for 160 HU and 90% specificity for 110 HU previously reported in the general population. Given higher fracture risk in DM, moderate osteopenia (n=106) was also examined as an outcome: 130 HU was 61% sensitive and 71% specific. This threshold had similar or improved sensitivity and specificity among subgroups of insulin users, men, and women under age 65.

Conclusion: Our results validate the use of opportunistic osteoporosis screening in patients with DM, which could help clinicians decide on the need for screening DXA. Patients with diabetes and L1 attenuation below 130 HU on CT scan should be considered for DXA screening to formally assess the risk of fracture.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Early Diagnosis and Management of Bilateral Transient Osteoporosis of the Hip in Pregnancy

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MON-351

Introduction: Transient osteoporosis of the hip (TOH) in pregnancy is a rare and under-reported condition. It is clinically characterized by a sudden onset of hip pain in young females without any history of systemic disorders or traumatic injuries (1). Bilateral involvement of the hips, such as in this case report, is less common than unilateral involvement.

Clinical Case: A 33 year old G2P1 female presented to the hospital at 30 weeks gestation for described sharp, bilateral inguinal pain, greater on the left than right, worse with movement, and with progressive difficulty ambulating, of three weeks duration. She had no significant PMH, notably denying thyroid or calcium disorder, nephrolithiasis, osteoporosis, or steroid treatment. She denied tobacco, alcohol, or illicit substance usage. She only took prenatal vitamins. On physical examination, she had reduced active and passive range of motion of both hips, but normal muscle strength and no signs of infection or neurological deficits. Labs including CMP, LFTs, and TFTs were within normal range. 24 hour urine free cortisol was 54 mcg/24h (normal 3.5-45mcg/24h); repeat post-partum was 21 mcg/24h. 25OH-D was 18.3 ng/mL. MRI without contrast demonstrated "extensive abnormal marrow edema within the left femoral head and neck and small effusion, suspicious for transient osteoporosis of the hip. A subtle small focus of edema on the right was also noted. No discrete fracture line or subchondral collapse was noted."

The patient was managed conservatively with analgesics, thromboprophylaxis, and education regarding reduction of weight bearing activities, rest, and mobility aids with crutches. She was started on vitamin D. Her bilateral hip pain resolved by the 38th week. She had an uncomplicated cesarean delivery at 39 weeks to a healthy male neonate. At the one-month postpartum visit, she was ambulating independently without difficulty. She denied further pain, and passive and active ROM were intact without tenderness.

Conclusion: TOH in pregnancy is usually a self-limiting disorder with no obvious etiology (2). It can present unexpectedly in the third trimester or early postpartum period in a healthy female with an otherwise uneventful pregnancy. In rare instances where fractures of the affected hip occur, surgical intervention may be necessary. MRI has become the diagnostic tool of choice for early diagnosis of TOH. Early diagnosis and optimal management are essential to prevent major complications such as traumatic fractures and deep vein thrombosis, as well as to prevent stress for the mother during the course of pregnancy.

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Bone and Mineral Metabolism

CLINICAL ASPECTS OF OSTEOPOROSIS AND VITAMIN D ACTION

Potential Relationship Between Hypothyroidism and Bone Loss at Dental Implants

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MON-397

Introduction: Hypothyroidism (HT) is an endocrine condition with autoimmune and inflammatory etiologies. Studies have shown that both periodontal disease and peri-implant bone loss are bidirectionally influenced by systemic inflammatory conditions, such as diabetes, adverse pregnancy outcomes, cardiovascular disease, and osteoporosis.[1] There also is evidence that HT is associated with decreased bone metabolism, depressed bone turnover, and a prolonged bone remodeling cycle.[2] Consequently, the objective of this study was to determine if the severity of bone loss around dental implants is related to the presence of HT.

Methods: Following IRB approval, medical, dental, and radiographic records of patients who received dental implant placement at a university-based postgraduate program in periodontics from 2000–2017 were reviewed (1480 implants; 635 patients). Rate of bone loss in mm/year was calculated from surgical implant placement and subsequent re-evaluation radiographs, with correction for radiographic distortion. Presence of HT was confirmed by review of patient medical records, clinical diagnosis of HT, and history of thyroid hormone supplementation. Populations were adjusted for smoking, diabetes, use of systemic steroids, presence of autoimmune disease (other than HT), and systemic inflammatory conditions. Calculations were performed using IBM SPSS Statistics v25.

Results: Patients with HT had a decreased rate of crestal alveolar bone loss around dental implants. Specifically, patients with HT experienced peri-implant bone loss at a rate of 0.42 mm/year, while bone loss from patients without HT was 1.34 mm/year (68.7% decrease; mean difference = 0.92 mm/year, 95% confidence interval = 0.39–1.50 mm/year, $P<0.002$). There were no significant differences in patient oral hygiene, or in implant service time, among any of the groups studied ($P>0.05$).

Conclusions: The results suggest that the rate of marginal alveolar bone loss at dental implants is significantly decreased in patients with HT, and occurs independently of any of the systemic conditions noted above. The findings imply that potential changes in bone metabolism and remodeling associated with HT might result in less peri-implant alveolar bone loss following implant placement surgery. As a result, there does not appear to be an increased risk of peri-implant crestal bone loss in patients with HT.

