decade of life. Main mechanisms involved in the pathogenesis are genetics and autoimmune causes, traumatic brain injury and infarction of the pituitary postpartum, known as Sheehan’s syndrome. Sheehan’s syndrome is characterized by postpartum hemorrhage, failure to lactate and menstrual irregularities and it can occur from immediate postpartum period to years after delivery. The most common hormone deficiencies are prolactin and growth hormone. Empty sella is the most common finding on brain MRI. We are reporting a case of a woman in her third decade with isolated ACTH deficiency due to Sheehan’s syndrome two years postpartum, able to lactate, with normal menses and normal brain MRI. Clinical Case: A 33-year-old woman G3P3A0 with hypothyroidism who was referred to Endocrinology clinics due to tiredness, fatigue and weakness. She reported postpartum hemorrhage requiring 4 PRBC transfusions and IV steroids after last pregnancy 5 years ago. Patient was able to lactate after pregnancy and continued in her usual state of health until 3 years ago when she referred loss of consciousness with traumatic head injury due to hypoglycemia. At Endocrinology office physical examination and vital signs were unremarkable, including no blood pressure or heart rate variations with positional changes. Despite hypothyroidism being adequately controlled, she continued with extreme fatigue and weakness affecting her quality of life, for which cortisol and ACTH levels were ordered. Laboratories showed normal electrolytes, negative autoantibodies, cortisol 0.20 μg/dL (5-25 μg/dL) and ACTH 22 pg/mL (10-60 pg/mL) suggesting partial isolated ACTH deficiency. ACTH stimulation test was done and noted with suboptimal response. Evaluation of other anterior pituitary hormones was normal. Brain MRI showed normal pituitary gland. She was started on hydrocortisone in AM and PM and symptoms resolved. Conclusion: Immediate recognition of isolated ACTH deficiency due to Sheehan’s syndrome is necessary due to the availability of effective treatment and morbidity and mortality associated with this serious condition. To our knowledge isolated ACTH deficiency due to Sheehan’s syndrome in which the patient was able to lactate and normal findings on brain MRI has not previously been reported. References: Shivaprasad C. Sheehan’s Syndrome: Newer advances. Indian J Endocrinol Metab. 2011 Sep; 15(3): S203-207. DOI:10.4103/2230-8210.84869.

Thyroid
THYROID NEOPLASIA AND CANCER
Primary Thyroid Lymphoma Developing from a Background of Lymphocytic Thyroiditis: First Report in Mice
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Primary thyroid lymphoma is rare, accounting for less than 5% of all thyroid malignancies. It typically develops in patients with Hashimoto thyroiditis who have approximately a 70 fold higher risk than other patients. The mechanisms underlying the development of thyroid lymphoma remain unclear, and no mouse model has been described. For our studies of papillary thyroid cancer and lymphocytic thyroiditis, we crossed TPO-Cre-ER transgenic mice and hBRAFV600E knock-in mice onto the NOD. H2b4 background to establish TPO-Cre-ER_hBRAFV600E NOD.H2b4 strain where papillary thyroid cancer is induced by the injection of tamoxifen and thyroiditis by the administration of iodine in the drinking water. Mice injected with corn oil or drinking regular water served as control. In 3 of 121 mice, TPO-Cre-ER_hBRAFV600E NOD.H2b4 mice injected with corn oil and drinking iodinated water we observed the development of thyroid lymphoma. At about 6 months after the injection of corn oil, these mice developed a marked increase in the size of the thyroid gland, which appeared hypoechoic on thyroid ultrasound. Fine needle aspiration on the thyroid gland under ultrasound was performed, along with measurement of TPO antibodies, H&E thyroid histology, immunohistochemistry, and flow cytometry at the time of sacrifice. Histology established a diagnosis of Hodgkin lymphoma with the typical Reed Sternberg cells. Flow cytometry identified an increased frequency of CD8+ effector memory T cells in the thyroid lymphoma. TPO antibodies were significantly higher in mice with thyroid lymphoma than in those without, perhaps suggesting their utility as predictive biomarkers. In summary, we report a mouse model of thyroid lymphoma that evolves from a background of lymphocytic thyroiditis with a predictable natural course that can be monitored by thyroid ultrasound and TPO antibodies. This model can be used to study the mechanisms and development of thyroid lymphoma in patients.

