and Puerto-Rican descent. He had difficulties in school and required eyeglasses and hearing aids. Past history included congenital scoliosis (right T11-12 rib fusion, wedged L1 vertebra, and incomplete fusion of posterior elements of L4 and L5), a small right kidney (per ultrasound examination), bilateral orchiopexy for undescended testicles (age 2), diagnoses of ADHD (at age 5); sensorineural hearing loss and psoriasis (age 12), and gastroesophageal reflux (age 13). Multiple paternal family members were reported to have abnormal calcium levels and hearing/vision problems, but no known diagnosis. On exam, he had no facial dysmorphism, but left supernumerary nipples, lumbar lordosis and thoracic kyphosis, and clindactyly. He had achieved Tanner 5 secondary sexual characteristics. There was no Chvostek’s sign. Laboratory investigation revealed Ca 7.9 mg/dL, phosphorus 5.9 mg/dL (3.1-4.7), alkaline phosphatase 123 U/L (50-380), 25-OHD 32 ng/mL, intact PTH 10.2 pg/mL. Treatment with calcium carbonate and calcitriol was begun. Whole exome sequencing identified a heterozygous mutation in GATA3 (c.1061C>T, p.Pro354Leu), predicted to be damaging. This variant has not been reported in literature or public database to our knowledge. Conclusion: This case highlights the importance of genetic testing in the setting of unexplained hypoparathyroidism, and identifies a likely novel mutation in GATA3, providing a basis to counsel the family and encourage medical follow up of suspected family members. References: Barakat, A., et al., Familial nephrosis, nerve deafness, and hypoparathyroidism. J Pediatr 91:61-64, 1977

Diabetes Mellitus and Glucose Metabolism
DIABETES TECHNOLOGY

A Case of the Use of the Eversense Continuous Glucose Monitor with Repeated Same-Pocket Insertion
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SAT-644

Introduction: The Eversense continuous glucose monitoring (ECGM) system is a 90-day implantable, subcutaneous device approved for patients with diabetes. The manufacturer recommends alternating arms for each subsequent insertion site to allow for appropriate healing. To date, there is few data describing use of the same subcutaneous pocket for all subsequent ECGM devices. We present the experience of a patient’s first year use of ECGM with 4 out of 5 device placements within the same subcutaneous pocket. The patient uses an insulin pump and is a concomitant user of Dexcom G5 continuous glucose monitor, allowing us to compare data from his two CGM systems.

Clinical Case: 42-year-old male with a 14-year history of type 1 diabetes, user of the ECGM from July 2018 to date. The first device was placed in his right arm; his second ECGM was placed in his left arm with all subsequent implant replacements in the same pocket, a total of 5 devices, four in the same pocket. Patient baseline glucometric data obtained from his first ECGM device (period 1 [P1]) included an estimated HbA1c (A1c [%]) of 7.06, average glucose (AG [mg/dL]) 156±61, coefficient of variation (CV) 39.1, time in range (TIR [%]) 66.73, time below range (TBR [%]) 2.59, time in serious low (SH [%]) 0.41, time above 180 mg/dL (TA180 [%]) 21.38, and time in serious high, above 250 mg/dL (TA250 [%]) 8.72. Data obtained from subsequent device insertions is described by periods of data time: period 2, 3 and 4 (P2, P3, P4). Glucometric data was as follows for the 3 time periods. A1c was 6.9, 6.9 and 7.1; AG 152±65, 153±64, 157±68; CV 42.7, 41.8, 43.3; TIR 65.7, 65.72, 64; TBR 3.81, 4.13, 3.68; SH 0.93, 0.7, 0.92; TA180 20.1, 20.2, 20.2; TA250 9.3, 9.1, 10.7 respectively for P2, P3 and P4. All four ECGM devices were replaced successfully within the same subcutaneous pocket with minimal bleeding and discomfort. There were no complications, and all devices functioned well, with excellent and constant transmitter signal and longevity. A 1-month overlapping Dexcom CGM data at the end of P2, start of P3 demonstrated A1c of 6.8; AG 144±64; TIR 67.2; TBR 7.5; SH 1.9; TA180 25.3 and TA250 8.2. ECGM data of the same time frame showed A1c of 7.3, TIR of 59.5, TBR 3.6, SH 1.09, TA180 22.1, and TA250 13.5.

Conclusions: Our experience with the ECGM over repeated insertions within the same pocket shows that using this technique is a feasible method for subsequent device implantation. We present one of our patients, user of ECGM, with the longest use of same insertion pocket, demonstrating adequate and constant signal and no complications. The ECGM procedure itself has minimal risk for bleeding, pain, and infection. The use of the same pocket further decreases this risk, by decreasing the number of punctures and procedure time. Consideration for same pocket insertion is proposed to be discussed with the patient, limiting the same pocket insertion to a maximum of one year, based on our clinical experience.

Adrenal

ADRENAL CASE REPORTS I

A Case of Polyglandular Autoimmune Syndrome
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SAT-215

Background: Polyglandular autoimmune syndrome is defined by the presence of Addison’s disease, Autoimmune thyroid disease and Type 1 Diabetes Mellitus.

Clinical Case: This is a case presentation of a 56 year old female with a multitude of endocrine disorders, classified as polyglandular autoimmune syndrome, type 2, persistently elevated ACTH levels.

