deficiency has a worldwide distribution. AV at NADPH-ligand region cause lower virilization. Genitalia virilization influenced sex assignment but not gender change which was influenced by cultural aspects across the countries.

Thyroid

THYROID DISORDERS CASE REPORTS II

Anorexia and Severe Hypothyroidism Driving Persistent Ascites and Liver Injury

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SAT-515

Background Unintentional weight loss, ascites, and altered mental status typically raise concerns for hepatologic-cardiac-oncologic disease. Herein we present a case that after exhaustive workup the etiology of the aforementioned findings was attributed to a combination of anorexia and severe hypothyroidism, states where slowed metabolism and altered nutritional status can interact synergistically. **Clinical Case** A 71 y/o lady with a history of hypothyroidism after RAI for Graves' disease, anxiety, depression, presented with 20-30 lbs unintentional weight loss over 5 months. She had poor appetite, nausea, constipation, increased abdominal girth, anasarca, fatigue, weakness, and altered mental status. On exam she had bradycardia, hypotension and hypothermia (BP 97/51, HR 54, RR 18, Temp 94, BMI 15.8). Labs revealed a TSH of 36, FT4 0.98, Tbili 0.3, alk phos 491, AST 435, ALT 651, gGT 151, albumin 2.3, prealbumin 14.7, INR 1.03, and pancytopenia. Treatment with IV levothyroxine resulted in mental status improvement. Over the next few months extensive testing revealed mild element deficiencies (zinc 44- nl range 60 - 130 mcg/dL, vit A 32- nl range 38 - 98 mcg/dL), no evident infectious/hematologic or inflammatory disease. Despite 3 paracenteses, 2 liver biopsies, multiple imaging studies (abdominal U/S, ECHO, XRs, CT chest/abdomen/pelvis, MRI abdomen/pelvis, enterography, MRI brain, PET scan), EGD/colonoscopy, bone marrow bx, and explorative laparoscopic abdominal surgery, no clear explanation was found. During the workup her liver enzymes remained elevated, TSH and fT4 normalized, but T3 remained low at 50 (76 - 181 ng/dL). Psychiatric evaluation revealed mild cognitive impairment, presumed to be secondary to depression and underlying disease. By exclusion, she was diagnosed with anorexia with a possible component of persistent hypothyroidism. She was fed through NG tube and followed closely by nutrition with a personalized high protein and calorie meal plan. Eight months later she had gained 5 lbs, her liver enzymes/electrolytes and CBC normalized, and most of her symptoms resolved. **Conclusion** Hypothyroidism can cause LFT abnormalities, and though rare, there are >50 cases of hypothyroidism induced ascites reported. Usually LFTs and ascites normalize promptly with hormone supplementation. However, our patient's case was complicated by severe anorexia, with nutritional status essentially equivalent to kwashiorkor sufferers. Though extremely rare in the developed world and in adults, kwashiorkor like physiology has been described in patients with anorexia, with impressive liver abnormalities, presumed to be due to autophagy. Though certainly this is a rare diagnosis, it does point to the fact that in our test driven culture, it is worth pausing, re-evaluating history and physical and thinking outside the box prior to subjecting our patients to countless tests and procedures.

Diabetes Mellitus and Glucose Metabolism

DIABETES DIAGNOSIS, TREATMENT AND COMPLICATIONS

Low Risk of Major Adverse Cardiovascular Events After Pancreas Transplantation Alone

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SUN-624

Low Risk of Major Adverse Cardiovascular Events after Pancreas Transplantation Alone

INTRODUCTION: Type 1 Diabetes (T1D) patients have an increased risk for major adverse cardiovascular events (MACE). Pancreas Transplantation Alone (PTA) in patients with T1D achieves near normal glucose control for a prolonged period but limited data are available to date regarding MACE during a 10 year follow up period after the procedure.

OBJECTIVE: We studied incidence of MACE after PTA in T1D patients over a 10 year follow-up period.

METHODS: Retrospectively, we studied 113 T1D recipients of PTA at Mayo Clinic, Rochester with the procedure performed between January 1998 and August 2018 and follow up of at least 1 year. Data were collected before transplantation and up to 10 year follow up after the first PTA. MACE data were gathered until primary non function, re-transplantation, or complete loss of c-peptide (<0.01ng/ml). We report vascular risk factors including hypertension, hyperlipidemia, smoking and BMI along with MACE (defined as cardiac events as unstable angina, Myocardial Infarction (MI), need for re-vascularization, cardiac death, cerebral events as Transient ischemic attack (TIA), stroke, need for re-vascularization and peripheral arterial disease as need for re-vascularization, gangrene and amputation).

RESULTS: Eighteen subjects had pre-transplant MACE. A total of 14 subjects had graft failure within 24 to 36 hours due to thrombosis, with 3 in pre-transplant MACE cohort and 11 in no MACE cohort. Thus, we followed 99 subjects for the development of post-transplant MACE for a period of 6.3 ± 3.6 years. T1D subjects with MACE (n=15) had baseline characteristics: Age 48± 7.8 years, gender F/M 9/6,, duration of diabetes 33 ± 12 years, BMI 26± 3.1(Kg/m^2), HbA1c 9.3 ± 1.5% and C-peptide 0.09 ng/ml. 84 T1D patients without MACE were age 42 ± 10.6 years, gender F/M 55/29, duration of diabetes 26.5 ± 10.7 years, BMI 26 ± 5.2(Kg/m^2), HbA1c 6.7 ± 2.5 and C-peptide 0.09 ng/ml. There are a total of 584 person-years of follow up to first MACE event and 632 person-years of graft failure, death or last follow-up. Nine patients developed 11 MACE events post-PTA. Therefore, the event rate is 1.5 MACE events per

100 person-years for first MACE event and the total event rate is 1.7 MACE events per 100 person-years of follow-up. Age, smoking (yes), gender, duration of diabetes, HTN and Hyperlipidemia presence did not show any significant impact on post-transplant MACE outcome based on univariate Cox regression but the pre-transplant BMI (HR = 1.14; CI = (1.04, 1.26); p = 0.008) and pre-transplant HbA1c (HR = 1.26; CI = (1.06, 1.51); p = 0.01) showed statistically significant impact.

