

SAT-467

17 year old female presented for evaluation of galactorrhea of 3 months duration. Patient was diagnosed with papillary thyroid cancer and underwent total thyroidectomy in 2011. She was prescribed Synthroid 175 mcg but was not compliant with taking her medicine. In April 2019 she developed bilateral galactorrhea for which she was evaluated at an outside facility and was found to have a prolactin 143.7ng/mL (n 7.2-63) and TSH 996 mIU/L (n 0.5 -4.0). Pituitary MRI revealed pituitary macroadenoma measuring 1.5 x 1.4 x 1.2 cm with slight elevation of the optic chiasm and infundibulum. She was advised to restart Synthroid and was referred for the neurosurgery team at our facility for surgical resection of pituitary macroadenoma. Over the same period of time, she gained 25 lbs, developed headaches, excessive fatigue, constipation, hair loss, lower extremity swelling, and puffiness of her face. Her menstrual cycles were regular but this was only after she was placed on norelgestromin/ ethinyl estradiol transdermal patch. She denied visual changes. By the time she was seen at our clinic in June 2019, she was taking Synthroid daily for 1 month. TSH decreased to 1.0 mIU/L and prolactin improved to 68 ng/mL. IGF-1 was low at 98 ng/mL (n 149-509). ACTH, morning cortisol, and 24 hour urine free cortisol were within reference range. Visual field testing showed no visual defects. We advised patient to continue taking Synthroid and to follow up in 1 month. On the follow up visit in July 2019, TSH was 0.2 mIU/L, prolactin was 52 ng/mL and IGF-1 was 105 ng/mL. Pituitary MRI showed pituitary hyperplasia that has decreased compared to previous MRI, now measuring around 1 cm at the largest dimension without contact with the optic chiasm and the Infundibulum was at midline. Galactorrhea and headaches resolved and fatigue significantly improved.

Tumor Biology**TUMOR BIOLOGY: DIAGNOSTICS, THERAPIES, ENDOCRINE NEOPLASIAS, AND HORMONE DEPENDENT TUMORS*****Impaired Fasting Glucose Is Associated with Insulin Resistance in Patients with Germline Mutations in the Multiple Endocrine Neoplasia Type 1 (MEN1) Gene***

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BACKGROUND: Multiple endocrine neoplasia type 1 (MEN1) is an autosomal dominant syndrome characterized by hyperparathyroidism, pituitary adenomas, and gastroentero-pancreatic neuroendocrine tumors. Patients with MEN1 mutations have impaired glucose homeostasis, but the role of insulin resistance and beta-cell function is unclear.

METHODS: Using a case-control study design, a retrospective analysis of germline mutation-positive MEN 1 patients

(n=289) seen at our institution between 1991-2019 was performed. Patients with diabetes and/or insulinoma were excluded. Subjects were age, BMI, sex and race matched 1:1 to unrelated, healthy controls. Fasting glucose, insulin, c-peptide, calcium, PTH, 25-OH vitamin D, cholesterol, LDL, HDL and triglycerides (TG) were compared between two groups. Homeostasis model assessment (HOMA-IR) and HOMA-beta cell function (HOMA-b) were used as surrogate measures of insulin resistance and beta-cell function, respectively. Data is presented as mean ± SD.

RESULTS: MEN1 subjects (n=40; age 41±11 years; BMI 29.2±7.2 kg/m²) were matched to healthy controls (age 41±11 years; BMI 29.1±7.5 kg/m²). Only 3 MEN1 patients had no evidence of pancreatic neuroendocrine tumors. Impaired fasting glucose was more prevalent in MEN1 compared with controls (53% vs 10%, p<0.0001). HOMA-IR was positively associated with BMI, but not age, sex, calcium or vitamin D levels in either cohort. HOMA-IR adjusted for age, BMI, and sex was higher in patients with MEN1 compared with controls (4.01 vs. 2.44, adjusted ratio of means 1.54, 95% CI [1.14, 2.07], p=0.005). HOMA-b was not significantly different between the groups (177 vs. 129, adjusted ratio of means 1.17, 95% CI [0.86, 1.58], p=0.23). There were no significant differences in total cholesterol, LDL, HDL, and TG between the groups.

CONCLUSION: Lower insulin sensitivity, but not impaired beta cell function may contribute to the higher prevalence of impaired fasting glucose in MEN1 patients compared with controls. Mechanistic studies into the role of menin loss in glucose homeostasis are warranted.

Neuroendocrinology and Pituitary**PITUITARY TUMORS II*****Desmopressin Test in Silent Corticotroph Pituitary Adenomas, a Preliminary Study - a Comparison with Cushing Disease***

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Introduction: Silent corticotroph adenomas (SCAs) account for 9 to 19% of nonfunctioning pituitary adenomas and behave as the most aggressive pituitary tumors with more invasiveness and high recurrence rate. The identification of these patients during the preoperative stage could predict better surgery results. Some authors refer to high basal ACTH level in the preoperative evaluation as the only marker but until this date, there are no clinical and hormonal markers that could predict its occurrence. The aim of this study is to evaluate the response to desmopressin test and the presence of silent corticotroph tumors.

Patients and methods: Among 475 patients with pituitary lesion, which underwent surgery, 332 were pituitary adenomas, (82 were acromegaly, 40 Cushing disease,