pro-quiescent hormone, and downregulated by E2, the pro-contractile hormone. This data reveals a new mechanism by which NALCN is regulated in the myometrium and may suggest a novel role for NALCN during pregnancy. Further investigation into these novel roles can provide an insight into potential targets to modulate uterine quiescence and contractility.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY AND ADVANCES IN CLINICAL TRIALS

Efficacy and Safety Comparison Between U100 Regular Human Insulin and U100 Rapid Acting Insulin When Delivered by a 24 Hour Wearable Insulin Delivery Device in Type 2 Diabetes

Pablo Mora, MD, David R. Sutton, Jr, MD, Ashwini Gore, MD, FACE, CDE, Bantwal S. Baliga, MD, MRCP, Rebecca Goldfaden, PharmD, CCRP, Carla Nikkel, BS, RD, LD, CDE, CDTC, John Sink II, MPA-C, CDE, Beverley Adams-Huet, MS.

1Dallas Diabetes Research Center, Dallas, TX, USA, 2Northeast Florida Endocrine and Diabetes Associates, Jacksonville, FL, USA, 3Jones Center for Diabetes and Endocrine Wellness, Macon, GA, USA, 4East Alabama Endo, PC, Columbus, GA, USA, 5East Coast Institute for Research, Jacksonville, FL, USA, 6Valeritas, Inc, Bridgewater, NJ, USA, 7Valeritas, Inc., Bridgewater, NJ, USA, 8Independent Statistician, Dallas, TX, USA.

OR30-02

Introduction: Increasing insulin prices have led to a renewed debate to determine if Rapid Acting Insulin (RAI) analogs offer an advantage over less expensive Regular Insulins (RHI). The steep increase in the cost of RAI has led to rationing of insulin or the total discontinuation of therapy by many patients due to cost. For many, RHI provides a more affordable option for insulin therapy when compared to RAI, especially if the limitations of the insulin profile can be overcome by delivering RHI through continuous subcutaneous insulin infusion (CSII) using a wearable insulin delivery device. To our knowledge, no data exists in a type 2 diabetes (T2D) population comparing RAI to RHI when delivered via CSII.

Methods: This 14-week multi-center prospective, randomized parallel, non-inferiority study in a T2D population comparing the efficacy and safety of RAI versus RHI when delivered by V-Go®, a 24-hr wearable patch-like insulin delivery device that provides a preset continuous basal rate of insulin and on-demand bolus dosing. This study was conducted in a real-world practice setting under usual standard of care. Glucose lowering agents were to remain stable unless removal warranted due to documented clinically significant hypoglycemia and the only specific guidance for insulin titration was to down-titrate if blood glucose levels were consistently lower than target range. Patients administering RAI with V-Go were randomized 1:1 to continue RAI or to switch to RHI. Primary endpoint assessed non-inferiority for the between group net difference in HbA1c derived from a mixed model analysis. Between group differences from baseline for insulin total daily dose (TDD) and hypoglycemia (based on 7 point glucose profiles) were evaluated as secondary endpoints.

Results: One hundred thirteen patients (59 RHI and 54 RAI) were evaluated. Baseline characteristics were similar between cohorts. The mean change in HbA1c with RHI was -0.60% from a baseline of 8.41% vs -0.38% from a baseline of 8.33% with RAI (estimated treatment difference [ETD]: -0.22%; 95% confidence interval [CI] -0.67% to 0.22%; non-inferiority margin<0.4% and p=0.007). The mean change in TDD with RHI was 0.8 U/day from a baseline of 61.0 U/day vs 1.8 U/day from a baseline of 61.3 U/day with RAI (ETD: -1.04 U/day; 95% CI: -3.18 U/day to 1.11 U/day; p=0.92). The absolute change in percent of patients reporting hypoglycemia (≤ 70 mg/dL) from pre-randomization to post-randomization was +5.08% with RHI vs + 5.56% with RAI (ETD: -0.48%; 95% CI: -10.6% to 9.1%; p=0.91). Severe hypoglycemia was not reported in either cohort. Conclusion: Patients with T2D administering RAI with V-Go can safely switch to RHI maintaining similar glycemic control.

Thyroid

THYROID CANCER CASE REPORTS I

Poorly Differentiated Thyroid Cancer Arising from a Hyperfunctioning Nodule Treated with I-131

Eli Miller, MD, Jonathan Robert Anolik, MD.

Temple University, Philadelphia, PA, USA.

SUN-478

Thyroid nodules are a common clinical problem with an incidence of up to 1% in men and 7–15% of cases representing thyroid cancer. Current American Thyroid Association guidelines do not recommend cytologic evaluation of hyperfunctioning nodules as they rarely harbor malignancy. We present a case of a hyperfunctioning nodule which years after ablation was diagnosed as a poorly differentiated thyroid cancer.

A 38 year old male had a 4cm thyroid nodule discovered in 1994. Nuclear Medicine (NM) imaging revealed a warm nodule though patient was euthyroid. Biopsy was benign with good sample. Nodule was followed with serial ultrasound (US) and TSH. In 2008 he became hyperthyroid. Scan showed hot nodule and he was given 27.3 mCi I-131 with normalization of the TSH. In 2013 patient again developed hyperthyroidism. NM imaging showed a hot nodule. After 29.5 mCi I-131 he became hypothyroid requiring levothyroxine. Intermittent US showed stability. In early 2019 nodule was 3.7cm, solid and hypoechoic but more heterogeneous. Despite TIRADS recommendation that nodule no longer be followed by US, FNA was performed and revealed Bethesda IV cytology. Gene classification with Thyroseq revealed a TERT mutation. On total thyroidectomy pathology demonstrated a 4.5cm poorly differentiated carcinoma thought to be of follicular origin. Tumor was parietally encapsulated with multiple areas of vascular invasion and extensive tumor necrosis. Tumor was present at inked margin but no extrathyroidal extension was noted. There was a <1mm metastasis noted in 1 peri-isthmus lymph node. One month post operatively thyroglobulin was 123.5 ng/mL. I-123 whole body scan demonstrated bilateral uptake in the region of the thyroid suggesting adenopathy; there were similar findings on FDG-PET scan but no adenopathy was identified on US or the CT portion of the
A 62-year-old male patient with acromegaly due to adrenal cortical carcinoma. We will describe the case of an acromegalic patient with ACTH-independent cushing syndrome caused by adrenocortical carcinoma in an acromegalic patient.