Over the years, the diagnoses of Primary Adrenal Insufficiency, Type 1 Diabetes, and Hypothyroidism, had revealed themselves, in this patient. Her initial diagnosis upon establishment into our clinic was Addison’s disease and hypothyroidism for which she was getting adequate treatment. Her clinical course had been complicated by multiple admissions for DKA, along with adrenal crises.

Following the adrenal crisis, her ACTH levels had been noted to be persistently elevated, at 3362, despite hydrocortisone replacement at optimal dosing and normal AM cortisol levels. Her hyperpigmentation continued to worsen. A 1mg dexamethasone suppression test failed to lower the ACTH levels.
Concern for a possible ectopic ACTH secretion prompted further investigation with imaging studies such as an abdominal Cat scan which showed no adrenal pathology. Pituitary MRI was ultimately performed which showed no evidence of pituitary lesions. These were followed by an 8mg Dexamethasone suppression test which adequately decreased the ACTH level. However re-check of ACTH levels, after weeks of being on her physiological hydrocortisone dosing, showed that her ACTH levels had started to rise again. Given she had also had multiple admissions for adrenal crises, the concern was raised for possible malabsorption. Given her risk for auto-antibody development, there was concern for another autoimmune process such as Celiac disease, as a potential cause for malabsorption. Her TTG IgA antibodies were checked, however they were absent. At this point, the decision was made to use prednisone as a means of suppression of ACTH, and she was given three days of 40mg Prednisone daily, followed by ACTH level testing, which showed a decrease from 2009 to 708. These results prompted us to change her hydrocortisone to prednisone daily dosing instead, and we converted her to a slightly higher dose of Prednisone. In the setting of underlying DM, this may pose an additional challenge with glycemic control, but we plan for close clinic follow up and repeat ACTH levels a few weeks after she has been on the new prednisone regimen.

**Conclusion:** This is a rare case of a patient with polyglandular autoimmune syndrome, type 2, with a persistently elevated ACTH level, requiring Prednisone, instead of hydrocortisone for treatment of primary adrenal insufficiency in efforts to reduce ACTH levels.

**References:** Neufeld M, Maclaren NK, Blizzard RM. Two types of autoimmune Addison’s disease associated with different polyglandular autoimmune (PGA) syndromes. Medicine (Baltimore). 1981;60(5):355.

### Cardiovascular Endocrinology

#### PATHOPHYSIOLOGY OF CARDIOMETABOLIC DISEASE

**A1AT: Novel Inhibitor of Active PCSK9**

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SUN-574

Heart disease is the principal cause of death and disability for both men and women in the US, accounting for 40% of all annual deaths. African American populations are disproportionately burdened with metabolic diseases, due in part to cholesterol metabolism deficiencies. Elevated low density lipoprotein (LDL) cholesterol levels and inflammation promote atherogenic conditions which lead to heart disease. Proprotein convertase subtilisin/kexin-9 (PCSK9) is a biomarker which enhances atherogenic progression by controlling the number of LDL receptor molecules expressed at the plasma membrane. PCSK9 indirectly regulates LDL-cholesterol levels. Previous reports show some patients do not respond well to general anti-cholesterolemic treatments. We believe this is due to altered PCSK9 activity, which is currently not being evaluated. We have developed a novel assay to detect active PCSK9. A1AT is a SERPIN family member whose primary objective is inhibition of proteases. Specific levels of A1AT are required to maintain metabolic homeostasis. Based on this, we hypothesized that a specific ratio between A1AT serum levels and PCSK9 activity levels would eliminate statin intolerance/resistance, regulating LDL-cholesterol metabolism congruently. Using this novel active PCSK9 detection assay, we provide evidence that A1AT interacts with PCSK9 in the medium of C3A hepatic-like cells, preventing the formation of PCSK9/LDL receptor complexes *in vitro*. There was an approximate 20% inhibition in PCSK9-LDL receptor complex formation when liver cells were treated with recombinant A1AT (rA1AT). A dose dependent response analysis proved 200ng/ml of rA1AT had an 46% reduction in PCSK9 activity. We determined PCSK9 activity and A1AT levels correlate with key diabetic factors in humans, suggesting that A1AT could effect diabetes progression.

### Adrenal

#### ADRENAL PHYSIOLOGY AND DISEASE

**Subclinical Alpha-1 Antitrypsin Deficiency Is Associated with Increased Free Cortisol Fraction in Plasma and Altered Glucocorticoid Delivery to Tissues**

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SUN-221

**Background**

Corticosteroid Binding Globulin (CBG) binds >85% of plasma cortisol and controls the circulating free cortisol pool. Proteolytic cleavage by neutrophil elastase is proposed to reduce CBG binding affinity and increase free cortisol availability to inflamed tissues. The CORtisol NETwork (CORNET) consortium found that genetic variation at a locus spanning *SERPINA1* (encoding alpha-1 antitrypsin, A1AT, the endogenous inhibitor of neutrophil elastase) and *SERPINA6* (CBG) contributes to morning total plasma cortisol variation. We hypothesised that A1AT deficiency increases CBG cleavage and hence free plasma cortisol, resulting in increased tissue cortisol delivery in adipose and in HPA axis negative feedback. We tested this in recall-by-genotype studies of people who are heterozygous for inactivating mutations in *SERPINA1*.

**Methods**

16 healthy carriers of one of the two most common A1AT-deficiency single nucleotide polymorphisms (ra17580 &