Healthcare Delivery and Education
EXPANDING CLINICAL CONSIDERATIONS FOR PATIENT TESTING AND CARE
Evaluation of the Timeliness of Serial Denosumab Administration at the University of Colorado Hospital
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MON-137
BACKGROUND:
Denosumab is an antiresorptive medication commonly used in the treatment of osteoporosis that works by slowing bone loss. This medication should not be delayed or interrupted without initiation of an alternative treatment (i.e. bisphosphonates) as studies have shown that this can lead to rapid bone loss, very high markers of bone turnover, and increased vertebral fracture (VF) risk. It is unknown how frequently dosing is delayed in practice settings and how best practices can ensure timely dosing. Our study aimed to (1) evaluate the frequency and causes of delayed denosumab doses at our institution and (2) compare the incidence of delayed doses before and after implementation of a new electronic ordering process.

METHODS:
We performed a retrospective chart audit for all patients receiving two or more denosumab doses at our institution between 1/1/16-8/11/18 and categorized those whose doses were >/=214 days (7 months) as delayed. We reviewed
notes, imaging, and labs in a subset of this population to assess reasons for the delay. On 8/11/18, a new outpatient infusion center (OIC) therapy plan went into place. This plan bundled a one-year denosumab order (2 doses) with an automatic referral to the OIC along with physician reminders for renewal. We compared rates of delayed denosumab dosing before and after implementation of this new order process.

RESULTS:
Between 1/1/16-8/11/18, 385 patients received 1295 doses of denosumab, with 160 (41.6%) receiving 193 instances of delayed doses. We reviewed the charts of 98 individuals who received 111 instances of late doses between 7/6/16-8/11/18. The most prevalent reasons for delays were: delays in follow-up by the patient (27.9%), delays in the provider placing an order for the drug and OIC referral simultaneously (27.9%), and delays in OIC scheduling (18%). During the 14 months after implementation of the new ordering process, 347 patients received 614 instances of denosumab, of which 123 (35.4%) received 128 instances of delayed dosing. This is a relative decrease of 17.5% (p=0.09) for the proportion of patients with a late dose.

CONCLUSIONS AND FUTURE DIRECTIONS:
Nearly half of the patients on denosumab in our hospital received delayed denosumab dosing. Delays were often due to a lack of coordination between subsequent dose order placement and referral to the OIC. Our institution successfully implemented a bundled therapy plan to improve timely dosing. By March 2020, we expect to reassess the post-intervention group to further describe reasons for dosing delays. We also will compare rates of VFs associated with delayed denosumab dosing pre- and post-intervention periods.

Cardiovascular Endocrinology
PATHOPHYSIOLOGY OF CARDIOMETABOLIC DISEASE

**PCS9 and Lp(a): Association Between PCS9 Level and Larger Apo(a) Isoform Size in African-Americans and Caucasians**
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**SUN-580**
**Introduction:** An elevated level of lipoprotein(a) [Lp(a)] is an independent causal risk factor for cardiovascular disease. Non-genetic factors do not appreciably influence Lp(a) levels due to a strong genetic control. However, inhibition of proprotein convertase subtilisin/kexin type 9 (PCSK9) has been shown to reduce Lp(a) levels. The association of PCSK9 with Lp(a) level and its major genetic determinant—apolipoprotein(a) [apo(a)] size—is not fully understood. In this study, we assessed the relationship between PCSK9, Lp(a) level, apo(a) size, age, and race/ethnicity.

**Methods:** Healthy Caucasian and African-American families were recruited from the general population (age range: 6–74 years, N=267). PCSK9 and Lp(a) levels were assayed enzymatically; apo(a) isoform and LPA allele sizes and isoform-specific Lp(a) levels were determined.

**Results:** In all participants, PCSK9 levels differed significantly by race/ethnicity, age, and sex. Thus, the mean PCSK9 levels were significantly higher in African-Americans vs. Caucasians (104 ± 29 vs. 95 ± 30 ng/mL, respectively, p=0.020), in adults vs. children (102 ± 29 vs. 92 ± 31 ng/mL, respectively, p=0.001) and in females vs. males (103 ± 30 vs. 94 ± 29 ng/mL, respectively, p=0.007). PCSK9 levels were not associated with total plasma Lp(a) levels neither in all participants nor in ethnicity-specific analyses. However, PCSK9 levels were significantly and positively associated with isoform-specific Lp(a) levels.