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.

References: [1]Kim J., Amar S., *Odontol*. **94**(1):10–21, 2006. [2]Tuchendler D., and Bolanowski M.,Thyroid Res. 7:12, 2014.

Adrenal

ADRENAL CASE REPORTS II

Adrenalitis Induced by Nivolumab

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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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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