterior inferior to the right lobe of the thyroid gland. Parathyroid SPECT/CT scan revealed no evidence of parathyroid adenoma. The hypoechoic lesion was suspected to be a lymph node according to imaging studies. However, according to laboratory and bone density results, we suspected primary hyperparathyroidism in which the patient required surgery due to current symptoms. The patient underwent surgery with an intraoperative finding of a nodule at the superior of the right parathyroid gland. A frozen section of the nodule was sent which confirmed parathyroid adenoma. The gland, weighted 483 mg, was removed with subsequent improvement of intraoperative parathyroid hormone level from 238.5 pg/mL to 26.5 pg/mL. Follow up calcium was at 9.5 mg/dL. The patient was supplemented with calcium and vitamin D afterward. Her symptoms improved significantly.
Conclusion: Hypercalcemia in a patient with prior history of radioactive iodine therapy should raise concern for parathyroid adenoma. Imaging of the parathyroid gland should be cautiously interpreted with laboratory tests as it could be a false negative. Due to patient met criteria for surgery, the surgical approach should be pursued for both diagnostic confirmation and definite treatment. Intraoperative parathyroid hormone monitoring is beneficial in equivocal imaging and in reflecting successful resection of uniglandular disease.

Thyroid

THYROID DISORDERS CASE REPORT

A Case of Rare Autoimmune Pancytopenia Due to Graves' Disease

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Introduction: Graves' disease has been associated with cytopenias, most commonly anemia. Pancytopenia is a rare complication and is not often encountered.
Case Presentation: A 54-year-old woman with history of multiple sclerosis on alemtuzumab was referred for abnormal thyroid function tests. At the initial visit, the patient reported a 20-pound unintentional weight loss, tremors, sweating and heat intolerance. She was 3 years post-menopausal and had no history of recent viral illness, contrast exposure or family history of thyroid disease. Physical exam revealed an anxious appearing woman with fine tremors in both hands, hyperreflexia but no lid lag or exophthalmos. Labs included TSH <0.005 uIU/mL (0.400-4.2), free T4 2.89 ng/dL (0.80-1.50), TSH receptor binding antibody 8.24 IU/L (< 2.00), TSI 7.27 IU/L (<0.56), WBC 2900/uL (4500-11000), ANC 1746/uL (1900 - 8000), hemoglobin 11.4 g/dL (11.7-15.0) and platelet 207,000/uL (150,000-450,000). Thyroid ultrasound noted subcentimeter hypoechoic/cystic nodules and 24-hour thyroid uptake showed 56.4% symmetric uptake (upper limit of normal 30%). Due to leukopenia, the patient was started on propranolol and underwent radioiodine ablation with 14.8 mCI. Two weeks later, she reported easily bruising and gum bleeding and her blood work was significant for pancytopenia. The patient was referred to hematology and two bone marrow samples were obtained. Though MDS/MPN was initially suspected based on atypia in both specimens, there were no immunophenotypic, cytogenetic or molecular abnormalities suggesting a neoplastic process. Instead the patient was thought to have autoimmune pancytopenia secondary to Graves' and the atypia was believed to be secondary to peripheral consumption. She was started on prednisone 1 mg/kg/day with resolution of her cytopenias.
Discussion: Graves' disease has been associated with hematological abnormalities including isolated anemia (the most common), thrombocytopenia or leukopenia. Pancytopenia is an uncommon complication and is rarely described in the literature. The exact mechanism remains unclear, but may be related to either reduced production of hematopoietic cells from the bone marrow or increased destruction of mature hematopoietic cells due to autoantibodies. Since thyroid hormones are known to increase erythropoietin, this leads to an exaggerated consumption of iron, folic acid and vitamin B12 and can cause various forms of anemias. Leukopenia may be secondary to immunologic destruction whereas thrombocytopenia may be due to antiplatelet antibodies or increased splenic sequestration. Pancytopenia is rare. Our patient was treated with high dose prednisone with resolution of her pancytopenia, which suggests an autoimmune process as the mechanism. Our case showcases a rare complication of Graves' disease and highlights that high dose steroid therapy may improve cytopenias associated with this condition.

Thyroid
THYROID DISORDERS CASE REPORT

A Case of Recurrent Paralysis and Low Potassium: Is It the Thyroid?

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Introduction: Thyrotoxic periodic paralysis (TPP) is a rare complication of hyperthyroidism that is characterized by episodes of hypokalemia and acute weakness. Although hyperthyroidism is more common in females, over 95% of cases of TPP have been observed in males, especially in Asian males with an incidence of 2% among hyperthyroid patients. In non-Asian populations, the incidence in hyperthyroid patients is estimated to be around 0.1 to 0.2% [1]. We describe a case of TPP seen in a Hispanic male.
Case Report: A 36-year-old Hispanic male with no past medical history presents with weakness in all extremities and difficulty breathing after consuming a carbohydrate heavy meal the night prior. He reports a recent, similar episode evaluated in another ER, which resolved after given potassium supplementation. He denied any vomiting, diarrhea, polyuria, diaphoresis, use of insulin or other medications, or any family history of paralysis.
His labs were significant for hypokalemia of 1.9, TSH of <0.005 (0.358-3.740), free T4 of 2.22 (0.76-1.46), and total T3 of 2.7 (0.60-1.81). Thyroid stimulating immunoglobulin was 0.12 (0.0-0.55). His symptoms improved and potassium levels normalized following the administration of potassium chloride. He was discharged on propranolol and advised to follow up for further workup of his hyperthyroidism with radioactive iodine uptake scan.
Discussion: Thyrotoxic periodic paralysis is a potentially life-threatening condition associated with cardiac arrhythmias and respiratory failure. Hyperthyroidism increases response to β-adrenergic stimulation, which increases activity of the sodium-potassium ATPase and causes hyperpolarization of skeletal muscle [2]. Hyperthyroid patients are prone to episodes of paralysis due to their increased susceptibility to the hypokalemic action of insulin, which activates the sodium-potassium ATPase pump, and epinephrine, which stimulates β-adrenoreceptors. Management of an acute attack of TPP includes potassium administration. In cases where paralysis and hypokalemia are not reversed, intravenous propranolol has been shown to resolve the attack by blocking the β-adrenergic receptors. Definitive treatment of TPP includes managing the hyperthyroid state with medical therapy, radioactive iodine therapy, or surgery. Until the euthyroid state is reached, a β-blocker can prevent episodes of acute paralysis. Avoidance of carbohydrate heavy meals, exercise, and stress are recommended as these factors can potentially exacerbate hypokalemia. In patient with acute paralysis, it is important to consider the diagnosis of TPP as this condition can be prevented once euthyroidism is achieved. Diagnosis and management will lead to prevention of morbidity and mortality associated with the hypokalemia.
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Thyroid
THYROID DISORDERS CASE REPORT

A Case of Rhabdomyolysis in a Patient With Hashimoto's Thyroiditis

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