We found no statistical significance for TWT, AG, CV, and TIR, as a whole and when analyzed by cohorts and period of data: P1 vs P2, P1 vs P3, P1 vs P4. TBR and SH decreased with continuous ECGM use. TBR mean difference comparing P1 vs P2, P3 and P4 was -0.15±0.9 (p=0.9), 1.16±1.8 (p=0.02), and 1.8±1.6 (p=0.001) and for SH -0.05±0.8 (p=0.8), 0.6±1.3 (p=0.09), and 0.9±1 (p=0.004). There were 3 device failures; 2 post first insertion, one post repeat insertion in the same pocket. No infections, or serious bleeding were observed.

Conclusions:
Continuous ECGM use decreased time below range and time in serious hypoglycemia, without change in TWT, AG, TIR and CV. There was no significant intrapatient variability comparing consecutive device insertions regardless of whether patients elected to use the same pocket or alternate arms, suggesting that using the same insertion pocket may have the same efficacy as placement into a fresh insertion site, while decreasing the number of punctures, pain, procedure time, risk of bleeding and infection.

Diabetes Mellitus and Glucose Metabolism
TYPE 1 DIABETES MELLITUS
A Case of Opdivo Induced Type 1 Diabetes
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SAT-677
Background: Opdivo, or Nivolumab is an immunotherapy medication that works as a checkpoint inhibitor. It is used in various cancers not amenable to surgery, including lung cancer and metastatic melanoma. Opdivo has proven to be a beneficial treatment, though it is not without complications including thyroid dysfunction, hypophysitis, and autoimmune-induced diabetes.

Clinical Case: An 80-year-old female with a PMH of type 2 diabetes mellitus, hypertension, hyperlipidemia, and melanoma with metastasis to the bone was evaluated by endocrinology for acute worsening of her type 2 diabetes. The patient had been diagnosed with diabetes 10 years prior. She was well controlled on 22 units of levemir and Janumet. Her baseline HbA1c was 6.7%. She had no known complications from her diabetes. Four months prior to presentation she had been started on monthly Opdivo infusions for her metastatic melanoma, which she was tolerating well.

On presentation to the hospital, she had persistent hyperglycemia in the high 400's, an elevated anion gap, and an elevated beta hydroxybutyrate. She had no signs of acute infection and no significant changes in her diet or activity level. She was started on an insulin drip for diabetic ketoacidosis until her anion gap resolved. At discharge she was continued on 22 units of levemir, a humalog sliding scale, and Janumet. At that time it was unclear what precipitated her acute change in glycemic control.

At her follow-up endocrinology appointment several labs were drawn including a c-peptide, though antibodies were not obtained. Her c-peptide was less than 0.1 with a concurrent blood glucose value of 370. It was determined that she had developed autoimmune-induced diabetes secondary to Opdivo therapy and would require life-long insulin therapy.

Her Janumet was discontinued. She was started on a T-slim insulin pump and Dexcom G6 sensor, with improvement in her glucose control. Her Opdivo treatments were discontinued. She has since developed worsening metastasis in her femur and will be started on Yervoy. Yervoy also carries a risk of further endocrine disorders, including increased risk of hypophysitis.

Discussion: Acute changes in glycemic control warrants further investigation to determine the underlying precipitating factor. The most common complications of Opdivo are rashes and fatigue, though endocrinopathies have been noted in several patients. It was crucial in this patient to identify this side effect of Opdivo, as it helped to prevent further episodes of DKA. Careful review of recent medication changes helped identify this uncommon complication of Opdivo and prompted a timely change in her diabetes care regimen.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS I
Familial Hyperparathyroidism - Due to a Rare Genetic Mutation
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SAT-353
Introduction: Hyperparathyroidism occurs most commonly in middle age patients, predominantly in women. It can be caused by parathyroid adenoma, hyperplasia or parathyroid carcinoma. Genetic predisposition can be found in about 10% of primary hyperparathyroidism due to certain gene mutations. This case emphasizes the importance of taking a detailed family history when patients present with hyperparathyroidism at a young age, so that familial hyperparathyroidism, if present, can be detected and relatives screened.

Clinical case: A 26 y.o. male presented with symptoms of fatigue and polydipsia for several years. He was noted to have a serum calcium of 12.4 mg/dL (8.5–10.5), with parathyroid hormone of 213 pg/ml (15–65). He denied any history of kidney stones, fractures and no palpable neck masses. The patient's family history was significant for his paternal half-sister who had parathyroidectomy for hyperparathyroidism at 20yrs old and paternal grandmother died of parathyroid cancer in her 50s. The patient's father died of pancreatic cancer at 41yrs old. A neck ultrasound revealed a mass posterior to the left inferior thyroid. A Sestamibi parathyroid scan revealed a parathyroid adenoma at the postero inferior aspect of the left hemithyroid. Labs for free metanephrines and normetanephrine, prolactin and gastrin levels were all normal.

