was significant for acute kidney injury (cr 11.33 mg/dl, n: 0.7–1.5 mg/dl) and CPK 171920 u/l (n: 22–269 u/l). He received 2 L of NS in the ED and was started on NS at 100cc/hour. He underwent hemodialysis on day 2; initially he was treated for hypocalcemia with calcium and vitamin D supplementation until day 11 were hypercalcemia (calcium 12.7 mg/dl, n: 8.7–10.3 mg/dl; ionized calcium 1.7 mmol/l, n: 1.12–1.32) was noted; this was associated with concomitant suppression of PTH (5 pg/ml, n: 10–65 pg/ml). He remained asymptomatic from calcium abnormalities during his hospitalization, his urine output recovered progressively, hemodialysis was discontinued on day 13. Upon discharge was recommended to f/u with nephrology.

Discussion: Various neurological and neuromuscular complications of heroin abuse have been described; one of these is rhabdomyolysis; its pathophysiology in heroin abuse is thought to be multifactorial; including acidosis, hypoxia, muscle compression and adulterants found in heroin. Narcotics may also have direct cell toxicity and alter membrane transport. Usually upon initial presentation hypocalcemia is one of the most common electrolyte imbalances seen with rhabdomyolysis. The proposed mechanism is precipitation of serum calcium salts in necrotic muscle. This may be followed by hypercalcemia during the diuretic phase of ARF which appears to be a relatively unusual complication associated with the presence of severe muscle damage due to metastatic calcium salts that are liberated from the necrotic muscle and the return to the serum.

Conclusion: This case report highlights the importance of recognizing potential electrolyte imbalances in patients with rhabdomyolysis; it appears, that concomitant rhabdomyolysis and ARF are needed for a patient to develop hypercalcemia.

Serum calcium should always be routinely measured and the appropriate treatment should be implemented to improve outcomes.

Cardiovascular Endocrinology
HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

A Rare Case of Laboratory Hypertriglyceridemia: Glycerol Kinase Deficiency

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SAT-578

Background: Hypertriglyceridemia (HTG) is common; however, pseudo-HTG due to high glycerol in glycerol kinase deficiency (GKD, MIM: 307030) is a rare cause of HTG that need to be delineated for appropriate management. GKD is an X-linked recessive disorder characterized by hyperglycerolemia and glyceroluria. Two of three GKD subtypes are known as "isolate" GKD due to a mutation in *GK* gene alone: (1) symptomatic juvenile form, and (2) benign adult form, associated with an incidental finding of HTG.

Since most commercial laboratories determine triglyceride (TG) levels by a glycerol measurement, TG-backbone, patients with GKD are mistakenly labelled as having HTG. Glycerol-blanking is required to reveal the actual TG, but it is costly. Since usual TG-lowering medications are ineffective or even harmful, novel methods to screen for individuals with GKD or pseudo-HTG are necessary.

Objective: Through identification of a clinical case of GKD that was diagnosed by glycerol-blanking, we are proposing two potential methods to screen for pseudo-HTG, and presenting their reliability.

Methods: The patient was recruited into an IRB-approved study investigating etiologies of dyslipidemia at the University of Pennsylvania. Patient provided consent for medical record review.

Results: A 49-year-old man was referred for HTG management. His reported TG levels ranged between 490 and 559 mg/dL without any other adverse lipid levels for several years without a history of pancreatitis or diabetes mellitus. Intriguingly, he reported a family history of HTG.

Since TG-lowering medications (fibrates and fish oil) had not reduced his TG levels, specialized lipid analyses were obtained: a non-blanked TG level of 521 mg/dL and a glycerol-blanked TG of 66 mg/dL, consistent with pseudo-HTG or hyperglycerolemia. Repeat glycerol blanked TG levels were 68 and 69 mg/dL, confirming the previous result, and the likely diagnosis of GKD.

With two methods, estimated TG levels were calculated, using some of his laboratory values: (1) modified Friedewald equation to solve for TG with a direct LDL (dLDL) value, and (2) the application of a newly developed formula derived from a collection of 17,545 patient samples, to calculate the absolute TG-gap, using apolipoprotein A and B, estimating TG levels (% deltaTG), and determining whether a TG mesurement might be falsely deviated from the "plausible" TG value.

Although neither methods showed perfect concordance, the calculated TG-valued derived by the two methods were significantly lower than the non-glycerol blanked TG values. The difference was statistically significant ($p<0.05$).

Conclusion: The patient was clinically diagnosed with GKD, and was taken off of fibrate and the recently added niacin. These two methods can be used quickly to screen for pseudo-HTG or patients with GKD. Currently, it is unknown whether high glycerol levels are associated with high cardiovascular risks.

