Conversion of hypothyroidism to hyperthyroidism is even more rare. We report a case of an established Hashimotos thyroiditis patient who converted into Graves disease.

**Case Description:** 67-year-old female with a past medical history of iron-deficiency anemia, dyslipidemia, and depression presented with a six-month history of fatigue, cold-intolerance, hair loss, and weight gain in September of 2015. Laboratory tests confirmed diagnosis of Hashimotos thyroiditis with an elevated TSH 80.7 (0.40-4.50 mIU/L), FT4 0.2 (0.8-1.8 ng/dL), and positive thyroid antibodies TPO 24 (0.0-8.9 IU/mL). She was started on Levothyroxine 88 mcg daily. Gradually she had a decreased requirement of Levothyroxine; from February 2016 to March 2017 she maintained a normal TSH range while on 50 mcg/day with resolution of her symptoms.

The patient was then lost to follow-up until she presented in the clinic in September 2018 with complaints of several weeks of easy fatigability, 10lb-weight loss, and periorbital edema. She was found to have a suppressed TSH 0.01, and elevated FT4 2.3, and FT3 8.4 (2.3-4.2 pg/mL). Her Levothyroxine 50 mcg/day was discontinued for four days and labs were repeated which still showed suppressed TSH and elevated FT4 and FT3. She was found to have a positive TRAB and a positive TSI which are consistent with hyperthyroidism. Thyroid ultrasound was performed which showed a heterogeneous thyroid gland with increased vascularity, confirming the diagnosis of Graves disease. She was started on Methimazole 10 mg daily. Her Methimazole dose was adjusted according to her thyroid function test until she had a total thyroidectomy in October 2019. She was started on levothyroxine post-operatively and as of March 2020 is on Levothyroxine 50 mcg/daily.

**Conclusion:** Despite the rarity of Hashimotos thyroiditis converting to Graves disease, it is possible that those affected can be encountered by primary care providers and hospitalists and could easily be mistaken for overreplacement of levothyroxine. Close monitoring of the patient along with regular thyroid function tests will be required for ongoing follow-up.

### Thyroid

**THYROID DISORDERS CASE REPORT**

**A Severe Case of Hypothyroidism: Immunotherapy Induced**

Sritani Chanchula, MD.
Cameron Regional Medical Center, Cameron, MO, USA.

**Background:** Immune check point inhibitors are playing a crucial role in the treatment of many cancers. Their primary role is to reactivate anti tumor cytotoxic T cells. Programmed cell death 1 (PD 1) is a transmembrane protein expressed on T cells, B cells and Natural killer cells. PD 1 ligand is expressed in many tumor cells along with hematopoietic cells and their interaction directly inhibits apoptosis of the tumor cell. Pembrolizumab binds PD1. FDA approved pembrolizumab initially for nonsquamous cell lung cancer and metastatic melanoma that express PD1 ligand which showed improved survival compared to standard therapy. Additionally it has been used in several others tumors with more than 50% PD L1 expression. Although well tolerated, these agents can cause immune related adverse events.
Thyroid dysfunction is reported to range between 3.2-10.1% patients receiving pembrolizumab. **Clinical Case:** 86 year old Caucasian female with past medical history of hypertension and osteoarthritis was diagnosed with stage III metastatic colon cancer in the hepatic flexure and underwent hemicolectomy. Pathology showed 6.5cm mass and 2/30 lymph nodes positive. Due to fear of side effects from chemotherapy, patient refused further treatment. Her follow up labs showed elevation of carcinoembryonic antigen (CEA). Her oncologist suggested Pembrolizumab, immunotherapy. She was started on 5 cycles every 3 weeks. She had fatigue and fell twice after first cycle, but her CEA levels were improving, so it was decided to continue treatment. Soon after fifth cycle she became extremely weak, lethargic, and unable to do any ADLS and was exhausted all the time. Her labs showed TSH 137, free T4 0.4. At that point decision was made to stop pembrolizumab. She was started on levothyroxine 25mcg by her oncologist and referred to me few months later. She complained of tiredness, low energy. On exam Pulse 78, Blood pressure 152/77. Her labs showed TSH 106 and Free T4 0.45. Her levothyroxine dose was increased to 75mcg and followed her 6 weeks later. Her energy levels improved significantly and she is back to her normal self. Her thyroid function was normal. At this point patient has decided not to undergo any treatments for colon cancer. **Conclusion:** Immune related thyroid events are increasingly noticed with wide use of pembrolizumab. There is insufficient evidence to suggest the cause of these events. Reversible destructive thyroiditis and overt hypothyroidism are the common clinical presentations. It is important to recognize patients at high risk for these adverse events so these highly efficacious agents can be safely used in patients. Thyroid has been a frequent target for anti PD 1 treatments, further research can be of benefit to use against thyroid cancers.

