SRLs, (2) treated with Pasi or Peg for active Acro. Patients with concomitant treatments with known action on glucose metabolism were excluded, with the exception of glucocorticoid replacement for central hypoadrenalism. **Results**: 72 pts with active Acro, mean age at study entry 37 ± 15 yrs, 47 females (65.3%). 28 (38.9%) pts were treated with Pasi and 44 pts with Peg (61.1%). Peg was monotherapy in 18 pts (40.9%) and in combo with first generation SRLs for 26 pts (59.1%). The number of pts with IGT and DM2 was superimposable between the 2 groups (Pasi and Peg). In Pasi group, 19 pts had Acro control (67.9%); glucose metabolism worsened in 16 pts (57.1%). Worsening of glucose metabolism occurred most frequently in pts with persistently active Acro (62.5%) and in pts with higher BG and HbA1c values at study start. Similarly, HbA1c was higher in pts with active Acro, although HbA1c worsened during Pasi treatment both in euglycemic and IGT at study entry, regardless of Acro control. In Peg group, 31 pts reached Acro control (73%); glucose metabolism worsened in 12 (27.3%) but improved in 5 pts (11.4%). All pts who experienced glucose metabolism improvement had controlled Acro, regardless of the use of a combo with first generation SRL. Among the 13 pts with active Acro after Peg. BG worsened in 5 cases (38.4%). Moreover, we found that pts with worsening BG control had higher HbA1c (p=0.03) and required higher Peg doses (mean \pm SD 25 \pm 10 mg/day; p=0.04). Patients with higher HbA1c had higher IGF-I, both at study entry and at study end and were treated with higher Peg dose (mean 25 mg/day). Conclusion: Impaired glucose metabolism was more frequent after Pasireotide treatment and in patients of both Pasireotide and Pegvisomant groups with altered pre-treatment glucose and persistently active disease. Therefore, in such acromegaly patients close monitoring of glucose status is recommended during treatment.

Reproductive Endocrinology CLINICAL STUDIES IN FEMALE REPRODUCTION I

Association of Inflammatory Markers with Depressive Symptoms Across the Perinatal Period

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SAT-026

Perinatal depression (PND) is a mood disorder affecting 10-15% of women during pregnancy and postpartum. Its aetiology is complex with contribution from both genetic background and psychosocial as well as environmental stressors that determine individual responses shaped

by chronic and acute disease burden (1). It is thought that the molecular basis of PND involves dysregulation of the HPA axis associated with neurotransmitter and neuroactive steroids imbalance. Inflammation appears to be a contributing mechanism, with increased levels of cytokines exerting adverse effects on serotonin metabolism, neuroplasticity and HPA hyperactivity (2). With only 50% of women detected through current screening strategies, there is an urgent unmet need for the development of biomarker-based strategies to identify women at risk of PND. In this study we used data and blood samples from the prospective Coventry and Warwickshire PND study; we investigated for inflammatory markers associated with depressive symptoms, assessed using the Edinburgh Postnatal Depression Score (EPDS) questionnaire between 24-29 weeks of gestation and again 6-10 weeks postpartum. A cut-off score of 10 categorize 'high' or 'low' risk for depression. Blood samples collected at 28 weeks of gestation were profiled for either IL-6 and IL-10 levels or a panel of 92 inflammatory markers. Individual inflammatory markers were compared across groups using Welch's ANOVA. Results suggest that IL-10 levels were significantly correlated with EPDS score, exerting a protective effect (r= -.10), with reduced levels in the highest severity category (EPDS \geq 15). The IL-6/IL-10 ratio was also associated with a raised EPDS score (r=.10, p=.01), as well as delivery complications (r=.09). The highest IL-6/IL-10 ratio is observed in women who had emergency caesarean section. Bayes' theorem analysis suggested that IL-6/IL-10 ratio could be used as a negative screen to rule out low risk pregnancies. From the 92 inflammatory markers, 14 analytes were below the limit of detection for more than 50% of samples and so were excluded from further analysis. Upon comparison of groups determined by antenatal and postnatal EPDS scores, 29 markers displayed a significance value of P<0.05. Upon the application of post hoc tests, 8 markers including: STAM-BP, SIRT2, CD40, CASP8 and ADA, all associated with apoptotic processes, remained statistically significant in pregnant women with raised antenatal EPDS scores. This data support an association between inflammatory markers and perinatal depression and adverse pregnancy outcomes. Detailed quantitative analysis of such biomarker signatures at different stages of pregnancy, might lead to early detection of disease and application of targeted treatment. (1) Pariante, C. M. & Lightman, S. L. (2008) Trends Neurosci, 31 (9): 464-468. (2) Raison et al., (2006) Trends Immunol, 27 (1): 24-31.

Bone and Mineral Metabolism BONE AND MINERAL CASE REPORTS II

The Mystery of Recurrent PTH-Independent Hypercalcemia with Severe Hypophosphatemia Bilal Shaikh, MD¹, Vishnu Priya Pulipati, MD¹, Garnet Meier,

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MON-343

Background

The differential diagnoses for PTH independent hypercalcemia with hypophosphatemia are broad. Careful

history with systematic evaluation for possible etiologies is necessary for accurate diagnosis and timely therapeutic intervention.

