clinical vignette, we present a patient with Allgrove’s syndrome who developed clinical manifestations of the third ‘A’ of Addison’s disease later in life. A 46-year-old female patient presented to our tertiary referral center for follow-up of Allgrove’s disease after having been diagnosed with genetic testing as an adolescent. Prior to presentation, she underwent esophagectomy in 1995 and additionally had confirmed alacrima with ophthalmology. She was undergoing annual surveillance testing with 8 am cortisol and ACTH stimulation test to monitor for the development of adrenal insufficiency. Prior to consultation, her baseline cortisol was 8.7 ug/dL. At presentation and the age of 46, her ACTH stimulation test (0.25 mg cosyntropin, 3 timepoints) was positive for adrenal insufficiency with a baseline cortisol of < 0.5 ug/dL (8 am), with 30-minute value of 4.4 ug/dL and 60-minute value of 6.3 ug/dL (peak). She was started on replacement dosing of hydrocortisone 20 mg at 8 am and 10 mg at 2 pm, in addition to calcium and vitamin D supplementation. Of note, her adrenal antibody (21-hydroxylase antibodies) were negative on two separate occasions. Allgrove’s syndrome is a rare condition described by the development of three, or at times four, characteristics with support of genetic testing. This case demonstrates that patients with Allgrove’s syndrome can present with two clinical manifestations of the condition (alicrima and achalasia) and develop the third (adrenal insufficiency) later in life. Therefore, regular screening for the missing clinical manifestation of this disease should be considered. References: 1. National Institute of Health: Genetic and Rare Disease Information Center. Triple A Syndrome. Genetics Home Reference. February 2010; http://ghr.nlm.nih.gov/condition/triple-a-syndrome. Accessed 2/1/2020. 2. Kimber J, McLean BN, Prevett M, Hammans SR. Allgrove or 4 “A” syndrome: an autosomal recessive syndrome causing multisystem neurological disease. J Neurol Neurosurg Psychiatry. 2003;74:654-657.

Diabetes Mellitus and Glucose Metabolism

**DIABETES COMPLICATIONS II**

**New Onset Insulin Dependent Diabetes Mellitus Secondary to Treatment With Immune Checkpoint Inhibitor:**

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**MON-LB121**

New onset Insulin dependent Diabetes Mellitus secondary to treatment with immune checkpoint inhibitor

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**Background:** Checkpoint inhibitors, immunomodulatory antibodies that are used to enhance the immune system, have substantially improved the prognosis for patients with advanced malignancy like melanoma and lung cancer. Despite important clinical benefits, checkpoint inhibition is associated with a unique spectrum of side effects termed immune-related adverse events (irAEs). IrAEs include dermatologic, gastrointestinal, hepatic, endocrine, and other less common inflammatory events. Among them is endocrine toxicity, most commonly targeting the thyroid, pituitary, or adrenal glands. New-onset diabetes mellitus has been reported in only around 1% of patients in a recent study.

Although rare, fulminant and even fatal toxicities may occur with immune checkpoint inhibitors, and therefore, prompt recognition and management is important. Here we are going to present a patient with new onset Insulin dependent Diabetes mellitus secondary to immunotherapy. It usually presents with diabetic ketoacidosis (DKA) and follows a rapid course. Awareness and prompt management are therefore key.

**History and investigations:** 62 year old lady diagnosed with Right Uveal melanoma more than 2 years ago and was treated with Enucleation followed by Rt prosthetic eye. Subsequently patient developed metastatic melanoma with subcutaneous lesion in right paravertebral region, right humoral head and right gluteal muscle. It was unclear whether patient had metastatic uveal or cutaneous melanoma. Other PMH includes were Hypertension and Anxiety. Patient was started on Ipilimumab (CTLA-4 inhibitor) and Nivolumab (PD1 inhibitor) 6 months ago, and Ipilimumab was stopped 8 weeks ago due to side effects but continued with Nivolumab. Other current medications were Amlodipine 10 mg once daily and Amitriptyline. Patient was complaining of extreme fatigue last one week and was diagnosed with Hypothyroidism with TSH >100 mIU/L (Normal 0.27-4.2) and FT4 5.4 pmol/L (Normal 12.0-22.0), subsequently patient was started on Levothyroxine 50 mcg once daily. Patient presented to emergency department with polyuria and polydipsia last 5 days and also blurred vision for last 3 weeks. Patient did not notice any recent weight loss and had widespread pain, worse on skin lesions and hip joints but did not had any other specific complaints. Patient was current smoker with more than 40 pack year history and was taking 25 units of Alcohol per week for many years. Patient did not had any significant family history including any history diabetes in the family. On examination, patient was clinically dry with capillary refill time was 5 seconds. Investigations showed-Venous blood gas-Blood Glucose - Hi (mmol/L out of range), later 22.7 mmol/L. LPh- 7.291, PCO2 6.14 kPa, HCO3 19.3 mmol/L, Lactate 2.2 mmol/L.Blood ketones- Hi (mmol/L out of range), later >7 mmol/L.

Other investigations showed-Na 131 mmol/L (Normal 135-145), K 5.4 mmol/L (Normal 3.5-5.1), Urea 7.1 mmol/L (Normal 1.7-8.3),Creatinine 139 umol/L (Normal 49-92) Bilirubin 12 umol/L (Normal 0-20), ALT 56 IU/L (Normal 10-35), ALP 157 IU/L (Normal 35-104),Amylase 59 IU/L (Normal 28-100), Albumin 48 g/L (Normal 34-50), Adjusted Calcium 2.56 mmol/L (Normal 2.2-2.69) am Cortisol 826 nmol/L (Normal 133-537), ACTH 28 ng/l (Normal 7.2-63.3)FSH 7.87 IU/L (Normal 25.8-134.8), LH 37.6 IU/L (Normal 7.7-58.5),IGF1 9.6 ng/mL (Normal 3.5-32.0), Prolactin 476 mIU/L (Normal 102-496)HbA1C 10.6% / 93 mmol/mmol (Normal 20-42) Serum Anti-GAD titre- 5 IU/L (Normal 0-10)

Patient was started on treatment for Diabetic Ketoacidosis (DKA) with intravenous fluid and also fixed rate Insulin infusion according to protocol. Patient responded well to treatment and biochemical profile improved with initial treatment, subsequently patient was started on
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 pathophysiological 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.

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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/m2, 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