Pediatric Endocrinology
PEDIATRIC GROWTH AND ADRENAL DISORDERS

Response to RHGH Therapy in Children with Isolated Short Stature with or Without an Identified Genetic Cause

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SAT-096

Introduction: Children with isolated (former known as idiopathic) short stature (ISS) have been treated with rhGH with a variable response. **Objectives:** To evaluate the short-term response to rhGH therapy in children with ISS with or without a genetic diagnosis. **Methods:** We analyzed retrospectively the growth rate and height SDS change in the first year of rhGH treatment according to the presence or absence of defects in genes that regulate growth plate. The decision to start rhGH treatment was based on clinical features and the genetic results were obtained during the follow-up. Patients were enrolled in several previous genetic studies using gene candidate approach or multigene sequencing analysis. **Results:** A total of 51 prepubertal children (36 boys) with ISS were treated with rhGH. Thirteen of these children start puberty during the treatment and three of them were concomitant treated with GnRH analog. Basal characteristics of these children were 7.7 ± 3.2 years of age, height SDS -2.5 ± 0.8; sitting height/height (SH/H) SDS 1.2 ± 1.4; BMI SDS 0 ± 1.0 and mild delay of bone age (-1.6 ± 1.3 y). The mean target height SDS was -1.2 ± 0.9y, 18 (35%) of these children have at least one parent with height SDS < -2 and 3 (6%) both parents are short. Consanguinity was present in 3 (6%) cases. Among this cohort, fifteen children had pathogenic or likely pathogenic allele variants in genes that regulate growth plate: *IHH* (n = 4), *SHOX* (n = 9) and *NPR2* (n = 2). Seven (47%) of these variants were inherited from a short stature parent. Children with or without an identified genetic cause have similar age and height SDS at the start of the treatment. A higher BMI and SH/H SDS were observed in children with genetic defects than in those without (BMI SDS 0.5 ± 1.1 vs. -0.15 ± 0.9, p = 0.02; SH/H SDS 2.0 ± 1.4 vs. 0.9 ± 1.3, p = 0.006). Additionally, children with genetic defects had a less marked bone age delay (-1.0 ± 1.3 vs. -1.9 ± 1.2; p = 0.02). Both groups were treated with similar rhGH dose (50 µg/kg/day). Patients with and without an identified genetic cause had similar improvement in growth velocity during the first year of therapy: 4.8 ± 1.6 to 8.9 ± 1.7 cm/y for patients with molecular diagnosis vs. 4.6 ± 1.2 to 8.5 ± 2.3 cm/y for those without. This resulted in similar height SDS change during this period for both groups (0.6 ± 0.3 vs. 0.6 ± 0.5 SDS for children with or without a genetic cause, respectively). Age at the start of treatment was the main variable that explains growth response variability during this first year (r^2 = 0.17, p = 0.009). **Conclusion:** The presence or absence of an identified genetic cause, involving genes that regulate growth plate, did not significantly influence the short-term growth response to rhGH therapy of children with ISS. Long-term follow-up is still needed to assess the final height of these children and possibly to assess whether there is a different growth rate related to each known affected gene.

Bone and Mineral Metabolism
CLINICAL ASPECTS OF OSTEOPOROSIS AND VITAMIN D ACTION

Implementation of an Osteoporosis Risk Assessment Instrument (ORAI) to Increase Referral Rates for DEXA Scanning in the Primary Care Setting

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MON-391
Abstract

Background/Purpose: Osteoporosis (OP)was first identified and named by healthcare professionals in the 18th century. Today, OP is still the source of fractures which impair mobility, leading to sub-acute stays at rehabilitation centers. A major obstacle is that primary care providers (PCPs) fail to identify warning signs of OP, and inform patients that Dual Energy X-Ray Absorptiometry (DEXA) scans that are one of the best procedures to assess bone health. This project addressed the issue of low rate of referrals for DEXA scans**. Theoretical Framework:** The Knowledge-to-Action (KTA) model was used to guide this study. **Intervention:** Implementation of osteoporosis risk assessment instrument. **Methods (Design, Sample, Setting, Measures, Analysis):** This includes pre-implementation phase, patients' charts were reviewed; post-implementation phase, the number of people referred to have DEXA scans were analyzed; the evaluation phase, results compared to the previous data. The project focus exclusively on women and men ages 50 to 89 years in two primary care offices in New Jersey. Descriptive analyses concentrated on whether or not ORAI was the tool to increase DEXA scans. **Results:** The data analysis reflected that the baseline referral rates increased from 1.3 % to 42 % and patients who scored high on the risk assessment instrument have been referred more often than not. Moreover, patients who are at risk and younger than 65 years of age, risk assessment tools led to a positive referral for a DEXA scan. Those who are older than 65 years, risk assessment tools like ORAI should be given with fracture risk assessment tools. This is especially the case when dealing with men, a demographic group often overlooked in the fight against OP. **Conclusions Implications:** If this project is to be applied at other clinics, more and more patients would be referred, raising awareness of the medical benefits of early detection. Reasonably, covering a broader section of patients, earlier in their lives, will increase clinical income, bringing more patients to primary care offices.

Adipose Tissue, Appetite, and Obesity
ADIPOSE TISSUE BIOLOGY AND OBESITY

Uc.336-As Inhibits White Adipocyte Differentiation and Promotes White to Brown Conversion

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SAT-584
Brown adipose tissue (BAT) has gained its popularity since it shows great potential in counteracting obesity and metabolic diseases development. Transcribed ultraconserved regions (T-UCRs), a novel class of long non-coding RNA (lncRNAs), have been implicated in regulating diverse biological processes, including the process of white fat browning. However, the functional and mechanistic details of T-UCRs in the browning process are poorly understood. Here, we identified that a T-UCR, uc.336-as, played an