presentation, she had been drinking several drinks with high sugar content. Her medications included Ziprasidone (Geodon), Trileptal, and Cogentin for her bipolar disorder. She was started on Ziprasidone in 2007, changed to Brand name Geodon in 2014. Except for dry mouth, her exam was unremarkable. Labs were significant for blood glucose of 1114 mg/dL, bicarbonate of 18mmol/L, beta-hydroxybutyrate of 3.33 mmol/L, serum osmolality of 334 mOsm/kg. She was diagnosed with new-onset diabetes mellitus presenting as diabetic ketoacidosis. Her mother was diagnosed with DM2 in her 40s. She ha difficult to control blood sugars despite aggressive hydration and required regular insulin drip for 3 days for her anion gap to close. Managing her BGs was challenging.

Discussion: Clozapine and olanzapine are the common atypical antipsychotics that can cause DKA[1, 2]. To our knowledge, Ziprasidone is associated with hyperglycemia within days of starting the drug and HHS but not with DKA. For atypical antipsychotic associated DKA, risk factors include the duration of antipsychotic therapy, polypharmacy with multiple antipsychotic agents, non-Caucasians, obesity and pre-diabetes[2, 3]. Proposed mechanisms include peripheral insulin resistance, alteration of pancreatic beta-cell function by inhibiting 5-HT1A/2A/2C and alpha 2 adrenergic receptors[1-3]. However, there is no explanation of why few people develop complications while others do not. There is hypothesis regarding leptin gene polymorphisms of receptors that may play a role[4]. While starting patients on Ziprasidone, close monitoring of blood glucose is necessary before initiation and regular follow up thereafter[3].

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Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

A Normal FGF23 Does Not Preclude Tumor Induced Osteomalacia (TIO)

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Background: The phosphaturic, bone-derived hormone FGF23, mediates bone loss in TIO. Tumor resection typically results in skeletal healing and reversal of biochemical defects.

Case: A 45-year-old Caucasian man with no past medical history presented with non-traumatic fractures of bilateral metatarsals. Over the subsequent 2 years, he sustained fragility stress fractures in bilateral femurs, eventually rendering him non-ambulatory; though he continued to work as a property manager in an office setting. History included 30 pack-years of smoking as well as osteochondrosarcoma in his mother. Labs showed a low phosphorus at 2.1 mg/dL [2.7–4.5], along with calcium 8.8 mg/dL [8.4–10.4], iPTH 48 pg/mL [12–88], and ALP of 155 [IU]/L [34–104]. 25-OH vitamin D and 1,25-$(OH)_2$ vitamin D were 27 ng/mL [30–80] and 12 pg/mL [20–80], respectively. Free testosterone was 4.4 ng/mL [5–21] and LH 2.2 mIU/mL [3.0–10.0]. Other pituitary hormones and brain MRI were unremarkable.

Vitamin D3, calcitriol, phosphate and testosterone were prescribed. Testosterone was discontinued 6 months later, after diagnosis with DVT/PE. Incidental rib fractures on CXR prompted a 3-phase ^{99}Tc-MDP bone scan, revealing multiple sites of uptake: ribs, scapulae, sternum, thoracolumbar spine, sacrum, bilateral ankles and feet. DXA revealed T-scores of -2.8 in the spine and -1.9 in the femoral neck.

Labs pointed to ongoing phosphate wasting, despite compliance with calcitriol 0.25 mcg, cholecalciferol 5000 IU and phosphorus 2250 mg in divided doses. Calculated TRP was 64% [>80%] and TmP/GFR 1.74 mg/dL [2.5–4.5], consistent with low phosphate reabsorption. Urine 24-hour calcium was 244.8 mg. Testing for causal mutations of hypophosphatemic rickets and osteogenesis imperfecta was negative. FGF23 was within the reference range at 138 RU/mL [LabCorp ELISA 44–215], interpreted as inappropriately normal given ongoing phosphate loss. This prompted a search for a suspected TIO locus.

Two years after presentation, ^{18}F-FDG-PET exhibited a hypermetabolic focus at the left suprapatellar recess. Biopsy was consistent with a mesenchymal tumor and chromogenic in situ hybridization (Mayo Clinic) was positive for FGF23 mRNA. After surgical resection of the 1.7 cm tumor, serum FGF23 declined to <50 RU/mL and was 90 RU/mL 3 weeks later [Mayo <180 RU/mL]. Calcitriol and phosphorus supplementation were discontinued. One year post-operatively phosphorus was 3.6 mg/dL [2.5–5.0], with a normal calcium, iPTH and 25-OH vitamin D. Lumbar spine T score improved to -1.0 (+46.43%).

Conclusion: A high clinical index of suspicion is required for TIO in the setting of phosphaturic osteomalacia, particularly with a normal serum FGF23. Recently, similar microRNA profiles were noted in osteosarcomas and TIO[1]. This, along with our patient's family history raises the question of a possible predisposition to skeletal neoplasms.

1. Green et al. *Bone Reports*, 2017.

Thyroid

THYROID DISORDERS CASE REPORTS III

Thyroid Abscess Due to Intravenous Drug Use: A Rare Cause of Thyroid Storm

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MON-463

Objective: Our objective is to discuss the clinical presentation, diagnosis and management of thyroid storm due to thyroid abscess.

Methods: We report a case of thyroid abscess due to intravenous drug use (IVDU) resulting in thyroid storm.

Results: A 28 year old female with history of IVDU and no known thyroid disease presented with neck pain and fever