show significant rises in gonadotropins. Bone age was advanced by more than 1 year. The patient was started on subcutaneous octreotide with a decrease in IGF-1 to 258 ng/mL after 1 month of therapy. On treatment, linear growth velocity slowed with no interval height gain over the initial 1-month period; however, the patient’s weight continued to increase with a gain of 1.8 kg. Parents additionally reported hyperphagia, which prompted concern for hypothalamic obesity in the setting of her known hypothalamic mass. Thyroid function remained normal on somatostatin therapy. To date, there has been no concern for diabetes insipidus.

**Conclusion:** Growth hormone excess may rarely complicate a diagnosis of NF-1 in the setting of intracranial gliomas. Increased height velocity and/or tall stature for family should raise clinical suspicion and prompt evaluation. Hyperphagia and significant increases in weight in the setting of hypothalamic gliomas in patients with NF-1 should raise suspicion for hypothalamic obesity and prompt lifestyle modifications to curb ongoing weight gain.

### Bone and Mineral Metabolism

**NEW INSIGHTS INTO PTH AND CALCIUM RECEPTOR SIGNALING**

**Identification of the First Case of Acquired Autoimmune Parathyroid Hormone (PTH) Resistance Due to PTH1 Receptor (PTH1R) Autoantibodies**

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**OR07-01**

**Background:** Here we describe a patient who presented with symptomatic hypocalcaemia and a biochemical picture suggestive of PTH resistance. PTH resistance is a hallmark of pseudohypoparathyroidism, a heterogeneous group of rare disorders caused by genetic or epigenetic alterations of PTH/PTHrP signaling. However, PTH receptor-related autoimmune etiology has not been identified as the underlying mechanism for PTH resistance. Here we describe the first case of acquired autoimmune PTH resistance that is secondary to PTH1R autoantibodies.

**Clinical Case:** A 60-year-old African-American woman, who previously had normal calcium homeostasis, presented with acute, symptomatic hypocalcaemia, hyperphosphatemia and markedly elevated serum PTH, consistent with parathyroid hormone resistance. She did not have other hormone resistance or a clinical phenotype suggestive of pseudohypoparathyroidism. Whole-exome sequencing and GNAS methylation analysis revealed no genetic or epigenetic defects of the PTH/PTHrP signaling pathway. Treatment with Calcitriol and Calcium supplements was initiated with good clinical response. Within 10 years of follow-up, the patient developed autoimmune hypothyroidism, alopecia and an unusual form of membranous glomerulonephritis, raising the suspicion for an autoimmune etiology for PTH resistance. Luciferase immunoprecipitation system assay identified antibodies against PTH1R with mapping to the N-terminal extracellular ligand-binding domain (amino acids 1-178). Using an in vitro biological assay in GP-2.3 cells, we found that the antibodies derived from the patient’s serum blocked PTH downstream signaling via G alpha/cAMP/protein kinase A pathway in a concentration-dependent manner.

The patient’s autoantibody profile led to the diagnosis of additional autoimmune diseases, including atrophic gastritis and Sjogren syndrome. Lymphocyte immunophenotyping using flow cytometry revealed an overall normal B and T cell profile, but with decreased frequencies and numbers of switched and non-switched memory B cell subsets and an increased frequency and number of the CD8 naïve cell population. Genes associated with autoimmune inflammatory disorders were sequenced but no pathologic changes were detected.

**Conclusions:** Identification of the first case of autoimmune PTH resistance secondary to PTH1R autoantibodies extends the etiologic spectrum of hypoparathyroidism and should be considered when a patient presents with findings consistent with pseudohypoparathyroidism, especially in the presence of additional autoimmune diseases.

### Pediatric Endocrinology

**PEDIATRIC ENDOCRINE CASE REPORTS I**

**A Novel De Novo GATA3 Gene Mutation in an Adolescent with HDR Syndrome**

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**SAT-065**

**Background:** GATA3 encodes a transcription factor critical for embryonic development of the parathyroid glands, kidney, inner ear, thymus, and the central nervous system. Heterozygous loss-of-function mutations in GATA3 are associated with hypoparathyroidism, sensorineural deafness and renal disease (HDR syndrome). **Clinical Case:** A 12 yo male with left hip pain underwent a closed reduction for left slipped capital femoral epiphysis. The pre-op evaluation revealed hypocalcaemia (serum Ca 7.7 mg/dL; nl: 8.8-10.2), creatinine 0.46 mg/dL (0.5-1.0), TSH 3.16 uU/mL (0.3-4.2), FT4 1.36 ng/dL (0.8-1.8). Oral calcium and vitamin D supplementation was begun, and 2 wks later, follow-up evaluation revealed serum Ca of 9.4 mg/dL, intact PTH 4.6 pg/mL (10-69), phosphorus 5.9 mg/dL (3.3-5.3), 25-OHD 26 ng/mL (30-100), and a normal chromosomal microarray. Bone density (DXA) Z-scores for hip and spine were -1.7 and 0.8, respectively. At age 13 he underwent bilateral osteotomy due to bilateral hip dysplasia and removal of hardware the next year. At age 15 he underwent total hip replacement for avascular necrosis. In the post-operative period hypocalcaemia recurred (5.9-6.7 mg/dL), and he was referred for endocrine evaluation. He was of mixed African American
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 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]) indicated 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 as was 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.