evaluation and was given methimazole 60mg TID, prednisone 20mg daily, and restarted cholestyramine. With his significant cardiovascular risk, aggressive medical management was attempted prior to surgical evaluation. After his thyroid function failed to respond to medical intervention, multidisciplinary discussion was had with patient, family, and physician teams regarding surgical intervention versus continued long-term monitoring. Family elected to pursue surgery. Thyroidectomy was performed by an experienced endocrine surgeon successfully and his FT4 and T3 decreased appropriately requiring him to be initiated on levothyroxine supplementation. AIT can be separated into Type I, in which there is an increase in synthesis of T4 and T3 with amiodarone providing increasing substrate, and Type II in which there is destructive thyroiditis, releasing excess T4 and T3. In the United States, approximately 5% of individuals who are on amiodarone therapy develop hyperthyroidism, majority being Type II. If possible, amiodarone should be discontinued in Type I AIT, but there is no clear evidence for discontinuation in Type II. Medical management includes thionamides for Type I AIT and glucocorticoids for Type II AIT. Patients who are refractory to drug therapy should be treated with thyroidectomy. The advantages of a surgical procedure
with careful cardiovascular monitoring overall outweigh the morbidity and mortality of uncontrolled thyrotoxicosis.

Thyroid

THYROID DISORDERS CASE REPORT

Amiodarone Induced Thyrotoxicosis: A Case of Refractory Disease Treated With Thyroidectomy

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Amiodarone induced thyrotoxicosis (AIT) is a challenging diagnosis that affects 3-5% of patients taking amiodarone in the United States. Type I AIT is seen in patients with preexisting thyroid disease and is generally treated with thionamides while type II AIT represents a destructive thyroiditis that responds to glucocorticoids. A mixed type exists and is associated with higher mortality, especially in older adults with cardiovascular disease. Thyroidectomy is considered a last resort option for patients intolerant or refractory to medical treatment.
A 70 year-old male with a history of coronary artery disease, ventricular tachycardia (VT), and heart failure was referred to the endocrine clinic for abnormal thyroid function tests that showed TSH <0.0023 uIU/mL (0.4-4.7), Free T4 2.51 ng/dL (0.7-1.48) and Free T3 5.37 pg/mL (1.71-3.71). He endorsed palpitations, excessive sweating, tremors, and reported taking amiodarone for 3 years prior to presentation. Vitals showed normal pulse and blood pressure. Thyroid autoantibodies including TSI and TBII were within normal limits. Thyroid ultrasound showed mild thyromegaly with normal vascularity and no nodules. AIT was suspected and he was started on methimazole 20 mg daily, prednisone 30 mg daily and continued on his home metoprolol 100 mg daily. Methimazole and prednisone were both up titrated in a week because his labs did not improve. One month later, he presented to the hospital with acute exacerbation of heart failure. His TFTs showed (TSH <0.0021 uIU/mL, FT4 >5.0 ng/dL, FT3 4.61 pg/mL). Thyroid RAIU showed severely decreased uptake secondary to the high iodine content of amiodarone. He remained thyrotoxic despite using higher doses of prednisone (60 mg daily) and methimazole (90 mg daily). He was changed to PTU (900 mg daily) and started on cholestyramine, with no improvement in overall status. Several weeks after admission, a total thyroidectomy was performed. His postoperative course was unremarkable except for hypoparathyroidism. He was clinically and biochemically euthyroid one week after his procedure. At 6 months follow up, he remained stable on levothyroxine 100 mcg/day but continued to require calcitriol and calcium supplementation.
We present an interesting case of mixed type AIT refractory to medical therapy associated with cardiovascular compromise. This case highlights the challenges in the diagnosis and management of such patients. Thyroid autoantibodies, thyroid ultrasound and RAIU were more indicative of Type II AIT, however, lack of response to high dose steroids was inconsistent with the diagnosis. While receiving the treatment for both Type I and II AIT, our patient had persistent clinical and biochemical thyrotoxicosis and required thyroidectomy. Although most AIT patients are treated medically, thyroidectomy is reserved for those most severe and refractory cases and is considered a viable option in such patients.

Thyroid

THYROID DISORDERS CASE REPORT

Amiodarone-Induced Hypothyroidism Initially Presenting as Decompensated Heart Failure and Hyponatremia

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Introduction: Amiodarone is an effective antiarrhythmic, but it is associated with altering thyroid function, ranging from thyrotoxicosis to hypothyroidism. In this study, we discuss amiodarone-induced hypothyroidism (AIH) presenting with hyponatremia and myxedema.
Case Presentation: A 75-year-old Caucasian man with a history of ischemic heart disease with an ejection fraction of 55-60% was seen in the ED with complaints of worsening lower limb edema and shortness of breath. Associated symptoms of constipation, generalized weakness, daytime sleepiness, and increased urinary frequency were noted. He was recently diagnosed with atrial fibrillation with a CHA_2DS_2-Vasc score of 5 and started on amiodarone nine months ago. He weighed 235 lbs with a BMI of 33.85 and vital signs are as follows: blood pressure 90/64, heart rate 64, respiration rate 13, and a temperature of 36.6 °C. Physical examination revealed a well-developed man in mild distress and slightly lethargic, with a palpable thyroid gland, heart sounds revealed an irregularly irregular heart rate, elevated JVD, bilateral rales, and bilateral pitting edema. Labs showed mildly low HB of 11.7, low Na of 125 mmol/L, elevated creatinine at 1.67 mg/dl, low urine osmolality at 270 mOsm/kg, and BNP of 24 with negative troponin. Chest x-ray showed cardiomegaly. TSH was

