regular basal bolus Insulin regime with the help from the diabetes team.

Discussion: Here we have presented a case with new onset Insulin dependent Diabetes Mellitus induced by immune checkpoint inhibitor. This kind of Diabetes progress rapidly to severe insulin deficiency compared to spontaneous Type 1 Diabetes, frequently patient present with DKA and do not go into remission. As this condition can develop rapidly, it is suggested that glucose level is to be monitored regularly and also to check HbA1C prior to initiating the immunotherapy. Their management requires complex Insulin regime to get good glycaemic control and add significant comorbidity along with the underlying cancer. The exact pathophysiologic mechanism and predictive biomarkers have not yet been established. The end result is permanent Insulin dependence. In future better characterization and further study is required to improve diagnosis and management, also to follow the natural history of this condition.

Reference:

1) Kotwal A, Haddox C, Block M, *et al*Immune checkpoint inhibitors: an emerging cause of insulin-dependent diabetes*BMJ Open Diabetes Research and Care* 2019;**7:**e000591. doi: 10.1136/bmjdrc-2018-000591

2) Immune checkpoint inhibitors and type 1 diabetes mellitus: a case report and systematic review.*de Filette JMK*[1], *Pen JJ*[2], *Decoster L*[3], *Vissers T*[4], *Bravenboer B*[1], *Van der Auwera BJ*[5], *Gorus FK*[5], *Roep BO*[6,7], *Aspeslagh S*[3], *Neyns B*[3], *Velkeniers B*[1], *Kharagjitsingh AV*[1,]

Adrenal

ADRENAL CASE REPORTS I

Bilateral Large Calcified Adrenal Leiomyoma Mimicking Adrenal Malignancy: A Rare Case Report With Literature Review.

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SAT-LB42

Background: Adrenal leiomyoma is a very rare benign soft tissue tumor, it is even more unusual if presenting bilaterally; 21 cases have been reported in the literature and only six had bilateral involvement; 5 in the pediatric population and only one in an adult patient. Radiological appearance may frequently be confused with malignancy especially if large, calcified and with central necrosis. We report a rare case of bilateral, large, calcified, non-functioning adrenal leiomyoma in a 20-year-old female, who was suspected for a malignancy preoperatively.

Clinical Case: A 20 year-old-female presented with chronic abdominal discomfort, fatigue, and inability to gain weight. On examination, she was normotensive, underweight with BMI of 15.6 kg/m^2, and there were no stigmata of Cushing's syndrome, Addison's disease or pheochromocytoma. A contrast CT scan of the abdomen revealed the presence of bilateral well-defined suprarenal lesions measuring 8.5 x 8.5 x 7.2 cm and 4.7 x 4.2 x 3.5 cm on the right and left side, respectively. The lesions showed large central areas of necrosis with multiple punctate calcifications and heterogenous peripheral enhancement. The radiological differential diagnosis included adrenal cortical carcinoma, adrenal metastasis, infectious etiology, and bilateral pheochromocytoma. Her hormonal assays showed normal free cortisol and catecholamine metabolites in the urine and normal serum androgens. Thus, the tumors were concluded to be non-functioning. Adrenal insufficiency was ruled out after a short Synacthen test. The patient underwent a successful right adrenalectomy. Resected specimen measured 10 x 9.5 x 7.5 cm. Histology revealed a well-circumscribed and pseudo-encapsulated smooth muscle tumor comprised of bland, spindle-shaped cells. The panel of immunohistochemical stains supported the diagnosis of leiomyoma. Postoperatively, the symptoms improved, she gained 4 kg weight over the following 4 months, and short Synacthen test confirmed an intact adrenal function. To avoid lifelong adrenal insufficiency and after discussion with the patient, we agreed to leave the left adrenal mass and follow it by serial imaging. There was only a minimal increase in the size over the following 4 years (5.5 x 4.5 x 3.8 cm).

Conclusion: Adrenal leiomyoma is an extremely rare adrenal tumor and can be confused with adrenal malignancy. Therefore, it should be considered in the differential diagnosis of adrenal incidentalomas. In the case of bilateral etiology, permanent adrenal insufficiency and longterm replacement therapy can be avoided in certain population by removing the larger tumor and continuous follow-up for the other side.

Adipose Tissue, Appetite, and Obesity

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

The Effect of the Ketogenic Diet on Aldosterone Over 6 Weeks

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MON-LB106

Introduction: A ketogenic diet improves type 2 diabetes, metabolic syndrome, and cardiovascular disease. Weight loss studies using caloric reduction have demonstrated a decrease in aldosterone, but there is limited data on the effect of a ketogenic diet on aldosterone. Thus, we evaluated the impact of a ketogenic diet on aldosterone in overweight or obese individuals over 6 weeks. Methods: This 3-arm prospective controlled feeding study evaluated aldosterone and renin concentrations over 6 weeks on a hypocaloric (25% energy restricted) ketogenic diet + placebo (KD+PL), ketogenic diet + ketone salt supplement (KD+KS), and a low-fat diet (LFD). Sodium intake consisted of 6100 mg, 2300 mg, and 2000 mg for the KD+KS, KD+PL, and LFD groups, respectively. Both ketogenic diets provided 40 grams(g) day of carbohydrates, 1.5 g/kg reference weight of protein and remaining calories provided as fat. The LFD

