MON-131
Background: Unlike with solid organ cancers, little data is available on how diabetes mellitus (DM) and hematologic malignancies interact to affect survival and glycemic control. We examined the impact of DM on survival in patients with leukemia and the effect of leukemia on glycemic control. Materials and Methods: Patients with leukemia with and without DM were matched 1:1 (2007–2017), 70 matched pairs (total N=140 pts) were included in the analysis. We compared characteristics between cases and controls and assessed survival with the Kaplan-Meier method and Cox proportional hazards model. Mixed models compared hemoglobin A1c (HbA1c) and glucose levels over time. Results: The median age of patients at diagnosis was 56 (18–94); 60% were male and 89% had acute leukemia. Among those with DM, HbA1c was only recorded in 25 of 70 patients during the year following cancer diagnosis and was 6.8%. There was no change in HbA1c values over time in these DM patients. Mean glucose was significantly different between DM and non-DM patients (p<0.001). Time (days since leukemia diagnosis) was significant (p<0.001) and there was a significant interaction effect (p=0.01). Glucose values increased in the DM patients during the year following diagnosis, while remaining stable in those without DM. Median follow-up time was 23.2 months. Three-year survival was estimated at 46% for DM patients versus 45% in non-DM pts by Kaplan Meier method (p=0.79). Hazard ratio (stratification for matched pairs) was 1.05 (95% CI: 0.57 - 1.94; p=0.88). Three-year relapse-free survival was estimated at 34% for DM patients versus 43% for non-DM patients (p=0.58). Hazard ratio (stratification for matched pairs) = 1.10 (95% CI: 0.61–1.98; p=0.76). Conclusions: DM did not adversely impact survival in patients with leukemia. Leukemia and its treatment did not affect glycemic control. This should be reassuring to hematologists and endocrinologists who treat patients with leukemia and diabetes.

Thyroid
THYROID CANCER CASE REPORTS II
False Positive I-131 Uptake
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MON-437
Background
Iodine has high specificity for thyroid tissue hence it plays very important role is management for differentiated thyroid cancer. Despite its high specificity and sensitivity, false-positive I-131 uptake could be seen on whole body scan (WBS).

We are presenting a case of false positive intense uptake in lung due to bronchiectasis.

Clinical case
Patient is a 78-year-old Asian female who initially present with left sided 5.8 cm thyroid nodule. The fine needle aspiration was performed, and the cytology came back as Bethesda category III. Per our institute protocol a molecular mutation panel was sent, which came back with NRAS mutation.

She underwent total thyroidectomy and the histology showed 6.2 cm Follicular carcinoma with extensive angioinvasion, oncocyte type. She received adjuvant 165.2 mci of RAI. Per protocol she had a one-week I-131 whole body scan.

There was intense abnormal uptake in left mid and upper chest. The stimulate thyroglobulin with a TSH of >100mcIU/ml was only 0.17 ng/dl with Tab negative.

Patient subsequently had a PET CT which showed a faint diffuse FDG activity noted in the cystic bronchiectasis predominantly in the left apex, lingula, and right middle lobe.

Patient informed us that she has history of pulmonary tuberculosis in 1970’s for which she was successfully treated in her home country. We had 10-year-old chest X-ray which showed stable cystic bronchiectasis lesion in the region of intense uptake.

Discussion
Although I131 whole body scan has high specificity and sensitivity but physician should be aware of potential false positive uptake to avoid unnecessary intervention. In a retrospective evaluation the most common non-thyroid conditions included were bronchiectasis, lung infection, subcutaneous injection into gluteal fatty tissue, aortic calcification, benign bone cyst, vertebral hemangioma, recent nonthyroidal surgical procedure site, rotator cuff injury, mature cystic teratoma and ovarian follicle cyst). The accumulation of the bronchial secretion is the proposed mechanism of the high false positive uptake in bronchiectasis.

Endnotes
1 Mol Imaging Radionucl Ther 2018; 27:99–106 DOI:10.4274/mirt.37450

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS I
A Case of Hypercalcemia Secondary to Rhabdomyolysis Induced by Heroin Use
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SAT-372
Background: Rhabdomyolysis is a potential life threatening condition defined as injury of the skeletal muscle, which results in the release of intracellular contents into the circulation. This muscle injury is often associated with the development of myoglobinuria, electrolyte abnormalities, and often ARF; it can be caused by diverse mechanisms including drugs and toxins. We present a case of rhabdomyolysis complicated by hypercalcemia and ARF in a patient with a history of polysubstance abuse after using intravenous heroin.

Case: A 28 y/o male with h/o polysubstance comes to the ED with c/o fatigue, nausea, vomiting and decreased urine output for about 5 days; patient acknowledged using heroin and afterwards he developed weakness and tenderness in upper and lower extremities. Additionally he admitted cocaine and marijuana abuse. On physical exam vital signs were unremarkable and he had some mild tenderness to palpation mostly of the quadriceps bilaterally. Laboratory data
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 be 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 hypercalcemia 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.

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**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 “isolated” 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 (fibates 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 measurement 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.

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**Pediatric Endocrinology**

**PEDIATRIC GROWTH AND ADRENAI DISORDERS**

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

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