should be initiated based on clinical suspicion without waiting for laboratory results.

We report a case of a 71-year-old female with a history of CAGB 10 years ago, who presented to the emergency department with a syncopal episode. The patient denied aura, vomiting, jerky movements, rolling eye movements, bowel/bladder incontinence, and palpitations. She reported having exertional chest pain for a couple of months. Her home medications included metoprolol succinate 50 mg daily, hydralazine 50 mg twice daily, Imdur 30 mg daily, and levothyroxine 300 mcg daily which the patient reported taking rarely. On admission, the temperature was 36°C, HR 43, BP 75/49 which dropped to 60/40, RR 9, and SpO2 of 93% on 2LPM. Physical exam showed euvoeema with brisk reflexes and delayed relaxation. Blood work revealed Na of 133 mM/L (L), Cr 6.4 mg/dL (H) from a baseline of 2.4 mg/dL, and a troponin of 0.26 ng/mL (H). EKG showed first degree AV block with new T-wave flattening not seen on previous EKGs. A fluid challenge for hypotension elicited no response and the patient was started on a dopamine drip for cardiogenic shock, a heparin drip for ACS, and was transferred to the CCU. BP improved to 140s/70s with a HR remaining in the 50s. On the 2nd day of admission, blood work revealed a TSH of >150 (H), free T4 of 0.6, total T3 of 34 (L) and a random cortisol of 15.5 microgram/dL. On the 3rd day of admission, the patient was given 200 mcg of IV levothyroxine. At that time, the patient was on a dopamine drip with a BP of 144/94 with a HR of 64. Twelve hours later, the patient's BP went up to 197/105 with a heart rate of 65 prompting discontinuation of the dopamine drip and administration of hydralazine and amlodipine. On the morning of the fourth day of admission, BP went up to 236/116 with a HR of 75 and the patient was started on a nicardipine drip. Blood work 3 days after starting levothyroxine revealed a TSH of >150, free T4 of 0.9, and a total T3 of 70. The patient was discharged on levothyroxine 150 mcg daily, amlodipine 10 mg, hydralazine 50 mg q6hrs, and Imdur 30 mg daily.

The patient’s myxedema score was 75 on admission. However, thyroid function tests were not checked with initial labs despite the presence of avert clinical features. As a result, the patient was started on a dopamine drip which could have been avoided. This underlines the importance of early recognition and management of myxedema coma. Having a low total T3 with a low normal FT4 could be explained by recent intake of high dose levothyroxine at home after a significant period of non-compliance. Fortunately, levothyroxine administration to this patient has reversed cardiovascular abnormalities including bradycardia and hypotension within hours when it was given promptly.

**Pediatric Endocrinology**

**PEDIATRIC ENDOCRINE CASE REPORTS I**

**Severe Hypocalcemia in an Infant with Abnormal Microarray and Dysmorphic Features**

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**Bone and Mineral Metabolism**

**BONE AND MINERAL CASE REPORTS II**

**Atypical Femur Fracture with Denosumab**

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Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

Glycogenic Hepatopathy. A Rare and Dramatic Manifestation of Poorly Controlled Type 1 Diabetes.
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SAT-671
Glycogenic Hepatopathy. A Rare and Dramatic Manifestation of Poorly Controlled Type 1 Diabetes
Background: Glycogenic hepatopathy (GH) is a well described, yet underdiagnosed disorder in type 1 diabetes. Erratic blood glucose values and high insulin levels promote the excessive deposition of glucose storage in the liver as glycogen, resulting in hepatomegaly, right upper quadrant pain and abnormal liver function. GH was first described with the introduction of insulin as a therapy to treat type 1 diabetes in the early 20th century. As our ability to effectively treat type 1 diabetes mellitus has improved, GH is seen much less commonly. Today, GH generally affects adolescent or young adult patients with poorly controlled type 1 diabetes mellitus and DKA. It is reversible with successful treatment of hyperglycemia.

