ordered. Thyroid stimulating immunoglobulin (TSI) were found to be elevated at 173 % baseline (\(<=140\)). Thyroid peroxidase (TPO) antibodies and thyroglobulin antibodies were also elevated at 362 IU/mL H* (\(<=9\)) and 2 IU/mL H* (\(<=1\)) respectively. The overall picture was consistent with evolving early Graves’ disease. Conclusion: Multiple factors are frequently cited in the pathogenesis of autoimmune hyperthyroidism including viral and bacterial infections\(^1\) and there have been several reported cases of autoimmune disease related to SARS-CoV2 infection\(^2\). This case is one of several emerging cases of autoimmune hyperthyroidism possibly linked to COVID-19. References: 1. Smith, T. J . Graves’ Disease. New England Journal of Medicine. 2016 October 20; 375:1552-1565 2. Mateu-Salat, M., Urgell, E., Chico, A. SARS-COV-2 as a trigger for autoimmune disease: report of two cases of Graves’ disease after COVID-19. Journal of Endocrinological Investigation. 2020 July 19

Thyroid

THYROID DISORDERS CASE REPORT

A Grave Initial Presentation of Graves’ Disease in a Patient With Moyamoya

Courtney Clutter, MD, Morgan Jordan, DO.
Brooke Army Medical Center, Fort Sam Houston, TX, USA.

Background: Moyamoya syndrome is chronic stenoocclusive disease involving the intracranial internal carotid arteries and their proximal branches along with an associated condition, such as hyperthyroidism\(^1\). The concurrence of moyamoya and Graves’ disease is rare. Ischemic stroke in moyamoya syndrome is postulated to be precipitated by thyrotoxicosis-induced hemodynamic instability. Clinical Case: A 63-year old Korean female with history of moyamoya disease with two prior ischemic strokes, hypertension and type 2 diabetes mellitus presented to the ER with 6 hours of left leg weakness and involuntary arm movements. A code stroke was activated and neurologic examination was notable for left leg paresis and left arm stereotypy. CT head showed loss of gray-white matter differentiation in the right frontal lobe concerning for acute ischemia. CT angiography of the head and neck showed diffuse stenosis of intracerebral vasculature and significant stenosis of the cavernous and supraclinoid portions of the internal carotid arteries. MRI brain later confirmed an acute infarct in the right ACA distribution. Neuroimaging incidentally showed a multinodular goiter with a 1.7 cm right thyroid nodule. Subsequently TSH was obtained and resulted as \(<0.030\) mIU/mL (0.27-5.00 mIU/mL) with a reflex FT4 of \(>7.00\) ng/dL (0.6-1.8 ng/dL). A review of her prior TFTs showed biochemical euthyroidism. Due to iodinated contrast administration on admission, RAIU scan was deferred. Thyroid ultrasound showed multinodular goiter with diffuse increased vascularity and multiple TI-RADS 4 and 5 nodules bilaterally. On further questioning, the patient reported tachycardia, diaphoresis, weight loss and decreased appetite prior to hospitalization. A diagnosis of Graves’ disease was confirmed with TSI of 70.7 IU/L (0.00-0.55 IU/L). She was started on methimazole 20 mg twice daily and propranolol 20 mg q6h. FT4 down trending from \(>7.00\) to 3.3 ng/dL at time of discharge. Following four weeks of methimazole 20 mg daily, FT4 normalized to 1.70 ng/dL. The patient chose to continue antithyroid drug therapy for treatment of Graves’ disease. Conclusion: Thyroid function assessment should be considered when evaluating a patient with moyamoya and acute ischemic stroke. If moyamoya syndrome associated with Graves’ disease is identified, treatment should be aimed at maintenance of euthyroidism. Reference: 1. Scott RM, Smith ER. Moyamoya disease and moyamoya syndrome. N Engl J Med. 2009 Mar 19;360(12):1226-37. Disclaimer: The views expressed herein are those of the authors and do not reflect the official policy or position of Brooke Army Medical Center, the U.S. Army Medical Department, the U.S. Army Office of the Surgeon General, the Department of the Army, the Department of the Air Force and Department of Defense or the U.S. Government.

