87.2 *vs* 154.1 ± 62.6, p=0.0219, respectively). Livoletide-treated participants experienced similar improvements in hyperphagia and food-related behaviors as measured by the HQ whether they were obese or non-obese.

Conclusions: These results highlight the potential of livoletide for treating hyperphagia in both obese and non-obese people with PWS and hyperphagia. Livoletide is being investigated further in the ZEPHYR Phase 2b/3 trial, an ongoing pivotal study on the long-term safety and efficacy of livoletide in the treatment of hyperphagia and food-related behaviors in people with PWS.

Thyroid

THYROID DISORDERS CASE REPORTS II

Sudden Onset of Malabsorption

Gabriela Mroueh, MD[1], James K. Burks, MD[2].
[1]Texas Tech University at the Permian Basin, Odessa, TX, USA, [2]Texas Tech Univ Hlth Sci Ctr, Odessa, TX, USA.

SAT-509

Celiac disease (CD) is an immune-mediated enteropathy caused by a reaction to gliadin which responds to a restriction to dietary gluten. It has been traditionally recognized in children and young adults, although, recently, detection in the elderly population has increased. CD occurs in 2–5% of patients with autoimmune hypothyroidism, and is more prevalent in this group than in the general population

An 82-year-old Caucasian woman with primary hypothyroidism and a BMI of 16 is referred to our endocrinology clinic for help with the management of hypothyroidism. She had a history of well controlled hypothyroidism on weight-dosed levothyroxine for many years until several months prior when she developed sudden onset of diarrhea and weight loss. Since then, her thyroid function tests showed an elevated TSH despite medication adherence. Her levothyroxine dose was steadily increased to 300 mcg daily and yet, her TSH still remained elevated. Laboratory work up was done which revealed elevated transglutaminase antibodies, suggesting the diagnosis of CD. The patient refused an endoscopy for a tissue diagnosis. Even though the patient has been diagnosed with CD, she has trouble following a gluten free diet and still has intermittent diarrhea and high levothyroxine requirements.

Although lack of medication adherence is common, it is important to exclude gastric or intestinal causes of malabsorption in patients with high thyroid replacement requirements. Elderly patients often have paucity of symptoms, so high clinical suspicion is necessary to diagnose these patients.

Thyroid

THYROID DISORDERS CASE REPORTS I

A Challenging Diagnosis of Thyrotoxic Periodic Paralysis

Gabriela Mroueh, MD[1], Ramachandra Rahul V. Chemitiganti, MD[2].
[1]Texas Tech University at the Permian Basin, Odessa, TX, USA, [2]Texas Tech University Health Sciences Center School of Medicine, Midland, TX, USA.

SUN-496

A Challenging Diagnosis of Thyrotoxic Periodic Paralysis

Thyrotoxic Periodic Paralysis (TPP) is a rare but potentially lethal manifestation of hyperthyroidism which is characterized by muscular weakness due to intracellular shift of potassium and subsequent hypokalemia. The muscular weakness may range from mild weakness to complete flaccid paralysis. It is predominantly seen in Asian young men. Graves' disease has been described as the most common cause of TPP. Other rare causes of hypokalemic periodic paralysis include inherited disorders and acquired cases due to drug abuse, specifically cocaine. It is important to recognize and diagnose TPP to provide appropriate treatment and prevent serious cardiopulmonary complications.

A 26 year old Hispanic male with past medical history of cocaine abuse presented to the emergency department with profound lower extremity weakness since that morning. Laboratory studies on initial evaluation revealed hypokalemia. He was admitted to the intensive care unit (ICU) for IV potassium replacement and cardiac monitoring. Upon obtaining further history, the patient had suffered a similar episode of weakness and hypokalemia two months prior. At the time, he had a positive urine toxicology for cocaine. He was treated with IV potassium with resolution of his weakness and was told the reason for the episode was cocaine induced periodic paralysis. No further work up was done due to patient leaving Against Medical Advice. The patient stopped recreational drug abuse after this diagnosis.

During current hospitalization, further laboratory studies revealed hyperthyroidism. TSI and TPO antibodies were elevated and thus patient was diagnosed with Graves' disease. On questioning, patient was asymptomatic and clinically euthyroid. He was treated with IV potassium, methimazole and propranolol with quick resolution of weakness. He has been followed in an out-patient basis and he has had no further exacerbations.

In this case, we present a case of TPP that was initially diagnosed as cocaine induced periodic paralysis which is an extremely rare disorder with only a couple of described cases in the literature. Diagnosis was initially missed as the patient was clinically euthyroid and had history of recreational drug abuse. Restoration of euthyroidism eliminates attacks of TPP. It is important to recognize and diagnose these patient to prevent further attacks.

Neuroendocrinology and Pituitary

ADVANCES IN NEUROENDOCRINOLOGY

Role of Activin, Follistatin, and Inhibin in the Regulation of KISS-1 Gene Expression in Hypothalamic Cell Models

Aki Oride, MD, Haruhiko Kanasaki, MD,PHD, Zolzaya Tumurgan, MD, Tuvshintugs Tumurbaatar, MD, Satoru Kyo, MD, PhD.
Shimane University, Izumo, Japan.