Clinical Case: An 18 year old woman with a history of poorly controlled type 1 diabetes mellitus and frequent admissions for DKA was admitted for DKA and pyelonephritis. On admission, the patient complained of significant right upper quadrant pain and was found to have elevated transaminase values of: AST 1199 U/L (<37 U/L), ALT 371 U/L (56 U/L), an elevated alkaline phosphatase of 319 IU/L (<135 IU/L) and normal indices of biosynthetic function (INR/PT). After inpatient treatment of DKA and pyelonephritis, the right upper quadrant pain persisted and required pharmacologic analgesia. Radiographic evaluation demonstrated severe hepatomegaly (24 cm in maximum length) without focal lesions. Laboratory evaluation for viral hepatitis, autoimmune hepatitis, Celiac Disease, Wilson’s Disease and hemochromatosis were unremarkable. Given the patient’s persistent symptoms and severity of hepatomegaly, hepatic biopsy was performed. Biopsy findings were consistent with glycogenic hepatopathy demonstrating steatosis and glycogen deposition with nucleic glycogenation and mega mitochondria. Our patient had higher than usual insulin requirements for type 1 diabetes (~1 unit/kg/day). Abdominal pain, hepatomegaly and elevated LFTs resolved over a 2 month duration with improvement in her blood glucose control. Conclusions: GH is an established yet rare complication of poorly controlled type 1 diabetes. Glycogen deposition in the liver leads to painful hepatomegaly due to stretching of the liver capsule. GH has a female predominance (77%) and is characterized by elevated AST >>ALT with preserved liver biosynthetic function. It is postulated that GH is a result of elevated blood glucose levels and elevated insulin levels. The patient we describe has long standing poorly controlled type 1 diabetes mellitus, frequent admissions for DKA and high insulin requirements. To our knowledge, insulin requirements have not been investigated or previously reported as a potential risk factor for this condition.

MON-356
Background: Osteoporosis (OP) is a systemic disease that is associated with increased risk for fractures. Anti-resorptive medications (ARM) are an effective modality for treatment and prevention of fractures in OP. Long-term use of ARM like bisphosphonates (BP) is associated with increased risk of atypical femoral fractures (AFF). Denosumab (DM) is monoclonal antibody that targets receptor activator of nuclear factor KB ligand (RANKL). A few cases of AFF with DM have been reported in patients who have previously been on long term BP therapy. We present a case where the patient had only received two zoledronic acid (ZA) infusions, last one three years prior to being initiated on DM and experienced AFF.

Case: A 75-year-old postmenopausal woman presented to the emergency room with sudden onset pain in the right thigh. Patient was standing and reaching into her closet when she felt a snap in right thigh followed by inability to move the right leg or bear weight on it. X-ray of the right femur showed a displaced femoral shaft fracture with a short transverse fracture line and a spike, without comminution.

History was significant for OP for which she received raloxifene for a couple of years followed by ZA in 2013 and 2015 by her primary care. Fracture history was significant for a wrist fracture in 2015, and T12 and L1 fractures in 2017 from coughing while on Medrol dospak for an acute episode of bronchitis. In 2017, bone density showed L1-L4 T score of -2.0, the left total hip T score was -1.8 and left femoral neck T score was -2.5 consistent with OP. Work up for secondary causes of OP was unremarkable. Due to recent T12 and L1 compression fractures, she was recommended DM. She received DM in June 2018 as she was undergoing a dental implant followed by December 2018 and June 2019 and presented with the right femoral fracture in November 2019.

Past history was significant for idiopathic pulmonary fibrosis which had been relatively stable without need for long-term steroid therapy. History was negative for diabetes or kidney disease. Menarche was at age 14, menopause at 52 and she did not take any hormone replacement therapy.

She underwent surgical fixation with intramedullary rod. Due to concern about ARM associated sub-trochanteric fracture in the right femur, left femur was imaged. She had cortical beaking in the distal third of the left femur, and underwent prophylactic medullary nailing of the left femur as well.

Conclusion: AFF are an uncommon complication of ARM used for OP. Not many cases of AFF have been reported with the use of DM and most of the reported cases are associated with prolonged BP therapy. Our case is unusual in that AFF occurred in the absence of prolonged BP therapy and raises concern that a couple of ZA infusions in the past can also increase the risk of AFF. Clinicians need to have a high index of suspicion and may consider doing femur x-rays in patients who have previously been on BP prior to starting DM.