Thyroid

THYROID DISORDERS CASE REPORT

A Grave Turn on Hashimoto’s Thyroiditis - A Case Series on Four Patients With Autoimmune Hypothyroidism that Converted to Grave’s Disease.

Michelle N. Lee, MD, Jeffrey A. Colburn, MD.
Department of Endocrinology, San Antonio Military Medical Center, Fort Sam Houston, TX, USA.

Disclaimer: The views expressed herein are those of the author(s) and do not reflect the official policy or position of Brooke Army Medical Center, the U.S. Army Medical Department, the U.S. Army Office of the Surgeon General, the Department of the Army, the Department of the Air Force, or the Department of Defense or the U.S. Government.

Introduction: The most common cause of hypothyroidism is Hashimoto’s thyroiditis, a destructive autoimmune injury to the thyroid gland. Rarely, autoimmune hypothyroidism can be caused by thyroid-stimulating hormone (TSH) receptor blocking antibodies (TSHRab), and can be difficult to differentiate clinically from Hashimoto’s. Grave’s disease is the most common etiology of hyperthyroidism, and is typically caused by activation from TSHRab acting as an agonist for the TSH receptor. Patients with autoimmune thyroiditis, whether from TSHRab or Hashimoto’s, have been infrequently reported to convert to Grave’s disease\(^1\)\(^-\)\(^3\). Presentation: We present four cases whom initially presented with typical symptoms of hypothyroidism, were diagnosed with autoimmune hypothyroidism and started on levothyroxine. All four cases were later found to be hyperthyroid and ultimately diagnosed and treated for Grave’s disease. Conclusion: Primary hypothyroidism can rarely transition to a hyperthyroid state, although these cases may be underreported. The mechanism isn’t well understood, but is hypothesized to be from a switch of a predominance of TSH receptor blocking antibodies (TBAb) to that of thyroid stimulating antibodies (TSAb)\(^4\). Assays using competitive binding for TSH receptor antibodies will not differentiate between blocking and stimulating antibodies\(^4\). A high index of suspicion is needed to diagnose these individuals.

References: 1. McLachlan SM, Rapoport B. Thyrotropin-blocking autoantibodies and thyroid-stimulating...
autoantibodies: Potential mechanisms involved in the pendulum swinging from hypothyroidism to hyperthyroidism or vice versa. *Thyroid*. 2013;23(1). doi:10.1089/thy.2012.03742.

Takasu N, Matsushita M. Changes of TSH-stimulation blocking antibody (TSAb) and thyroid stimulating antibody (TSI) over 10 years in 34 TSAb-positive patients with hypothyroidism and in 98 TSAb-positive Graves’ patients with hyperthyroidism: Reevaluation of TSAb and TSI in TSH-receptor-antibody (TRAb)-positive patients. *J Thyroid Res*. 2012;2012. doi:10.1155/2012/1821763.

Gonzalez-Aguilera B, Betea D, Lutteri L, et al. Conversion to Graves’ disease from hashimoto thyroiditis: A study of 24 patients. *Arch Endocrinol Metab*. 2018;62(6). doi:10.20945/2359-39970000000864.

Li Y, Kim J, Diana T, Klasen R, Olivo PD, Kahaly GJ. A novel bioassay for anti-thyrotropin receptor autoantibodies detects both thyroid-blocking and stimulating activity. *Clin Exp Immunol*. 2013;173(3). doi:10.1111/cei.12129

**Thyroid**

**THYROID DISORDERS CASE REPORT**

*A Killian-Jamieson Diverticulum Masquerading as a Thyroid Nodule*

Reema Patel, MD, Maha Abdulla, DO, Jianyu Rao, MD, Abemayor Elliot, MD, PhD, Dianne Cheung, MD. UCLA, Los Angeles, CA, USA.

**Background:** A Killian-Jamieson diverticulum is a rare outpouching in the cervical esophagus, just below the cricopharyngeus muscle, that can be easily mistaken for a thyroid nodule on ultrasonography (1).