provided 25% total fat, 1.5 g/kg reference weight of protein, and 100g of carbohydrates. Serum aldosterone was drawn fasting in upright position at 0, 2, 4, and 6 weeks. Scatter plots were used to explore the residual and predicted associations between aldosterone with other measures after accounting for time and group effect.Results: Twenty-four participants in the ketogenic diet groups were matched for age and body mass index, then randomized to either the KD+PL or KD+KS group. A separate group of 12 matched participants were specifically recruited for the LFD group. The median age was 33 years. Weight decreased 6, 8, and 7 kg on average in the KD+KS, KD+PL, and LFD groups, respectively, over 6 weeks ($p<0.05$ for all). Systolic blood pressure (SBP) improved from 117 and 115 mmHg in the KD+KS and KD+PL groups to 110 mmHg over 6 weeks while the baseline mean SBP 118 in the LF group did not change. Baseline mean aldosterone of 13.6 and 13.6 ng/dL in the KD+KS and KD+PL groups increased to 33.3 and 27.3 ng/dL over 6 weeks ($p<0.001$). Baseline mean aldosterone of 8 ng/dL in the LF group non-significantly changed to 11.5 ng/dL over 6 weeks ($p>0.05$). Using predicted value associations, increases in ketones were positively associated with higher aldosterone ($R2=0.86$; $p<0.001$). Conclusion: Participants on a ketogenic diet had significantly elevated aldosterone levels throughout the study while participants on low fat diet had little change. Unexpectedly, aldosterone was significantly higher in the high sodium vs. low sodium ketogenic diet. There was a significant association between ketones and aldosterone suggesting that ketones may play a stimulatory role on aldosterone synthesis or secretion.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

The Association of Paget's Disease With Inclusion Body Myositis and Fronto Temporal Dementia (IBMFTD)

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MON-LB70

The association of Paget's Disease with Inclusion body myositis and Fronto Temporal Dementia (IBMFTD)

Background:Inclusion body myopathy associated with Paget disease of bone (PDB) and/or frontotemporal dementia (IBMPFD) is a rare, autosomal dominant condition, characterized by adult-onset muscle weakness, early-onset PDB, and premature frontotemporal dementia. Paget's disease is a chronic disorder of bone resulting in increased bone resorption, followed by a disorganized and excessive formation of bone.

Clinical Case:A 43 year old gentleman was referred to neurology services with foot drop, limb weakness and cognitive impairment. Following a prolonged period of diagnostic evaluation a mutation in valosin-containing protein (VCP) gene was uncovered

IBMPFD has a variable phenotype which may include PDB. This gentleman denied bony pain and had an alkaline phosphatase within reference range. Plain film radiographs at multiple sights demonstrated no signs of PDB. An MRI whole body was performed which reported coarse trabecular markings in L2 vertebral body and multilevel degenerative changes with bridging osteophytes in the lumbar spine consistent with PDB.

At initial review in endocrine clinic, he denied fractures or bone pain. He had no signs of increased cardiac output or cranial nerve deficits. A radionuclide bone scan identified intense radiopharmaceutical accumulation in L2 vertebral body consistent with MRI findings and also curvilinear increased activity in the left occipital bone and low-grade activity in the left hip, thus confirming polyostotic Paget's disease.

He received a 5mg IV infusion of zolendronate, side effects of which included myalgia, pyrexia and lethargy.

Six months following the zoledronate infusion a repeat bone scan demonstrated that the extent of uptake at affected sites had decreased significantly.

He subsequently fractured his left femur following a mechanical fall. PDB can affect up to 50% of patients with IBMFTD. PDB can lead to complications such as bone pain, localized pain and deformity of the long bones, pathologic fractures and deafness. This case highlights the association of Paget's disease with IBMFTD and as it can be asymptomatic, as in our case, radiological imaging is required for diagnosis. It also reminds us that Paget's disease can be due to genetic causes. Understanding the role of VCP in the cell cycle may help in further understanding bone physiology.

Neuroendocrinology and Pituitary

CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

Acute Sterile Meningitis as a Primary Manifestation of Pituitary Apoplexy

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SAT-LB54

Background: Pituitary apoplexy (PA) is a rare endocrinopathy that requires prompt diagnosis and treatment. Presentation with acute neutrophilic meningitis is uncommon.

Clinical Case: A 67-year-old man presented to our hospital with a 2-week history of worsening bilateral frontal headache, nausea, and dry heaving. On admission, the patient was somnolent with a score of 13 on the GCS assessment (E2, V5, M6). The neurological exam was overall normal with normal ocular motion and intact cranial nerves, except for a left eye peripheral vision defect. Plain head CT revealed a well-circumscribed ovoid pituitary mass with suprasellar enlargement, consistent with a pituitary macroadenoma. Sellar MRI showed a pituitary mass, roughly 20 x 19 x 24 mm, bulging into the suprasellar cistern with optic chiasm elevation. Analysis of pituitary function revealed low ACTH concentration of 2.8 pg/mL (n = 7.2 - 63.3 pg/mL), a low random cortisol level of 1.7 ug/dL (n = 2.9 - 19.4 ug/dL), a low TSH of < 0.1 uIU/mL (n = 0.35 - 4.9 uIU/mL), a low free T4 level of 0.72 ng/dL (n = 0.77 - 1.48 ng/dL), a low LH of 0.8 IU/L (n = 1.7 - 8.6 IU/L) with a very low total testosterone