anterior pituitary hormone evaluation was normal and the patient was scheduled for endoscopic endonasal resection of a presumed non-functioning pituitary adenoma. Family history was negative for pituitary tumors or hyperparathyroidism. Physical examination was notable for medial deviation of her left eye but neurologic examination was otherwise normal. Laboratory studies were notable for a normal TSH [1.769 uIu/ml (normal: 0.3–5.0)] and low free T4 [0.44 ng/ml (normal: 0.89–1.78)] consistent with central hypothyroidism; an inappropriately normal FSH for a postmenopausal woman [5.6 mIu/ml (normal: 0.3–10.5), and a normal prolactin level [16 ng/ml (0.6–20)]. An 8am cortisol was low at 2mcg/dL (5–21) with an ACTH level of 10 pg/mL (9–46). IGF-1 was normal at 89 ng/mL (41–279). Repeat pituitary MRI imaging demonstrated a homogenously enhancing sellar/suprasellar mass measuring 3.8 cm with displacement of the optic chiasm. Serum IgG4 levels were normal. The patient was started on 50mg IV hydrocortisone every 8 hours for central adrenal insufficiency and levothyroxine 88 mcg daily for central hypothyroidism and underwent an endoscopic endonasal biopsy of the lesion. Surgical pathology was notable for plasma cell-rich lymphohistiocytic hypophysitis and IgG4 plasma cells constituted >40% of the total plasma cell population. The patient subsequently received 1g of rituximab and repeat imaging one week later showed marked improvement in the size and extent of the lesion. The patient was discharged on prednisone and levothyroxine and received a second dose of rituximab at follow-up. The patient reports a decrease in the frequency of her headaches but continues to endorse diplopia.
Conclusion:
IgG4-related hypophysitis typically presents as part of a multifocal systemic process. This case highlights a rare entity of IgG4-related hypophysitis without other features of systemic disease and with normal serum levels of IgG4. Although glucocorticoids are universally regarded as the first line of therapy, an immunosuppressive agent or B-cell depletion therapy such as Rituximab may improve remission and decrease the risk of relapse.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

Hypoglycemia Following OGTT Is More Frequent and Pronounced in CF Compared with Controls

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MON-660

Glucose homeostasis is often abnormal in people with pancreatic insufficient cystic fibrosis (PI-CF). This dysfunction is viewed on a continuum from "normal" glucose tolerance to cystic fibrosis-related diabetes (CFRD), and may also include postprandial and oral glucose tolerance test (OGTT)-related hypoglycemia. This study aimed to delineate the mechanism(s) underlying OGTT-related hypoglycemia. We compared extended OGTT with frequent blood sampling of glucose and insulin in adolescents and young adults with PI-CF [CF(+)] to historical data from a healthy cohort [CF(-)]. We hypothesized that the subset of CF(+) with hypoglycemia would demonstrate 1-hour glucose ≥ 155 mg/dL and impaired early phase insulin secretion (insulin secretion within first 30 min of OGTT). Hypoglycemia [hypo(+)] was defined as plasma glucose <65 mg/dL and was used to assign subjects to exposure groups. We restricted analyses to 180 minutes given available control data. Glucose and insulin incremental areas under the curve (Glc-AUC; Ins-AUC) for 30-minute intervals were calculated. One-hour glucose, nadir glucose, Glc-$AUC_{0\text{-}30}$, and Ins-$AUC_{0\text{-}30}$ and were compared between CF(+) and CF(-) subjects using Student's t-test or Wilcoxon Rank Sum depending upon normality. Participants were 60.5% male, age: 25.4±4.8 years, with BMI-Z: 0.06±0.96kg/m^2 [no differences for CF(+) vs CF(-)]. FEV1%-predicted for CF(+) was 83±21. 69.6% of CF(+) participants self-reported prior episodes of hypoglycemia, 68.7% of whom reported confirmation via glucometer. Hypoglycemia occurred by 180 minutes [hypo(+)] in 15/23 (65%) CF(+) and 5/15 (33.3%) CF(-) subjects (p=0.028). For hypo(+), nadir glucose occurred on average at 180 minutes for both CF(+) and (-). Hypo(+) CF(+) had higher mean 1-hour glucose (197±49mg/dL vs 134±66mg/dL, p=0.035), lower mean glucose nadir (48±7mg/dL vs 61±4mg/dL, p<0.01), and lower early-phase insulin secretion (Ins-$AUC_{0\text{-}30}$: 263±168 versus 650±275 μU/mL, p<0.01) than hypo(+) CF(-). There was no difference in Glc-$AUC_{0\text{-}30}$ for hypo(+) CF(+) vs CF(-). Hypoglycemia is frequent in CF, and is associated with early glucose dysregulation (elevated 1-hour glucose) and compromised early-phase insulin secretion compared to controls with presumed non-pathologic reactive hypoglycemia. The mechanism of hypoglycemia in CF appears to be different than that seen in healthy individuals, and its association with progression to CFRD warrants further evaluation.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Novel CASR Gene Mutation as a Cause of Familial Isolated Primary Hyperparathyroidism

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Background: Primary hyperparathyroidism (pHT) is one of the most common causes of hypercalcemia. About 10%