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Adrenal

ADRENAL CASE REPORTS II

Adrenalitis Induced by Nivolumab

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SUN-183

Introduction: Tumor cells often express a programmed death-ligand 1 (PD-L1), which binds to the programmed death receptor-1 (PD-1) on activated T-cells to induce immune tolerance. Among the class of immune checkpoint inhibitors (ICI), Nivolumab is an anti-PD-1 antibody which blocks these tumor cell interactions. Although some endocrinopathies have been reported for other PD-1 inhibitors, the adverse event of adrenalitis with nivolumab has not been reported before. **Clinical Case:** A 65-year-old female presented to the hospital with complaints of nausea, vomiting, fatigue, and headache for five days. She was recently diagnosed with metastatic lung adenocarcinoma, complicated by cerebellar metastases, and the left cerebellar mass was resected. She was also started on Nivolumab. Her blood pressure was 98/65 mmHg on the presentation. Serum sodium was 122mEq/L (normal 135–145) and potassium was 5mEq/L (3.5–5). TSH, LH, and prolactin were all normal. Aldosterone was low: 23 pmol/L (27.7–582.5) and renin was high: 11 ng/ml/h (0.167- 1.38). Morning cortisol levels were low: 2.2 ug/dl (5- 25) and concomitant ACTH was high: 78 pg/ml (7.2- 63.3). Upon standard high dose cosyntropin stimulation test, basal cortisol was 2.0 ug/dl (5- 25). Cortisol level 30 minutes post cosyntropin was 7.1 ug/dl, while Cortisol 60 minutes post cosyntropin was 12.2 ug/dl (normal >18 -20 ug/dl). Considering the low cortisol levels with high ACTH, and an inadequate rise in cortisol after the ACTH stimulation test, adrenal insufficiency was suspected as a result of adrenalitis due to Nivolumab. Hyponatremia along with low aldosterone and high renin levels also reinforced this clinical diagnosis. A computerized tomographic scan of the chest abdomen and pelvis only showed calcified uterine fibroids. She was initially resuscitated with intravenous fluids. Hydrocortisone 100 mg every 8 hours was started and then gradually tapered down to 60mg every 12 hours. Fludrocortisone was also initiated at 0.2mg daily. Symptoms began to improve, and sodium levels normalized to136 mEq/dl. She was discharged on 30mg of hydrocortisone and 0.1 mg of fludrocortisone daily and is stable since then. **Conclusion:** This is a rare case of Nivolumab-induced adrenalitis. It highlights the importance of checking for adrenal insufficiency in a patient who presents with symptoms of hypotension and hyponatremia while being on ICI drugs, as unidentified adrenal insufficiency and adrenal crisis can be fatal.

Adipose Tissue, Appetite, and Obesity

OBESITY TREATMENT: GUT HORMONES, DRUG THERAPY, BARIATRIC SURGERY AND DIET

Post-Bariatric Hypoglycemia: A Clinical Vignette on an Increasingly Recognized Disease

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MON-599

Introduction: Previously referred to as late dumping syndrome, post-bariatric hypoglycemia (PBH) is thought to represent at least 1% of all hospitalizations for hypoglycemia and 10% of all clinically recognized hypoglycemia cases. However, through the advent of CGM and more strict criteria over the last decade these numbers are likely an underestimate. As obesity continues to remain prevalent and with rising bariatric centers to help deal with this epidemic, endocrinologists will play an increasing role in managing PBH patients.

Clinical Case: A 39-year female with a PMH of hypothyroidism and bariatric surgery (BS) in 2009 presented to our ER for a seizure. She has been having seizures nearly every 2 weeks for one year. Neurology started her on Keppra; however, no etiology was identified. EMS had documented a blood glucose of 40 mg/dL; the patient was given an amp of D50 with resolution of neuroglycopenic symptoms. TSH and cortisol levels were within normal range. A sulfonylurea panel in the ED was negative. The patient states the symptoms can occur while fasting but also mainly post-prandial. A 72-hr fast was conducted with the patient nadir POC glucose of 77. Subsequently, the patient had a mixed meal tolerance performed and after 2 hours had a seizure and was found to have a BG of 50 mg/dL with an insulin level of 49 uIU/mL and a c-peptide of 18.8 ng/mL. The patient was diagnosed with PBH, and was discharged with a CGM, started on acarbose and was seen by nutrition to discuss dietary modifications. She is now seen in our clinic with control of her symptoms with the addition of diazoxide.

Conclusion: Altered anatomy after bariatric surgery, particularly after gastric bypass and sleeve gastrectomy is thought to play a major role in developing PBH. By bypassing normal anatomy, gastric emptying is increased 2–3 x, which leads to a higher and more rapid appearance of glucose in the distal foregut. This subsequently leads to an amplified incretin response leading to a hyperinsulinemic response in patients who have had bariatric surgery; however, for unclear reasons some patients develop an even more amplified hyperinsulinemic response that leads to subsequent hypoglycemia. History of neuroglycopenic symptoms 1–3 hours after eating in a patient who had a gastric bypass > 6–12 months and with relief of symptoms with carbohydrates should raise an endocrinologist's suspicion of PBH. Fasting hypoglycemia is an atypical feature that should raise one's suspicion of a