**Clinical Case:** A 65-year-old woman underwent a thyroid ultrasound after her primary care physician noted left-sided thyromegaly. The ultrasound described a 33 mm solid, hypoechoic, wider-than-tall nodule in the left mid gland with an obscured posterior margin as well as macro- and microcalcifications. Given the size and highly suspicious features on ultrasound, she was referred to endocrinology clinic for a fine needle aspiration (FNA). She underwent ultrasound-guided FNA of what appeared to be the previously described thyroid nodule. Surprisingly, the pathology report noted degenerative changes with amorphous debris and possible foreign materials (vegetable or food) without any thyroid tissue. She was sent for an MRI neck, which showed the left neck mass communicating with the esophagus, favoring a left lateral projecting Killian-Jamieson esophageal diverticulum with internal debris. She was referred to head and neck surgery. Given only minimal symptoms of dysphagia, there are no current plans for surgery.

**Conclusion:** This case illustrates the possibility of mistaking a Killian-Jamieson diverticulum as a thyroid nodule. Recognition of this rare disease process in the differential diagnosis of thyroid nodules with high risk ultrasound characteristics may prompt more advanced imaging with MRI or CT, and lead to an accurate diagnosis prior to subjecting patients to unnecessary and potentially harmful FNAs (2).

**References:**

(1) Kim HK, Lee JI, Jang HW, et al. Characteristics of Killian-Jamieson diverticula mimicking a thyroid nodule. *Head Neck*. 2012;34(4):599-603.

(2) Bonacchi G, Seghieri M, Becciolini M. Killian-Jamieson diverticulum: real-time sonographic findings. *J Ultrasound*. 2016;19(4):295-298. Published 2016 Apr 21. doi:10.1007/s40477-016-0208-3.

**Thyroid**

**THYROID DISORDERS CASE REPORT**

*A Million Reasons for Hyperthyroidism. Reporting a Case of Thyrotoxicosis in a Setting of Metastatic Choriocarcinoma*

Ali Samim Ahmed, MD, MPH, Elias Said Siraj, MD. Eastern Virginia Medical School, Norfolk, VA, USA.

**Background:** Human chorionic gonadotropin (hCG) induced transient hyperthyroidism is often seen during pregnancy. Other rare causes of beta hCG induced hyperthyroidism include trophoblastic tumors (hydatidiform mole and choriocarcinoma) and in men, germ cell tumors. The mechanism for hCG induced thyrotoxicosis lies in the structural similarity between TSH and hCG molecules leading to the direct stimulation of the TSH receptors. In regard to treatment, while gestational thyrotoxicosis is usually mild, self-limited and does not need medications, the hyperthyroidism of trophoblastic tumors may require antithyroid medications in addition to treating the underlying tumor. Thionamide use is reserved for moderate to severe cases of hyperthyroidism and for presurgical optimization. In our case report, we present a 22-year-old African American female with choriocarcinoma induced thyrotoxicosis requiring thionamide therapy.

**Clinical Case:** A 22-year-old African American female presented to our emergency room after a witnessed generalized tonic-clonic seizure. Her brain CT scan showed a 4 CM mass concerning for AVM malformation with a subsequent brain MRI confirming parenchymal hematoma with surrounding vasogenic edema. Past medical history was significant for molar pregnancy managed with dilation and curettage followed by laparotomy to remove the pelvic mass. Given her history of molar pregnancy, pelvic ultrasound was performed which showed complex heterologous structure of uterine origin concerning for malignancy. Additional imaging studies of the lung, brain, abdomen and pelvis were performed, which showed possible metastasis to the lung, adnexa, and to the brain. Her clinical exam showed heart rate of 130 beats/min with a normal rhythm, fine tremors, aphasia, and a decrease grip strength in her right upper extremity. Her thyroid gland was slightly enlarged with no tenderness. Laboratory tests showed hCG of >1,000,000 mIU/mL, TSH of < 0.01mcU/mL, free T4 of 5.1ng/dL. Her TPO and TSI were negative. With a diagnosis of hyperthyroidism due to trophoblastic disease, she was treated with methimazole and propranolol resulting in rapid clinical and biochemical improvement. Later, left frontal craniotomy and hematoma evacuation was performed with histology confirming the diagnosis of metastatic choriocarcinoma. She was later transferred to a specialized center where she received chemotherapy. Her hCG subsequently dropped down to 699 mIU/mL.

**Conclusion:** We report a rare case of metastatic choriocarcinoma causing symptomatic hyperthyroidism. The diagnosis is made by excluding other common causes of hyperthyroidism and it should be