post-ACTH response who had a serum cortisol ≥250 nmol/L drawn during the same admission prior to stimulation testing. Cut-offs were based on previous analysis of 195 outpatient stimulation tests.

Results:
During the one-year study period there were 40 inpatients who had an ACTH stimulation test. Nineteen (48%) were considered unnecessary because patients either had a pre-ACTH serum cortisol ≥250 nmol/L and/or a 0-minute cortisol value just prior to the ACTH stimulation test ≥250 nmol/L. Except for a single instance where the patient was inappropriately on prednisone when basal cortisol was tested, all patients with any pre-ACTH cortisol ≥250 nmol/L had a normal post-ACTH response.

Conclusion:
Institutions may avoid unnecessary inpatient ACTH stimulation tests by implementing protocols which ensure that basal cortisol levels are drawn and below locally determined cut-offs before proceeding to dynamic testing. To characterize further, a three-year analysis of inpatient ACTH stimulation tests is underway.

**Pediatric Endocrinology**

**Pediatric Obesity, Thyroid, and Cancer**

**Childhood Diabetic Ketoacidosis (DKA) in New-Onset and Established Patients in Central Illinois: Contributing Factors and Risk Stratification**

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**MON-083**

Introduction: DKA is the leading cause of hospitalizations in children with type 1 diabetes mellitus (T1DM). Although most cases are preventable, DKA continues to occur in established patients.

Aim: To identify contributing factors and outcomes of DKA pediatric admissions in a tertiary referral center with a large rural catchment area to assess for actionable items to prevent DKA.

Methods: A retrospective, single-center chart review assessing children <19 years old admitted to DKA from October 2014 to May 2018. DKA was defined as a pH of ≤7.3 or bicarbonate of ≤15. Demographic data included gender, age, zip code, insurance type and ethnicity. Admission measures included HbA1c, DKA group (new-onset “NT1” or “ET1” established T1DM diagnosis), DKA severity (severe pH <7.1, CO2 <5mEq/L), contact with clinic, home insulin delivery. Outcomes included length of stay (LOS), total admission costs (TAC) and reimbursements amounts (RA).

Results: 272 patients were included (mean age 11.7 y, range 4.4-16; 60% female, 83% Caucasian, 14% African American). Of these, 3% were NT1 DKA. Compared to NT1 DKA, ET1 DKA patients were older (8.7 vs. 13.1 years, p < 0.0001), more likely female (49% vs. 65%, p 0.034) with public insurance (55% vs 63%, p 0.028); 73% didn’t contact the diabetes team prior to admission and 52% used an insulin pump. There were no significant differences in HbA1c or DKA severity.

LOS was similar between NT1 and ET1 DKA (p 0.051). Severe DKA was associated with longer LOS (RR 1.47, p < 0.0001). Public vs. private insurance was associated with 1.28 times longer LOS (p < 0.0001).

While there was no difference in TAC between NT1 and ET1 DKA groups (p 0.877), costs were higher with public vs. private insurance (>900, p 0.050) and severe DKA (RR 1.92; 95% CI 1.62-2.27; p <0.0001). TAC were different between regions within central Illinois (RR 1.39; 95% CI 1.08-1.80; p 0.002).

Hospital RA was higher for NT1 vs. ET1 group (RR 1.26; 95%CI 1.03-1.54; p 0.0237) and higher DKA severity (RR 1.57; 95% CI 1.26-1.95; p <0.0001); but lower for public vs. private insurance (RR 0.43; 95% CI 0.35-0.52; p <0.0001).

Discussion: Established DKA patients tended to be rural teenage females, poorly controlled and public health insured. Severity of DKA and LOS did not differ between the groups. While TAC were similar among the groups, TAC were higher with public insurance and severe DKA. Lower hospital RA were seen for recurrent cases and public insurance. This study provides valuable information about non-metropolitan at-risk population characteristics to inform targeted preventive interventions. These findings suggest a significant difference in hospitalization RA, providing incentive for health care facilities / providers to invest in early outpatient interventions and QI initiatives.

**Diabetes Mellitus and Glucose Metabolism**

**TYPE 1 DIABETES MELLITUS**

**A Case of Hyperglycemia in Post Pancreas Transplant Patient-Is It Insulin Deficiency or Insulin Resistance**

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**SAT-665**

Introduction
Pancreatic transplant (PTx) is a less commonly utilized option for patients with Type 1 Diabetes (DM1), usually with co-morbid end stage renal disease. Hyperglycemia in the post-PTx population can be multifactorial. We report a case of hyperglycemia with ketosis in a post-PTx patient due to insulin resistance and relative insulin deficiency.

Case Presentation
55-year-old male patient with DM1 who underwent simultaneous kidney-PTx. His immunosuppressant (IS) medications were tacrolimus, mycophenolate and prednisone. Tacrolimus levels were therapeutic and his transplant course was benign. He did not require insulin or other glucose-lowering medications post-PTx for 29 years. However, during evaluation for subacute illness with his PCP, he was found to have HbA1c of 12.6%, plasma glucose (PG) 776 mg/dl, and positive urine ketones. He was started on insulin degludec 24 units daily, which was quickly tapered to 8 units daily due to hypoglycemia. OGTT showed fasting PG of 93 mg/dl, 2-hour PG of 115 mg/dl, and stimulated C-peptide of 7.5 ng/ml. HbA1c improved to 7.4% after being on insulin for 1 month. Insulin degludec was tapered and discontinued within 2 months due to hypoglycemia despite...
low doses and robust PTx function. Home blood glucoses (BG) were controlled and HbA1c stable at 6% off insulin therapy. Eighteen months later, he was hospitalized for hyperglycemia with ketosis, without acidosis, during an upper respiratory infection. He was restarted on insulin (glargine, aspart). Once the acute stress resolved, his insulin dose needs reduced significantly, tapering down to insulin glargin 5 units daily. Along with daily home BG monitoring, HbA1c and C-peptide are performed every 3 months, with recent HbA1c 5.2% and C-peptide 3.0 ng/ml (simultaneous PG of 103 mg/dl).

