showed large pericardial effusion with findings suggestive of right heart failure, Echocardiogram showed left ventricular dysfunction and large pericardial effusion. The patient was taken to the operating room for emergent pericardial window creation with pericardectomy and was admitted to the Cardiac Care Unit for management of tamponade status post pericardial window. Levotyroxine 150 mcg, Lithotrynine 25 mcg and Hydrocortisone 50 mg were started, the steroid was discontinued after adrenal insufficiency was ruled out. The pericardial drain was removed after 8 days and repeated tests showed TSH: 13.1 uIU/mL, fT3: 3.37 pg/mL, fT4: 0.5 ng/dL, studies of pericardial fluid only showed polymorphonuclear cells. The patient’s symptoms resolved and she was discharged on Levotyroxine 150 mcg and Lithotrynine 25 mcg. During follow up visits the thyroid function tests were normal, Lithotrynine was discontinued and a repeated Echocardiogram showed normal systolic function. **Conclusions:** Pericardial effusion can be found in 9–30% of patients with hypothyroidism but only in very rare cases (less than 3%) is associated with cardiac tamponade and occurs when there is a severe underlying condition like myxedema coma or prolonged untreated hypothyroidism (1). It is important not to miss that dizziness and presyncope in a patient with hypothyroidism may be a manifestation of cardiac tamponade. Once the diagnosis of hypothyroidism is made it is imperative to start treatment early as untreated hypothyroidism can cause severe cardiovascular complications but even when such are present, they can be reversible with thyroid replacement therapy. Reference: (1) Kahaly, G. and Dillmann, W. (2005). Thyroid Hormone Action in the Heart. Available at: https://academic.oup.com/edrv/article/26/5/704/2355198 [Accessed July 12 2019].

**Bone and Mineral Metabolism**

**Bone and Mineral CASE REPORTS I**

**Cinacalcet Resistant Tertiary Hyperparathyroidism with One Large Parathyroid Gland**

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**SAT-349**

Tertiary hyperparathyroidism is thought to develop after long term secondary hyperparathyroidism, such as CKD on dialysis. In this case, all parathyroid glands are significantly enlarged. We report a case of an 84-year-old female with a past medical history of ESRD on HD and recurrent nephrolithiasis who was found to have an enlarged multinodular goiter with the dominant mass in the lower pole of the right gland measuring about 4.7 cm on thyroid ultrasound. Blood work was done which showed elevated intact PTH levels at 2720 pg/mL (12–88). Her calcium level was normal at that time at 10.5 mg/dL (8.6–10.8) with an albumin of 4.2 g/dL (3.5–5.7), and a phosphorus of 5.7 mg/dL (2.5–4.5). Patient had a DEXA scan which showed severe osteoporosis in the lumbar spine, left hip, and right forearm. Patient had increased PTH levels despite being on Cinacalcet. She had a nuclear medicine parathyroid scan with SPECT CT which showed increased uptake along the right inferior thyroid concerning for a large right lower parathyroid adenoma or functional thyroid nodule. FNA of the nodule was done and showed colloid nodule but PTH wash showed elevated PTH at 7634 pg/mL. She was referred for right lower parathyroidectomy and Cinacalcet was discontinued prior to surgery. She had right and left inferior parathyroidectomy and 4 gland exploration. Pathology showed the right inferior parathyroid gland to be markedly hyper-cellular, weighing 36 grams consistent with hyperplasia and the left inferior parathyroid gland to be slightly hyper-cellular parathyroid gland consistent with hyperplasia. No evidence of malignancy was noted on pathology. Prior to surgery her calcium level of 10.6 mg/dL which went down to 9.6 mg/dL post-operatively. Patient then developed severe hypocalcemia and hungry bone syndrome following the surgery requiring a calcium drip for 3 days post-operatively. Was changed to oral calcium but required large amounts of calcium gluconate and calcitriol supplementation and an extended hospital stay of 13 days. However, throughout the hospital stay, PTH levels continued trending back up to 239 which may represent either increased activity from remaining parathyroid glands or residual parathyroid adenoma with incomplete resection. Post-operative US neck showed Post-surgical collection in the lower pole of the right gland measuring 4.8 cm compatible with recent resection. In conclusion, tertiary hyperparathyroidism can develop due to one enlarged parathyroid gland or an adenoma, which can be resistant to cinacalcet.

**Bone and Mineral Metabolism**

**BONE AND MINERAL CASE REPORTS I**

**Etanercept Induced Hypercalcemia**

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**SAT-345**

Non-PTH mediated hypercalcemia has multiple etiologies including medications. There are some case reports showing TNF-alpha inhibitors causing sarcoidosis or sarcoidosis type presentation involving lungs, skin, lymph nodes, and kidneys.