---

**Adrenal**

**ADRENAL CASE REPORTS I**

**Steroid Induced Pheochromocytoma Crisis**

Danielle Eagan, DO, RD, Juan Munoz Pena, MD, Diana Barb, MD.

University of Florida, Gainesville, FL, USA.

SAT-214

**Background:** The factors triggering adrenergic crisis in pheochromocytoma are most often related to induction during anesthesia or manipulation of tumor during surgery. A variety of drugs have also been reported to be associated with adrenergic crisis, however there are only scarce case reports on pheochromocytoma crisis induced by steroids. Here we present a case of steroid induced pheochromocytoma crisis.

**Clinical Case:** A 58-year-old female patient with history controlled hypertension on Lisinopril 10 mg daily, presented to hospital with two day history of fatigue, shortness of breath, and abdominal pain. A CT chest and abdomen showed emphysema and an indeterminate right adrenal gland nodule. Due to patient symptoms and findings of emphysema patient was started on Prednisone 40 mg daily. Home Lisinopril was restarted and patient was placed on sodium restriction. Ten hours after the start of prednisone the patient developed sudden onset hypertension with BP >200/100 mmHg for which she was given labetalol intravenously. The patient developed orthostatic hypotension hours after with BP readings as low as 50s/40s. Due to labile BP a work-up for adrenal adenoma was started. Plasma and urine metanephrines and normetanephrines returned 7–11 times and 4–15 times above upper limit of normal (ULN) respectively. Plasma and urine metanephrines were repeated when patient was off steroids and normotensive and still returned abnormally high (about 2–3 times ULN) along with elevated normally high (about 2–3 times ULN) along with elevated catecholamines (epinephrine 6.5 times ULN and with only a slight elevation in norepinephrine and normal dopamine levels). A dedicated adrenal scan showed a 2.2 cm right adrenal mass with homogeneous enhancement, 130

---

**Neuroendocrinology and Pituitary**

**CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES**

**Acromegaly by Pituitary Adenoma Associated with ACTH-Independent Cushing Syndrome by Adrenal Carcinoma: Case Report.**

Ricardo Kunde Minuzzi, MD, Giulia Menucci Landenberger, MD, Julia Fernanda Semmelmann Pereira Lima, MD, PhD, Miriam da Costa Oliveira, MD, PhD, Carolina Garcia Soares Leães Rech, MD, PhD.

UFCSPA, Porto Alegre - RS, Brazil.

SAT-259

**Introduction:** The coexistence of acromegaly and Cushing’s syndrome is quite rare. Case reports with this association have been described in the literature, including both ACTH-dependent and ACTH-independent Cushing’s syndrome. In these cases, when considering ACTH-independent hypercortisolism, the main etiology reported is adrenal adenoma. We will describe the case of an acromegalic patient with ACTH-independent cushing syndrome due to adrenal cortical carcinoma.

**Clinical Case:** A 62-year-old male patient with acromegaly diagnosed by headache investigation. He had a previous medical history of T2DM for 20 years, grade III obesity (BMI 40.3), hypertension, obstructive sleep apnea and depression. Initial investigation showed IGF-1 levels of 818 ng/mL (81–225), GH: 3.39 ng/mL (<0.97), prolactin diluted: 2.578 ng/mL (2.1–17, 7), LH: <0.07 mIU/mL (1.5–9.3), FSH: 0.6 mIU/mL (1.4–18.1), total Testosterone: 51 ng/dL (241–827) Cortisol at 8 AM: 15 µg/dL, TSH: 1.54 µg/dL (0.55–4.78), free T4: 1.0 ng/dL (0.89–1.76) and brain MRI and suprasellar compressing the optic chiasm, suggestive of pituitary macroadenoma. He underwent transsphenoidal resection with histology confirming a prolactin and GH co-secretory pituitary adenoma with Ki-67: 5%. He started treatment with octreotide LAR (30 mg/month) and cabergoline (3.5 mg/week) and underwent 25 radiotherapy sessions. Three years after the diagnosis of acromegaly, the patient underwent CT scan of the abdomen, which identified a 3.8 cm left adrenal nodular lesion that evolved in the 12-month control exam to nodular image with lobulated contours (5.0 x 3.4 cm) and non-contrast phase density > 25 HU. At that time, he had two 24-hour cortisuloria samples: 640.9 and 637 µg/24hs (54–403) and ACTH <5.0 pg/mL (<46).The patient underwent videolaparoscopic adrenalectomy confirming the pathology of the lesion compatible with adrenal cortical carcinoma with invasion of the capsule and peri-adrenal adipose tissue and Ki-67: 20%. Even after primary resection of the adrenal lesion, the patient evolves with local and metastatic progression of the disease, dying a few months later, due to infectious complications of a new surgical approach.

**Conclusions:** To the best of our knowledge, this is the first case of ACTH-independent Cushing's syndrome caused by adrenocortical carcinoma in an acromegalic patient.