Due to his young age and the possibility of having familial hyperparathyroidism, he underwent bilateral neck exploration and parathyroidectomy, with removal of his left inferior, right superior, left superior parathyroid glands and left upper thymus. Surgical pathology revealed, hypercellular parathyroid tissue. Post operatively, his calcium and vitamin D remained within the normal range.
Genetic studies revealed a mutation in the paraffibromin gene - CDC73 (also called HRPT2), a tumor suppressor gene, which is on chromosome 1q25. The patient currently has 6 children ranging from age 5 months to 6 years. He was advised to have his children tested any time from age 7 years for the gene mutation. The patient has remained stable 4yrs post operatively, with normal calcium and PTH levels. He does not have any history of jaw tumor. He never had an ultrasound kidney done. He is being monitored with yearly lab tests.

**Conclusion:** CDC73 gene mutation-associated disorders are inherited as an autosomal dominant fashion, with variable penetrance. This gene mutation can be found in conditions such as hyperparathyroidism jaw tumor, familial hyperparathyroidism and parathyroid cancer.

Reference:
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**Thyroid**

**THYROID DISORDERS CASE REPORTS II**

**NIFTP: A Painstaking Diagnosis Through the Pathologist’s Eyes**
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SAT-461
Non-invasive encapsulated follicular variant of papillary thyroid cancer (EFVPTC) was recently reclassified as non-invasive follicular thyroid neoplasm with papillary-like nuclear features (NIFT-P). In 2018, revised and stricter criteria were proposed for a lesion to qualify as NIFT-P including no well-formed papilla or BRAF-V600E mutation. We are presenting an interesting case to highlight the importance of scrutinizing pathology slides to diagnose NIFTP with these more strict criteria.

35-year-old female from Puerto-Rico was diagnosed with Graves’ disease. After 2 years of methimazole treatment, total thyroidectomy was planned for definitive treatment of Graves’ disease. During the work up, she was noted to have a cystic nodule in isthmus, a 1.1 cm isoechoic rounded nodule in left mid-lobe and a 1.1 cm hypoechoic cystic nodule in left mid-lobe. ThyroSeq mutational analysis of tissue blocks for features and <1% papillae, without tumor capsular invasion was performed. The pathology showed an invasive follicular thyroid neoplasm with papillary-like Nuclear Features (NIFT-P) stage 1 with intermediate ATA risk for which she received adjuvant therapy of 101 mCi $^{131}$I. Although classification into NIFTP has been shown to reduce overtreatment of low risk encapsulated PTC, pathology slides should be closely scrutinized to ensure fulfillment of all criteria in order for a lesion to qualify as NIFT-P. This will minimize failure to recognize PTCs, that would warrant closer follow up and surveillance for recurrence.

1. Rossi, Esther D, et al. Noninvasive Follicular Thyroid Neoplasm with Papillary-Like Nuclear Features: Update and Diagnostic Considerations—a Review. *Endocrine Pathology* 30.2 (2019)
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**Pediatric Endocrinology**

**PEDiatric ENDOcrine CASE REPORTS II**

**A Perplexing Case of Hyponatremia and Abdominal Pain**
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MON-067
Previously healthy 20-year-old female presented with diffuse lower abdominal pain, cramping in nature, multiple episodes of emesis as well as urinary frequency. On day 2 of symptoms, she was treated for a urinary tract infection with antibiotics, as well as NSAIDs and opiates for pain relief. Her serum sodium was 133 mmol/L at this time. On day 3 of symptoms, a CT scan of the abdomen was performed however did not reveal pathology. Her serum sodium was 129 mmol/L at this time. She presented to our ED, on day 5 of symptoms, where serum sodium was down to 122 mmol/L. Despite IV fluids, her sodium continued to decrease to a nadir of 117 mmol/L. Further testing, including a serum osmolality of 242 mOsm/kg, urine osmolality of 540 mOsm/kg, and urine sodium of 207 mmol/L, was consistent with a diagnosis of SIADH. Given persistence of abdominal symptoms along with SIADH further imaging studies, including US abdomen, CT brain and Chest XR, were ordered and returned unremarkable. The constellation of SIADH along with persistent abdominal pain, with negative imaging, lead to consideration of acute intermittent porphyria as a diagnosis. Random urine porphobilinogen was found to be elevated to 147.2 mcmol/L (≤ 2.4) leading to the presumptive diagnosis of acute intermittent porphyria presenting as a neurovisceral attack. Biochemical and genetic testing is being pursued to confirm her diagnosis.

Acute intermittent porphyria is an autosomal dominant hematologic disorder characterized by deficiency in porphobilinogen deaminase, an enzyme in the heme synthesis cascade. Acute attacks are caused by accumulation of porphyrin resulting in autonomic and peripheral