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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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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