SUN-244

Kisspeptin (encoded by the Kiss-1 gene) in the arcuate nucleus (ARC) of the hypothalamus governs the hypothalamic-pituitary-gonadal (HPG) axis by regulating pulsatile release of gonadotropin-releasing hormone (GnRH). Meanwhile, kisspeptin in the anteroventral

periventricular nucleus (AVPV) region has been implicated in estradiol (E2)-induced GnRH surges. Kiss-1-expressing cell model mHypoA-55 exhibits characteristics of Kiss-1 neurons in the ARC region. On the other hand, Kiss-1 expressing mHypoA-50 cells originate from the AVPV region. In the mHypoA-55 ARC cells, activin significantly increased Kiss-1 gene expression. Follistatin alone reduced Kiss-1 expression within these cells. Interestingly, activin-induced Kiss-1 gene expression was completely abolished by follistatin. Inhibin A, but not inhibin B reduced Kiss-1 expression. Activin-increased Kiss-1 expression was also abolished by inhibin A. Pretreatment of the cells with follistatin or inhibin A significantly inhibited kisspeptin- or GnRH-induced Kiss-1 gene expression in mHypoA-55 cells. In contrast, in the mHypoA-50 AVPV cell model, activin, follistatin, and inhibin A did not modulate Kiss-1 gene expression. The subunits that compose activin and inhibin, as well as follistatin were expressed in both mHypoA-55 and mHypoA-50 cells. Expression of inhibin βA and βB subunits and follistatin was much higher in mHypoA-55 ARC cells. Furthermore, we found that expression of the inhibin αsubunit and follistatin genes was modulated in the presence of E2 in mHypoA-55 ARC cells. The results of this study suggest that activin, follistatin, and inhibin A within the ARC region participate in the regulation of the HPG axis under the influence of E2.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Severe Postoperative Encephalopathy in a Patient with Oncogenic Osteomalacia and Neurofibromatosis Type 1

Sauradeep Sarkar, MD[1], Amteshwar Singh, MD[2], Esther Irina Krug, MD[1].
[1]Sinai Hospital of Baltimore, Baltimore, MD, USA, [2]Johns Hopkins School of Medicine, Baltimore, MD, USA.

MON-364

Background: Acute hypophosphatemia superimposed on chronic phosphate depletion results in decline in intracellular ATP and 2,3-diphosphoglycerate levels, affecting virtually all organ systems. It can lead to metabolic encephalopathy, impaired myocardial and diaphragmatic contractility, skeletal and smooth muscle dysfunction, increase in erythrocyte rigidity leading to hemolysis, and increased risk for postoperative morbidity and mortality. Risk of symptomatic hypophosphatemia may be greater in patients with urinary phosphate wasting syndromes hospitalized for intercurrent illness. Tumor-induced (oncogenic) osteomalacia (TIO) is a rare paraneoplastic disorder associated with mesenchymal tumors caused by tumor production of fibroblast-derived growth factor-23 (FGF-23). FGF-23 impairs transport of phosphorus in renal tubules, inhibits renal 25(OH)-1-α-hydroxylase activity, causing decreased levels of calcitriol, leading to hyperphosphaturia, hypophosphatemia and osteomalacia. We present a case of a patient with underlying TIO who presented with acute hypophosphatemia following hip surgery.

Case: 50-year-old woman with Neurofibromatosis type-1(NF-1) complicated by TIO, presented after a fall resulting in left femur fracture requiring surgery. During her initial management in an outside hospital for 4 days and following transfer her home calcitriol and phosphorus replacement regimen was not administered leading to treatment interruption of 7 days. On postoperative day 1 she became hypotensive, tachycardic with non-specific ST-T changes and encephalopathic requiring ICU care. At the time of endocrinology consultation, she was non-verbal, not following simple commands, had severe hypophosphatemia, transaminitis, hyperbilirubinemia, elevated LDH, anemia and elevated troponin. She required aggressive phosphate and calcitriol replacement reaching 4 doses of 20mEq IV Na- Phos daily, Na-KPhos Q6H and Calcitriol IV 0.5mcg Q8H over the course of 72 hours. After 4 days in the ICU, once phosphorous levels normalized, she had dramatic improvement in mental status and gradual resolution of laboratory abnormalities.

Discussion: To our knowledge, this is the first report of a patient with TIO associated with NF-1 presenting with severe symptomatic hypophosphatemia causing metabolic encephalopathy and multiple organ dysfunction following surgery. It underscores the fact that abrupt interruption of calcitriol and phosphorus supplementation in such cases can cause acute hypophosphatemia carrying high morbidity. High phosphate replacement requirements are indicative of severity of phosphate depletion. This case illustrates importance of recognition of presence of urinary phosphate wasting syndrome in hospitalized patients in order to minimize risk of preventable complications and morbidity.

Bone and Mineral Metabolism

OSTEOPOROSIS: DIAGNOSIS AND CLINICAL ASPECTS

Potential Impact of Using Male vs Female T-Scores for BMD Classification in Men

Raleigh A. Fatoki, MD, MBA[1], Bruce Ettinger, MD[2], Malini Chandra, MS MBA[2], Kate M. Horiuchi[2], Joan Chia-Mei Lo, MD[2].
[1]Kaiser Permanente, Oakland, CA, USA, [2]Kaiser Permanente Division of Research, Oakland, CA, USA.

SUN-383

BACKGROUND: Osteoporosis is traditionally associated with post-menopausal women, but up to up to one-third of osteoporosis-related fractures occur in elderly men. The International Society for Clinical Densitometry (ISCD), the World Health Organization, and the Fracture Risk Assessment Tool (FRAX) all recommend using a white female reference for BMD T-score for men. However, in clinical practice and previous clinical trials, a sex-specific white male reference T-score is used. This report examines the implications of using a female versus male reference for T-score calculation in men.

METHODS: We reviewed BMD findings in 703 men (age 70-85y) who experienced a proximal femur, humerus, or distal radius/ulna fracture. For this cohort, femoral neck BMD was used to calculate a BMD T-score using either the young adult male and young adult female peak values (mean BMD 0.930 ± 0.136 and 0.849 ± 0.111 g/cm2, respectively). Osteoporosis was defined by BMD T-score ≤ -2.5, and osteopenia by BMD T-score < -1.0 and > -2.5. We also calculated FRAX-estimated fracture risk for hypothetical men