females and males presented GH, LH, FSH, and TSH levels similar to wild type with reduced PRL levels. At P60, it was observed decreased GH, FSH, and PRL levels for both sexes and TSH levels with no difference from WT, LH secretion was decreased in mutant females and normal in males compared to WT. There was no significant difference in follicular number and classification between mutants and wild type in all analyzed periods, despite the observation of ovarian hypoplasia in the mutants. Johnsen score was in the average of 7.5 in mutant males and 8.8 in WT, with no significant difference between periods. **Conclusion:** Despite the absence of PROP1 and the hypopituitary phenotype, mutant females and males were able to secrete some pituitary hormones, mainly during puberty, a pituitary high demand period. Although the ovaries and testis from mutant mice are hypoplastic, cellular morphology and classification are similar to WT.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Calcium Gone Crazy: Recurrent Hypercalcemia in Patient with History of Hypoparathyroidism

Omer H. Tarar, MD[1], Ambika Rao, MD[2].
[1]University of South Carolina, Columbia, SC, USA, [2]University of South Carolina/ Dorn VAMC, Columbia, SC, USA.

MON-369

Introduction

Patients with post-operative hypoparathyroidism mostly require Calcium supplements and Calcitriol with goal of maintaining calcium levels in low normal range. However, calcium balance can further be dysregulated by other factors including renal failure in patients with otherwise stable levels.

We present a case of recurrent symptomatic hypercalcemia leading to multiple hospital visits in a patient with otherwise stable longstanding post-operative hypoparathyroidism that was precipitated after episode of Acute Renal Failure.

Case Presentation

71 Year old male with past medical history of Atrial fibrillation, Hypertension, Primary Hyperparathyroidism status post Parathyroidectomy (Parathyroid hyperplasia with removal of 3/12 glands) in 2003 with subsequent Hypoparathyroidism presented with lethargy and confusion few days after cardioversion attempt for atrial fibrillation. He was noted to have bradycardia and hypotension post procedure which improved subsequently. His medications included Calcitriol 0.25 mcg twice daily and Calcium Carbonate 1.25 grams twice daily. His Calcium had remained stable over the years on this regimen.

On presentation, he was found to have elevated corrected calcium of 12.5 mg/dl and Acute Renal failure with Creatinine of 8.3 mg/dl which was normal 1 week prior. He underwent hydration and subsequently required few sessions of hemodialysis with improvement in kidney function and further hemodialysis was not needed. On discharge his corrected calcium was 8.6 mg/dl and Creatinine was 3.9 mg/dl. His Calcitriol and Calcium were resumed on discharge. However, 3 weeks later he presented again with lethargy and calcium was again elevated at 14.8 mg/dl. All other workup for hypercalcemia including PTHRP, Bone marrow biopsy, Serum electrophoresis were negative. Subsequently, he had 2 more admissions for hypercalcemia and 1 admission for hypocalcemia within 1 month. Finally his calcium levels stabilized with reduced dose of Calcitriol and Calcium with close weekly monitoring initially as outpatient.

Discussion

In advancing kidney disease, the kidneys are no longer able to increase urine calcium excretion, and this removes an important safety mechanism to prevent calcium excess in patients with Chronic kidney disease. In patients with hypoparathyroidism on Calcitriol and Calcium supplements, renal failure may offset the calcium balance leading to dysregulation and erratic levels with increased tendency towards hypercalcemia.

Conclusion

Acute Renal Failure may lead to hypercalcemia in patients with otherwise stable levels. However, limited understanding of calcium balance/ regulation in renal failure, especially in setting of hypoparathyroidism further complicates the situation and may lead to difficult titration of medications.

Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS

Human Hair Aldosterone Measurements for Evaluation of Primary Aldosteronism

Andrew Paul Demidowich, MD[1], Jancarlos Camacho, BS[2], Maria de la Luz Sierra, PhD[2], Elena Belyavskaya, MD[2], Charalampos Lysikatos, MD[2], Crystal Kamilaris, MD[2], Mari Suzuki, MD[2], Mihail Zilbermint, MD[1], Fady Hannah-Shmouni, MD FRCPC[2], Constantine A. Stratakis, MD, PhD[2].
[1]Division of Endocrinology, Diabetes and Metabolism, Johns Hopkins University School of Medicine, Baltimore, MD, USA, [2]National Institutes of Health, Bethesda, MD, USA.

SAT-543

Background: Primary Aldosteronism (PA) is the most common cause of endocrine hypertension in US. Diagnostic techniques such as a 24 hour urine collection or saline suppression test (SST) can be laborious for both patients and staff. Our group previously showed that human hair cortisol measurements correlated with urine and serum cortisol levels in patients with endogenous cortisol excess. In this study, we explored whether human hair aldosterone correlated with other measures of aldosterone production.

Methods: 41 adult subjects were evaluated at the NIH Clinical Center for adrenal disorders. A pencil-width of hair near the occiput was removed, and the 1cm segment closest to the scalp was analyzed by enzyme immunoassay for aldosterone, reported as pg aldosterone/mg dry hair. Not all subjects underwent complete workup for PA. Data were transformed as necessary to maintain assumptions of normality. Student's t-test and Pearson correlations were used for statistical analysis.