**Thyroid**

**THYROID DISORDERS CASE REPORT**

**Acute Inflammatory Demyelinating Polyneuropathy (AIDP) Masked by Autoimmune Thyroiditis**

Stephanie Wirtshafer, DO1, Iqra Iqbal, MD2.

1Philadelphia College of Osteopathic Medicine, Philadelphia, PA, USA, 2Abington Memorial Hospital, Horsham, PA, USA.

Hashimoto’s thyroiditis and Guillain-Barre syndrome (GBS) are autoimmune disorders that are both well-known in their own right. Hashimoto’s is one of the most common causes of primary hypothyroidism, and GBS involves immune mediated damage to the peripheral nervous system. The association between the two is a rare clinical entity. This case demonstrates that these entities can occur together and could be related in similar pathophysiology. A 37 year old male presented with complaints of bilateral hand and feet numbness for one month. The numbness started in the hands, then involved the feet, and was mostly felt in tips of extremities. He also complained of weakness in arms and legs. Neurology exam showed bilateral patellar, ankle, and biceps hyporeflexia. Muscle strength was 5/5 in all extremities, but decreased grip strength was noted in the hands. Initial lab work including complete blood count, comprehensive metabolic profile and urinalysis were all in normal range. Computerized tomographic scan (CT) head was normal while CT abdomen/pelvis showed hepatic fatty infiltration. Other lab tests including HIV, syphilis, Hepatitis B, Hepatitis C, glycosylated hemoglobin A1c, lipid panel, anti-nuclear antibody, anti-neutrophil cytoplasmic antibodies, serum/urine protein electrophoresis, alcohol level, vitamin B1, B6, folate, copper, and creatine kinase were all negative or within normal range. Lab abnormalities included elevated thyroid stimulating hormone (TSH) of 20.2 mIU/l and low normal B12 level of 289 pg/ml. His triiodothyronine (T3) and thyroxine (T4) hormone levels were in normal range. A thyroid peroxidase antibody level came back as high as 966 IU/ml. A diagnosis of Hashimoto’s thyroiditis leading to subclinical hypothyroidism was made. Patient was discharged on vitamin B12 and 112mcg of Synthroid. Instead of getting better, he returned 1 week later with worsening numbness and tingling which was now ascending upward to bilateral knees and elbows. Meanwhile TSH improved to 10 mIU/l and vitamin B12 increased to 1162 pg/ml. A magnetic resonance imaging (MRI) of the cervical/thoracic spine was unremarkable. A lumbar puncture showed negative xanthochromia, 0 WBC, 0 RBC, 0 neutrophils, 0 lymphocytes, 0 monocytes, glucose 63 mg/dl, elevated protein of 57 mg/dl, and culture was negative. Guillain-Barre syndrome was then the working diagnosis, more specifically its most common subtype, acute inflammatory demyelinating polyneuropathy (AIDP). Patient received five days of intravenous immunoglobulins and his symptoms improved. He was then discharged to follow up with endocrinologist. This subtle presentation of GBS/AIDP masked by Hashimoto’s thyroiditis and vitamin B12 deficiency suggests a close association of autoimmune etiology between these disorders. Although rare, endocrinologists should consider this rare association in cases of paresthesias with unexplained symptoms.

**Thyroid**

**THYROID DISORDERS CASE REPORT**

**Adenomatous, Unilateral, Retrosternal Extension of the Thyroid Gland presenting as a Mediastinal Mass**

Aisling Glass, BMBS, BSc1, Margaret Elizabeth Griffin, MD, MRCPI2, Carla M. Moran, MRCPI, PhD3.

1Endocrine Department, Beacon Hospital, Dublin, Ireland, 2Beacon hospital, Dublin, Ireland, 3Beacon Hospital, Dublin, Ireland.

**Background:** Thyroid-related causes of mediastinal masses include retrosternal goiters and thymic enlargement associated with Graves’ disease. Here, we present a case of significant unilateral retrosternal growth of the thyroid gland, presenting as an incidental mediastinal mass, without any evidence of contralateral disease. Associated subclinical hyperthyroidism presents a therapeutic challenge. **Clinical Case:** A 68-year-old gentleman presented to the emergency department with a non-infective exacerbation of known Chronic Obstructive Pulmonary Disease. CT Pulmonary Angiogram revealed a right sided 5.6cm paratracheal mass, which seemed to originate from the posterior aspect of the right lobe of the thyroid and extended...