Clinical Case

A 72-year old female was referred to our clinic for evaluation of hypercalcemia with low iPTH level. Review of lab workup over the past two years showed frequent occurrences of mild hypercalcemia, low iPTH and moderate to severe hypophosphatemia. She denied use of calcium, vitamin D, Vitamin A or herbal supplements and over the counter (OTC) medications. Age-appropriate cancer screening was up to date. She had no clinical evidence of granulomatous diseases or malabsorption syndrome. Labs revealed corrected calcium (cCa) 9.9 (8.5-10.5 mg/dL), iPTH 6.36 (12-88 pg/mL), phosphate 1.5 (2.5-4.5 mg/dl), magnesium 1.6 (1.8-2.7 mg/dl), 25-OH Vit D 30 (30-100 ng/mL), PTHrP 12 (14-27 pg/mL), 1,25 OH Vit D 32 (18-72 pg/ml), TSH 1.8 (03-5.6 uIU/mL), GFR 49 (>90 mL/min/1.73 m²) and bicarbonate 30 (23-31 mEq/L). FGF23, 1-mg ODST and SPEP were normal. 24-hour urine phosphate was 3 mg/24h, ruling out renal phosphate wasting.

Two months later, she developed myalgia and generalized weakness. Labs showed cCa 11.3mg/dl, iPTH 3.79 pg/ml, phosphate <1 mg/dl, magnesium 1.2 mg/dl, bicarbonate 33 mEq/L. She was hospitalized for severe hypophosphatemia. Here, she revealed she was intermittently taking a "milky" antacid obtained from Mexico, for her GERD. She required IV phosphate and magnesium replacement and her mineral abnormalities normalized within 24 hours. She was advised to stop use of OTC antacids and provided daily phosphate and magnesium supplementation. 4 weeks later, she developed recurrent hypercalcemia 10.4 mg/dl, hypophosphatemia 1.1 mg/dl and alkalosis with bicarbonate 35 mEq/L, which corrected with supervised phosphate and magnesium supplementation and restricted access to home OTC medications.

Discussion

Milk-alkali syndrome, characterized by the triad of hypercalcemia, metabolic alkalosis and renal failure, has been classically associated with ingestion of large amounts of milk and absorbable alkali. Once a common cause of hypercalcemia, its incidence declined rapidly with advent of new therapies for PUD. Recently, this syndrome, now described as calcium-alkali syndrome (CAS) has re-emerged due to use of high dose calcium carbonate supplements and OTC antacids, with a prevalence of 9-12% among hospitalized patients with hypercalcemia.

We hypothesize our patient's mineral abnormalities are explained by CAS. High doses of calcium from antacids can bind phosphate in the gut, leading to poor phosphate absorption and hypophosphatemia.

Conclusion

CAS is emerging as a frequent cause of hospital admissions for hypercalcemia. Severe hypophosphatemia can be a rare manifestation. Primary therapy is withdrawal of offending agent.

Reproductive Endocrinology TRANSGENDER MEDICINE AND RESEARCH

Evaluation of a 52-Year-Old Transgender Man During the First Year of Testosterone Therapy - Biochemical Changes, Body Composition and Cardiovascular Aspects at the Ergometric Test: A Case Report.

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SUN-053

Introduction: Testosterone (T) therapy is able to promote biochemical, body composition and cardiovascular (CV) changes in transgender men (TM). However, existing data concerns TM between 18 and 50 years old. Objective: To describe the first year of T therapy in a 52 yo TM - biochemical changes, body composition and CV aspects during exercise. Methods: Medical record review was accessed as well as laboratorial and image exams performed during the first year of T use. **Results:** TM, 52 yo, in perimenopause, normal weight, without chronic diseases, no previous usage of T, initiated testosterone undecanoate 1.000mg. A second dose was given 6 weeks after the first one and then every 12 weeks. Lab exams were collected on the day before the next shot of T. By the third month of treatment, it has been noted the highest level of T (586 ng/dL). Initial hemoglobin (Hb) was 13.0 g/dL and hematocrit (Ht) 37.1%. After 7 months of treatment they reached their highest levels, 16.3 g/dL (23%) and 47,1% (27%). LDLc increased from 106 to 139 mg/dL (31%) by the seventh month. HDLc dropped from 73 to 60 (-13 mg/dL) by the seventh month. Initial bone mineral density (BMD) was normal and increased 3.1% in lumbar spine (L1-L4) and 2.7% in femoral neck after 1 year. The muscle mass (MM) increased 10.9% in one year. The ergometric test (ET) at the beginning of treatment showed an increase in systolic blood pressure (SBP) of 38.4% (130 to 180 mmHg) during exertion and a decrease of 27.7% in the third minute of passive recovery; as well as an increase in Heart Rate (HR) of 73 bpm during exertion and a reduction of 72 bpm at third minute of rest. One year after the use of T, SBP increased by 61.5% (130 to 210 mmHg), with a decrease of 52% and after three minutes of rest. HR increased 67 bpm during exertion and decreased by 75 bpm at the third minute of rest. Discussion: According to existing data the increase of Hb in young TM ranges from 4.9% to 12.5% and Ht from 4.4% to 17.6%, whereas in our case it varied 23% and 27%, respectively. The average HDLc drop between 3 and 24 months of T use is, respectively, -6,5 and -8,5 mg/ dL, less than what is found in this report (-13 mg/dL).