Results: Of the evaluated subjects, 18 were diagnosed with PA, 22 subjects did not have PA, and 1 subject was indeterminate. The mean hair weight was 33.0±13.7mg. For hair

samples weighing greater than 10mg, hair weight was not correlated with hair aldosterone concentration (p=0.40). There was no difference in measured hair aldosterone between the subjects with and without PA (2.01±1.09 vs. 2.52±2.45 pg/mg; p=0.82). Among all subjects, hair aldosterone did not correlate with serum aldosterone (p=0.92), aldosterone-to-renin ratio (ARR; p=0.94), 24 hour urine aldosterone (Ualdo; p=0.85), or the serum aldosterone at the 4 hour time point of a SST (4hrAldo; p=0.98). Serum aldosterone, ARR, Ualdo, and 4hrAldo all correlated highly amongst each other (all p's<0.001).
Conclusions: Hair aldosterone levels do not correlate with other markers of PA. Further work is needed to understand whether optimization of study conditions could improve the usefulness of hair aldosterone measurements in the evaluation of PA.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Silent Presentation of Urinary Bladder Paraganglioma: A Case Report

Johnathan Kirupakaran, DO, Giovanna Rodriguez, MD, Gul Bahtiyar, MD, MPH.
Woodhull Medical Center, Brooklyn, NY, USA.

MON-907

Background: Paraganglioma is a rare extraadrenal, neuroendocrine tumor of the sympathetic or parasympathetic ganglia (1). Bladder paraganglioma accounts for 1% of all paragangliomas and 0.05% of all bladder tumors (1,2). Characteristic presentation of this tumor includes hypertensive crisis and postmicturition syncope (3).
Clinical Case: 35 year old Middle Eastern man with no pertinent past medical history presented with gross hematuria associated with dysuria, purulent penile discharge, urinary frequency, suprapubic pain, and bilateral flank pain for eight months for which he was given a course of an unknown antibiotic in his home country. Social history revealed smoking half a pack of cigarettes/day for 7 years. Has no family history of cancer. On physical exam he had normal blood pressure with mild tenderness to palpation in suprapubic region. U/A was remarkable for hematuria. Patient was treated for UTI with a course ciprofloxacin without resolution of symptoms. Culture returned negative. CT abdomen/pelvis yielded an approximately 1.9 cm enhancing mural nodule involving the anterior aspect of the bladder. Free metanephrine, nor-metanephrine, plasma catecholamine, and urine vanillylmandelic acid were within normal limits. Cystoscopy and transurethral resection of the tumor (TURBT) was completed; a 5-6mm tumor was visualized and resected. Surgical pathology demonstrated tumor growth into the lamina propria and muscularis propria. Neoplastic cells in the sample were diffusely positive for synaptophysin and chromogranin. S100 protein immunostain highlighted sustentacular cells. The diagnosis of paraganglioma was made based on surgical pathology. Post TURP MRI yielded an approximately 1.7 x 0.9 cm transmural hypervascular enhancing mass lesion involving the anterior aspect of the bladder. Given transmural extension of the tumor, an open partial cystectomy was performed. The tumor was resected successfully with resolution of symptoms.
Conclusion: This is an exceedingly rare example of a hormonally silent paraganglioma, mimicking the presentation of a UTI. This case may present the unique consideration of tobacco specific carcinogens as a potential risk factor for the development of bladder paraganglioma.
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Adrenal

ADRENAL - HYPERTENSION

Biochemical Abnormalities in Endocrine Function Associated with Lutetium 177-DOTATATE Therapy in Metastatic Pheochromocytoma and Paraganglioma

Sriram Gubbi, MD[1], Mohammad Al-Jundi, MD[2], Abhishek Jha, MD[2], Marianne Knue, CRNP[2], Joy Zou, RN[3], Jaydira Del Rivero, MD[3], Baris Turkbey, MD[3], Jorge A. Carrasquillo, MD[4], Karel Pacak, MD,PhD[2], Joanna Klubo-Gwiezdzinska, MD,PhD[1], Frank I. Lin, MD[3].
[1]National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD, USA, [2]Eunice Kennedy Shriver National Institute of Child and Human Development, National Institutes of Health, Bethesda, MD, USA, [3]National Cancer Institute, National Institutes of Health, Bethesda, MD, USA, [4]Memorial Sloan Kettering Cancer Center, New York, NY, USA.

MON-214

Background: Lutetium-177 (^{177}Lu) DOTATATE (Lutathera ®) is a form of peptide receptor radionuclide therapy (PRRT) that has shown efficacy in the treatment of neuroendocrine tumors through its action on somatostatin receptor 2. The effects of Lutathera on endocrine function in metastatic pheochromocytoma and paraganglioma (PPGL) has not been evaluated. **Methods:** We performed a prospective analysis on 21 patients (10 female, 11 male) with metastatic PPGL receiving ^{177}Lu DOTATATE (NCT03206060) at our center from July 2017 to August 2019. Hormonal evaluation was obtained 24 and 48 hours after each ^{177}Lu DOTATATE administration and 4 weeks ± 1 week after each cycle to assess for biochemical endocrine abnormalities (BEAs). Blood samples were obtained after 30 minutes of resting with an in-dwelling intravenous catheter. We excluded BEAs that were present either prior to the initiation of ^{177}Lu DOTATATE or due to a pre-existing endocrine disorder. **Results:** We observed BEAs in 18 of 21 (85.7%, 7 female, 11 male) patients. BEAs most commonly involved the pituitary-adrenal axis [ACTH (N: 5-46 pg/mL): 6/21 (28.5%, 5 high, 1 low); serum cortisol (N: 5-25 mcg/dL): 5/21 (23.8%, 2 high, 3 low)], followed by pituitary-thyroid axis [TSH (N: 0.27-4.2 IU/mL): 6/21 (28.5%, 4 high, 2 low); free thyroxine (N: 0.9-1.7 ng/dL): 2/21 (9%, 0 high, 2 low)],