Discussion
Causes of post-PTx hyperglycemia include graft failure due to acute or chronic rejection, insulin resistance, beta cell dysfunction due to IS medications, pancreatitis, or, rarely, recurrence of autoimmunity. Factors predicting hyperglycemia are pre-PTx insulin dose, BMI and acute rejection. New onset type 2 diabetes can occur due to insulin resistance from IS medications and genetic predisposition. Measurement of C-peptide after OGTT can help determine the cause. Insulin is the standard treatment even with detectable C-peptide, though oral glucose-lowering medications have been used in the setting of insulin resistance. In this patient, severe hyperglycemia occurred during stress. He had very low insulin dose requirements when the stress and hyperglycemia resolved and reasonable C-peptide values. This scenario was most consistent with hyperglycemia due to insulin resistance and subsequent relative insulin deficiency during stress, with continued PTx function. This illustrates the importance of detailed assessment and personalized treatment in patients with post-PTx hyperglycemia.

Adrenal
ADRENAL CASE REPORTS II
17-beta Hydroxysteroid Dehydrogenase 3 Deficiency in 1 Month Old Infant
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SUN-159
Background: 17-beta hydroxysteroid dehydrogenase 3 deficiency is a rare autosomal recessive disorder caused by mutations in HSD17B3 encoding the enzyme which converts androstenedione to testosterone. It is characterized in 46, XY males by incomplete virilization, including micropenis and hypospadias.

Clinical Case: We report a 1 month old infant who presented with ambiguous genitalia. Prenatal non-invasive screening showed a Y chromosome, however, fetal ultrasound revealed female genitalia. The infant was born with micropenis (~1.4 cm in length) and proximal hypospadias, with enlarged labioscrotal folds and palpable gonads bilaterally. The urethral meatus had been relocated surgically to the glans. There was an apparent vaginal orifice with a normally positioned anus. Initial testing revealed a normal serum 17-OHP (90 ng/dl, n<200 ng/dl) and normal electrolytes. Abdominal US showed normal kidneys. Pelvic US demonstrated no Mullerian structures; gonads thought to be testes were identified in the labioscrotal folds. At 3 months of age, the infant underwent a 3 day HCG stimulation testing with a borderline testosterone response to 132 ng/dl, androstenedione 78 ng/dl and DHT 25 ng/dl. T/A ratio was unremarkable at 1.7 (n>0.8). Thus, hormonal testing was unsupportive of a testicular steroidogenic enzyme deficiency or androgen insensitivity syndrome. Karyotype was confirmed as 46, XY with microarray evidence of multiple regions of homozygosity. Genotyping with a 46, XY DSD panel (GeneDx) revealed a homozygous pathogenic variant c.608 C>T (p.A203V) in exon 9 of the HSD17B3 gene, consistent with a diagnosis of autosomal recessive 17-beta hydroxysteroid dehydrogenase 3 deficiency. Parents are of Arabic descent and are consanguineous. An older brother was also born with ambiguous genital and was later found to be homozygous for the same mutation. This mutation has been identified in the homozygous state in several unrelated affected patients. Previously published functional studies demonstrated loss of enzymatic activity with this missense mutation (1). Male gender was assigned at birth, and parents wish to continue male sex of rearing.

Conclusion: Molecular genetic analysis utilizing a commercially available candidate gene panel for 46, XY disorders of sex development diagnosed 17 beta-HSD3 deficiency in this case where hormonal testing was not informative. Early and correct diagnosis is key in planning medical treatment to facilitate pubertal development.

References: 1) Geissler et al., 1994 Nature Genetics 7(1): 34-9.

Bone and Mineral Metabolism
OSTEOPOROSIS: DIAGNOSIS AND CLINICAL ASPECTS

SUN-371
Multiple epidemiologic studies have associated chronic hyponatremia with both osteoporosis and bone fractures. Studies in experimental animals and cultured cells have demonstrated that reducing the extracellular sodium concentration ([Na⁺]) causes bone loss primarily by increasing osteoclast formation and bone resorbing activity. In osteoclastic cell cultures, reducing [Na⁺] activated biochemical and functional changes in osteoclast activity, which appear to occur by direct sodium-sensing mechanisms on osteoclasts that are independent from changes in osmolality. Whether the pathological changes in bones induced by hyponatremia can be reversed by correction of hyponatremia has not been studied. The present studies were initiated to address this question. 22-month-old F344BN F1-hybrid rats were made hyponatremic using a desmopressin continuous infusion while fed a liquid diet. After 3 months of chronic sustained hyponatremia, a cohort of the hyponatremic rats were corrected to a normal [Na⁺] by removal of the desmopressin minipumps.