CONCLUSIONS: At our center, MACE is low in PTA recipients. There is no impact of presence of pre-transplant MACE on development of post-transplant MACE but pre-transplant BMI and HbA1c account for risk of MACE.

Adrenal

ADRENAL - CORTISOL EXCESS AND DEFICIENCIES

Body Weight Reduction Not Always Reduced Adrenal Gland Volume in Obese Patients

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MON-161

Body Weight Reduction Not Always Reduced Adrenal Gland Volume in Obese Patients **Abstract**: Recent studies have shown that obesity is a major risk factor for idiopathic hyperaldosteronism (IHA). IHA patients have greater AGV than normal controls. However, it is unclear whether such changes are caused by obesity and whether losing weight could reverse the morphological and functional abnormalities of the adrenal gland. This study was to investigate the association of obesity with adrenal gland volume (AGV) and the effects of weight loss on AGV. This study recruited obese patients (N=25) who underwent sleeve gastrectomy and age- and sex-matched normal-weight (N=25) and overweight healthy volunteers (HV) (N=21). Thin-slice computed tomography was used to evaluate adrenal morphological changes. AGV was measured semiautomatically based on the digital imaging and communications in medicine (DICOM) image. The effects of weight loss on AGV were evaluated in patients for one year or more after sleeve gastrectomy. The results showed that left, right and total AGV were larger in obese patients than those in overweight and normal- weight HVs (6.77±0.36, 5.76±0.31, and 12.53±0.64 cm^3 vs. 3.88±0.14, 3.09± 0.13 and 6.97± 0.24 cm^3 vs. 3.38±0.23, 2.67±0.15 and 6.04±0.36 cm^3). No statistically significant difference was identified between overweight and normal-weight HVs. Sleeve gastrectomy significantly reduced body weight (-27.1±2.5 kg), left AGV (-0.80±0.26 cm^3), and right AGV (-0.88±0.20 cm^2). However, the adrenal volume in five patients was not reduced, despite significant weight loss postsurgery. In brief, obesity leads to increased AGV, and in some cases, this effect seems to be irreversible. We speculate that obesity causes permanently adrenal morphological changes (increased volume or hyperplasia), and under certain circumstances, it results in excessive aldosterone secretion via altered adipokines (leptin, CTRP1, etc.).

Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS

Use of Optimal Cutting Temperature Compound (OCT)-Embedded Adrenal Tumor Tissue for Intratumoral Steroid Hormone Profiling

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SAT-553

Use of optimal cutting temperature compound (OCT)-embedded adrenal tumor tissue for intratumoral steroid hormone profiling

Background: Primary aldosteronism (PA) is the most common cause of secondary hypertension, accounting for 5-8% of all hypertension. PA is most commonly attributed to an aldosterone-producing adenoma (APA) or to bilateral hyperaldosteronism (BHA). Mutations in the inward-rectifying K^+ channel (m*KCNJ5*), which increase autonomous aldosterone production, are most frequently detected in APAs. APAs with *mKCNJ5* display aberrant expression of aldosterone synthase (CYP11B2) and 17α-hydroxylase (CYP17A1), which are involved in aldosterone and cortisol synthesis, respectively. Co-expression of these enzymes results in the production of a set of "hybrid" steroids, which have been proposed as serum biomarkers. The relative production of hybrid steroids in adrenal tumors vs. adjacent normal adrenal (NA) tissue has not been investigated. **Objectives:** To determine the utility of OCT-embedded adrenal tumor tissue for steroid profiling. To use immunohistochemistry (IHC)-guided OCT tumor capture for intratumoral hybrid steroid profiling in *mKCNJ5* APA and NA tissue. **Methods:** OCT-embedded adrenal tissue from 9 patients (8 women, Age 45.9 ± 3.3 years) with APAs harboring known *KCNJ5* mutations were used for the study. Where available OCT-embedded normal adrenal (NA) tissue adjacent to APAs were used as controls (n=4). IHC was performed for CYP11B2 and CYP17A1 on OCT tissue allowing guided APA capture from serial sections. Steroids were extracted from APA and adjacent NA tissue, and quantified by liquid chromatography/tandem mass spectrometry. Steroids measured were normalized to the protein content of the extracted tissue. **Results:** Compared to NA, APA tissue demonstrated 23-, 5.6- and 6.4-fold higher levels of aldosterone, 11-deoxycorticosterone, and 18-hydroxycorticosterone, respectively ($P<0.05$). In addition, the "hybrid" steroid products, 18-oxocortisol and 18-hydroxycortisol, were significantly elevated in APA vs. NA ($P<0.01$). Conversely, the adrenal androgens dehydroepiandrosterone and 11-hydroxyandrostenedione were lower in APA as compared with NA ($P<0.05$). All m*KCNJ5* APAs were also found to co-express CYP11B2 and CYP17A1. **Conclusion:** IHC-guided *mKCNJ5* APA capture and steroid extraction identified a distinct intratumoral hybrid steroid signature that associated with co-expression of CYP11B2 and CYP17A1.These findings also demonstrate that OCT-embedded tissue can be used to accurately define intra-tissue steroid profiles, which will have application for steroid-producing and steroid-responsive tumors.