We report here a 69-year-old male with a past medical history of prostate cancer status post prostatectomy (in remission), CKD stage 3, and seronegative rheumatoid arthritis who was sent to the ED from clinic due to hypercalcemia. On admission, patient had a calcium level of 14.2 (8.7–10.4 mg/dL), albumin 4.1 (3.4–4.8 g/dL), magnesium 2.0 (1.3–2.7 mg/dL), creatinine 2.5 (0.7–1.3 mg/dL), iPTH <2 (14–88 pg/mL), PSA 0.13 (0–4 ng/mL), 25-OH vitamin D 45.3 (30–100 ng/mL), 1,25-OH vitamin D 144 (19.9–79.3 pg/mL), alkaline phosphatase 46 (46–116 U/L) along with generalized weakness, nausea, vomiting, poor appetite, decreased oral intake, dizziness, and slight epigastric pain. Patient was given IV fluids and a dose of Reclast 3.3 mg IV was given 2 days after admission. At home for at least the past year before admission, patient was on etanercept for seronegative rheumatoid arthritis which was held on admission. CT thorax was done which showed geographic
ground glass worst in the bilateral upper lobes where there is interlobular septal thickening and interstitial consolidation highly suspicious for interstitial pneumonitis and reactive appearing mediastinal lymphadenopathy. During the admission PTH-rP was 2.5 (0–2.3 pmol/L) and angiotensin converting enzyme was 36 (9–67 U/L). Based on previous case reports, etanercept was held. Upon discharge 6 days later, calcium had improved to 10.0 mg/dL, albumin 3.3 g/dL, and creatinine to 1.5 mg/dL. Upon follow up, etanercept was continued to be held. One month later, calcium level improved to 9.2 mg/dL, albumin 4.0 g/dL, iPTH 187 pg/mL, PTH-rP 3.0 pmol/L, and 1.25 OH vitamin D 50.7 pg/mL. Patient's calcium levels remained within normal range for over one year after admission and 1.25-OH vitamin D remained normal. Moreover, patient had nuclear medicine parathyroid scan which showed no evidence parathyroid adenoma. An ultrasound thyroid was done which was negative for any thyroid/parathyroid mass or nodule. Whole body bone scan was done which showed no evidence of osseous metastatic disease. Given up trending PTH-rP levels, patient was evaluated by oncology and was found to be up to date on age recommended cancer screening and no evidence of current malignancy. Repeat CT thorax done over one year later showed stable to mildly improved bilateral ill-defined centrilobular ground glass nodules in both lungs, greater on the left side, may represent improving infectious/inflammatory process. In conclusion, when in doubt, a good medication review is essential in the evaluation of hypercalcemia because TNF-alpha inhibitors like etanercept may cause a sarcoidosis like presentation.

Thyroid

Thyroid Disorders Case Reports I

Hypokalemic Periodic Paralysis

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Background:Hypokalemic periodic paralysis is a rare disorder associated most often with a genetic defect in electrolyte channels, which can also occur in the setting of thyrotoxicosis. The presenting state in the acquired form has a low potassium, low TSH, high free T4 or high T3. It is more common in men, and in the Asian population with an approximate incidence of 2%. Symptoms can last from hours to days and are often precipitated by stress, exercise, and/or high carbohydrate intake. It is believed that the excess thyroid hormone creates an increased catabolic state which drives potassium inward and hyperpolarizes the muscle membrane to create a paralytic state. Case:A 23 year old African American male presented with chest discomfort and palpitations. TSH was <0.01 μU/mL (normal 0.530 - 6.340) with a free T4 of 3.31 ng/dL (normal 0.60–1.60). EKG showed ventricular conduction delay and he was sent home on propranolol 20mg daily and methimazole 5mg three times daily. He returned 5 days later with worsening palpitations and now new onset weakness. He was found to have a potassium of 1.60 (normal 1.60–2.5) and magnesium of 1.2 mg/dL (normal 1.6–2.5). EKG showed normal sinus rhythm at a rate of 97, prolonged QT at 524msec (normal 330–470 msec), with repeat EKG 20 minutes later showing atrial tachycardia with a rate of 114 (normal 60–100). He was not able to move anything beyond his head, other than minor upper extremity hand movements, and could not sit up in bed. His potassium was initially repleted with eight doses of 10mEq KCl given q1h. His paralysis significantly improved within the first 4hrs, and was completely resolved by the next morning. Additional lab workup revealed thyroid stimulating antibody level of 13.00% (normal 0.0–0.55). He was discharged on methimazole 10mg twice daily and propranolol 10mg to be consistent with a chylothorax. Blood work done after chest tube placement demonstrated severe hyponatremia and hyperkalemia. Additionally, the patient was noted to be irritable. Both lab and clinical findings raised concern for possible underlying adrenal insufficiency, and further work-up (i.e. cortisol level, ACTH level) was sent. The patient was started on stress-dose steroids. ACTH and cortisol levels resulted normal. Steroids were subsequently discontinued. The patient’s chest tube output was slowly replaced with normal saline with noted improvement in both lab and clinical findings.

Conclusion:Although not well documented in pediatric literature, hyponatremia and hyperkalemia are complications of large-volume drainage of chylothoraces without proper fluid replacement. The subsequent lab findings could be consistent with adrenal insufficiency, however a full clinical picture along with a cortisol and an ACTH level can help differentiate etiologies.

Adrenal

Adrenal Case Reports II

Hyponatremia and Hyperkalemia in a Child with Chylothorax: Is This Adrenal Insufficiency?

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SUN-203

Hyponatremia and Hyperkalemia in a Child with chylothorax: Is this adrenal insufficiency?

Context: Chylothoraces are relatively rare within the pediatric population. Drainage of a chylothorax can lead to potentially serious complications including electrolyte imbalances and protein loss. The literature on hyponatremia and hyperkalemia developing after a chylothorax drainage is sparse. We report a case of electrolyte derangements after drainage of a large-volume chylothorax without adequate fluid replacement which showed no evidence parathyroid adenoma. An ultrasound thyroid was done which was negative for any thyroid/parathyroid mass or nodule. Whole body bone scan was done which showed no evidence of osseous metastatic disease. Given up trending PTH-rP levels, patient was evaluated by oncology and was found to be up to date on age recommended cancer screening and no evidence of current malignancy. Repeat CT thorax done over one year later showed stable to mildly improved bilateral ill-defined centrilobular ground glass nodules in both lungs, greater on the left side, may represent improving infectious/inflammatory process. In conclusion, when in doubt, a good medication review is essential in the evaluation of hypercalcemia because TNF-alpha inhibitors like etanercept may cause a sarcoidosis like presentation.