elevated at 93 uIU/mL and low free T4 at less than 0.25 ng/dL with a negative anti-TPO. A diagnosis of amiodarone-induced hypothyroidism was made, and the patient was started on increased furosemide and levothyroxine 25 mcg daily. Significant improvement was noted in mental status, sodium level, and volume status within three days; and the patient was discharged home on 50mcg of levothyroxine.
Discussion: This case illustrates the need to constantly investigate the etiology of decompensated heart failure, especially when new medications with potential culprit side effects are noted or suspected. Amiodarone which is helpful in the management of atrial fibrillation has been known to cause thyroid dysfunction as hypo or hyperthyroidism. What is not widely known is that these endocrine dysfunctions can occur just a few weeks after therapy initiation.
Conclusion: We recommend that patients who started on amiodarone be monitored for thyroid dysfunction; especially when they present with deterioration in the cardiac function or show symptoms of endocrinopathy.

Thyroid

THYROID DISORDERS CASE REPORT

An Unusual Cause of Thyrotoxicosis and Gynacomastia

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We describe the case of a 24 year old man with metastatic non-seminomatous germ cell tumour who presented with hyperthyroidism and gynaecomastia associated with elevated human chorionic gonadotrophin(HCG). Following a 3 month history of gynaecomastia, flank pain, nausea and 7kg weight loss, the patient attended the emergency department with persistent vomiting. Initial laboratory investigations reported TSH <0.05mU/L (normal range 0.3-4.3) and free T4 29.5pmol/L (12-22), TPO antibodies negative, FSH <1 and LH 1 U/l, testosterone >52 nmol/L (9-29.0), bioactive prolactin 1048mU/L (63-245), SHBG 110.6 nmol/l (18.3-54.1), and oestradiol 3935pmol/L (<223). Clinical examination revealed bilateral tender gynaecomastia and supraclavicular lymphadenopathy. Testicular examination identified a left testis irregularity that was confirmed on ultrasound. Chest Radiograph revealed multiple bilateral opacities measuring up to 6 cm. Initial urine HCG was negative when tested on two occasions; however given suspicion for a HCG-secreting tumour, serum HCG was measured and reported as 503,944 IU/ml (<5) and AFP 17.6 IU/ml (0-5.0). The negative urine HCG is believed to be due to "hook effect". CT revealed bulky retroperitoneal lymphadenopathy measuring 13 cm consistent with metastatic spread from the non-seminamatous germ cell tumour of testicular origin. Up front chemotherapy protocol with 4 cycles cisplatin based therapy was initiated given his disease burden and degree of symptoms. Radical orchidectomy was deferred until after chemotherapy - at which time germ cell tumour was only identified on immunohistochemistry staining. Over the 2 months following chemotherapy, all endocrinopathies resolved with corresponding reduction of HCG to 65.0 IU/ml. Symptoms of hyperthyroidism abated with treatment and his TFTs were biochemical normal. Oestrogen level also normalised with treatment. HCG-induced hyperthyroidism is a rare cause of hyperthyroidism. Endocrine manifestations occur in <5% germ cell tumour presentation but should be considered particularly when multiple endocrine abnormalities are present.

Thyroid

THYROID DISORDERS CASE REPORT

An Unusual Etiology of Graves' Disease: Alemtuzumab

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Cluster of differentiation 52 (CD-52) is a glycoprotein expressed on the surface of most lymphocytes and it is the most prevalent marker in T cells, B cells, natural killers and monocytes. Alemtuzumab, a CD-52 monoclonal antibody, is one of the initial therapies approved for patients with relapsing-remitting multiple sclerosis. It acts by inducing rapid and prolonged depletion of lymphocytes with a consequent immunosuppression. Although not clearly understood, Alemtuzumab has been associated with the development of autoantibodies. These have been reported to cause thyroid injury resulting in 10-15% incidence of new-onset Graves' disease.
This is the case of a 38 year-old man with medical history of relapsing-remitting multiple sclerosis who came to the Endocrinology clinic to establish care due to abnormal thyroid function tests. Patient has received Alemtuzumab since the past two years. Three months prior to arrival, he was found with weight loss, hyperdefecation and tremors by his Neurologist. Patient was found with suppressed TSH for which Methimazole was commenced. Thyroid ultrasound showed normal size and homogenous right and left thyroid lobes, and no evidence of cystic or solid masses. Thyroid stimulating immunoglobulins were found elevated which correlated with Graves' disease. Moreover, thyroid scintigraphy showed 34% radioiodine uptake at 24 hours indicating de novo synthesis of thyroid hormone in this patient with hyperthyroidism. As Alemtuzumab was identified as a precipitating cause of Graves' disease, therapy was discontinued and plasmapheresis will be given for the treatment of relapsing-remitting multiple sclerosis. Monoclonal antibody use has increased since the past decades. It has been well described in literature that monoclonal antibodies against programmed cell death receptor 1 (PD-1) and programmed cell death ligand 1 (PD-L1) may cause autoimmune thyroid disease. Nonetheless, it is important to recognize that other monoclonal antibodies may have similar adverse effects. Alemtuzumab is a monoclonal antibody and antineoplastic agent used for relapsing multiple sclerosis, some hematologic malignancies, and as an induction agent for solid organ transplantation. Its main effects include autoimmunity with thyroid being one of the most described targets. In these patients, expert clinicians should recognize the possibility of thyroid disease for prompt treatment which will improve quality of life.