showed a mixed cystic and solid nodule measuring 4.7 x3 x 4cm, with no calcification. Given the increased size of the lesion, her age, and difficulty breathing when supine, a decision was made to proceed with left hemithyroidectomy for definitive diagnosis and treatment. Pathology of the specimen revealed an encapsulated papillary thyroid carcinoma with focal capsular invasion. Right hemithyroidectomy was performed three weeks later, followed by I-131 ablation one month after surgery. The patient is currently doing well and euthyroid on thyroid hormone replacement therapy, with no evidence of disease. She is undergoing surveillance with ultrasound imaging and laboratory evaluation. **Conclusion:** This is a rare case of AFTN harboring papillary thyroid carcinoma. Although the majority of cases of AFTN are benign, an FNA was performed and was negative for malignancy. Due to an increase in size, new symptoms and ultrasound changes, surgery was performed and revealed the final diagnosis. The behavior of thyroid nodules in pediatric patients can be different than adult patients. Even though the majority of AFTN are benign, we should still keep malignancy in our differential when the nodule has a growth pattern, new US findings or patient develops worsening symptoms.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

Melanocortin 4 Receptor Contributes to Glucose Homeostasis by Regulating Kidney Glucose Reabsorption via the Glucose Transporter GLUT2

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MON-639

Melanocortin 4 receptor (MC4R) is essential for normal body weight and food intake. Deficiency of MC4R causes obesity in humans and mice. While the function of MC4R is well established in appetite regulation, its direct role in glucose homeostasis is unclear. Humans and mice with MC4R deficiency exhibit hyperinsulinemia and insulin resistance; however, they remain protected from fasting hyperglycemia/diabetes. To determine the role of MC4R in glucose homeostasis, we performed oral glucose and intra-peritoneal insulin tolerance tests (OGTT / ITT) in male and female *Mc4r* knockout (KO) and wild type (WT) mice. Remarkably, *Mc4r* KO mice exhibited improved glucose tolerance compared to WT mice (Area under the curve for OGTT, male: 29,125±2,028 vs. 38,493±1,161 mg/dL.min; female: 36,322±1,100 vs. 49,539±1,911 mg/dL.min, $p<0.0001$). The improvement in glucose tolerance was despite insulin resistance in *Mc4r* KO mice (Plasma insulin, male: 9.9±1.7 vs. 0.7±0.1 ng/mL, female: 6.2±2.0 vs. 1.1±0.3 ng/mL, $p<0.05$; Area under the curve for ITT, male: 13,174±1,073 vs. 8,132±255 mg/dL.min; female: 13,927±1,253 vs. 7,506±267 mg/dL.min, $p<0.01$). Based on our previous findings from POMC deficient mice, we hypothesized that the improved glucose tolerance in the *Mc4r* KO mice is due to their elevated glycosuria (excretion of glucose in urine). To test this hypothesis, we challenged *Mc4r* KO and WT mice with oral glucose (250 mg) and collected their 24h urine to evaluate glycosuria. Indeed, the KO mice demonstrated elevated glycosuria compared to their WT littermates (Urine glucose, male: 284±48 vs. 0.4±0.03 mg/24h, female: 63.4±14 vs. 1±0.6 mg/24h, $p<0.002$). To assess molecular mechanisms underlying elevated glycosuria in *Mc4r* KO mice, we measured the gene expression and levels of the kidney glucose transporters GLUT1, GLUT2, SGLT1 and SGLT2. *Glut2* mRNA was reduced by~ 40% and the protein level was decreased by~ 20% in *Mc4r* KO mice compared to their WT littermates. The other glucose transporters remained unchanged. Altogether, our study demonstrates that MC4R contributes to glucose homeostasis by regulating kidney glucose reabsorption via GLUT2. These findings may explain why MC4R deficient mice or humans remain protected from diabetes despite their longstanding obesity and insulin resistance.

Neuroendocrinology and Pituitary

CASE REPORTS IN NEUROENDOCRINOLOGY BEYOND THE PITUITARY

Late Diagnosis of ACTH-secreting Pulmonary Neuroendocrine Tumor by Repeated ^{68}Ga Dotatate Pet/ct: Influence of Tumor Size in Abnormal Uptake?

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MON-250

Background: ^{68}Ga DOTATATE PET/CT (^{68}Ga-PET) has been proposed as a superior method in identifying ectopic ACTH syndrome (EAS). However, recent systematic review suggests its sensitivity is not as high as believed (1). We report a challenging case of EAS whose source was uncovered only after repeated ^{68}Ga-PET. Clinical Case: A 15-year-old male presented with rapid onset of typical features of Cushing's syndrome (CS) and metabolic impairment. Hormone evaluation confirmed severe ACTH-dependent CS. Pituitary transsphenoidal surgery was performed due to positive responses in desmopressin stimulation and high dose dexamethasone suppression test, in addition to a 4 mm nodule in pituitary MRI. No tumor was found in surgical specimen and no hormonal improvement was observed after surgery. Inferior petrosal sinus sampling demonstrated no central to peripheral ACTH gradient. Neck US, thorax/abdomen/pelvis CT were negative and PET-CT/FDG was inconclusive. OctreoScan® identified anomalous uptake on left mediastinum and led the patient to a thoracic surgery (TS) with nodule resection at left hilum. Pathology confirmed ACTH positive 10 mm neuroendocrine tumor (NET) infiltrating a lymph node. The patient had transient clinical and hormonal improvement, with recurrence 7 months later. Thoracic CT (T-CT)