adrenal incidentaloma. MRI/MRA of the abdomen with and without gadolinium contrast showed a 2.5 x 2.6 cm left adrenal nodule, described as a benign lipid rich adrenal adenoma. Biochemical testing revealed no evidence of pheochromocytoma, hyperaldosteronism, or hypercortisolism. Follow-up CT scan in 2003 showed the left adrenal nodule was slightly larger (3.0 x 2.5 cm) but remained lipid rich (<10 HU). There was also a new sub-centimeter nodule in the left medial-posterior limb with similar appearance. In the interim, she was diagnosed with a melanoma on her back in 2003, which was resected without any evidence of invasion. In 2004, abdominal MRI with and without gadolinium contrast showed stable left adrenal nodules. As she continued to have persistent hypertension, uncontrolled with several medications, biochemical work-up for pheochromocytoma, hyperaldosteronism, and hypercortisolism was repeated and was again negative. Surveillance CT imaging in 2005 did not show any changes to her adrenal adenomas. In 2016, she presented to the emergency room with a hemorrhagic cerebrovascular accident. MRI of the brain was consistent with metastatic lesions. CT scan of the chest, abdomen and pelvis showed metastatic lesions in the lungs, liver, bone, and spleen. There was a new 8 mm right adrenal nodule noted with no changes in the left adrenal nodules. Biopsy of a subcutaneous chest wall nodule revealed metastatic melanoma. Thus, she was started on palliative immunotherapy with nivolumab. During her follow-up, she had a series of PET CT scans over a 6 month period, which showed increasing size (up to 4.3 cm) and FDG uptake in the left adrenal nodule. Surprisingly, the left adrenal nodule had a predominantly fatty density (mean of 5 HU) but with an area of hyperdensity which could represent either an adenoma with a coexisting metastatic lesion or angiomyolipoma. Biopsy of the left adrenal nodule revealed a metastatic melanoma. **Conclusion** This case describes a benign adrenal nodule coexistent with a metastatic lesion. As the patient had metastatic melanoma, a PET-CT was ordered. Melanoma is known to metastasize to the adrenal. This case serves to remind clinicians to perform a careful medical history as management and outcomes can be affected.

Adipose Tissue, Appetite, and Obesity

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

Serum CD163, but Not Gal-3, Predicts Response to Liraglutide Therapy in Obese Patients

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MON-605

Soluble CD163, But Not Gal-3, Predicts Response To Liraglutide Therapy In Obese Patients

Liraglutide is a GLP-1 Receptor Agonist licensed to treat T2DM and obesity. Soluble CD163 (sCD163) is a marker of macrophage activation, the integral immunological component in inflammation associated with obesity. Gal-3 is a β-galactoside-binding lectin that has been implicated in the development of cardiovascular diseases and insulin resistance. Recent studies have suggested that Gal-3 is raised in obesity with levels correlating with markers of inflammation.

In this study, we aim to elucidate if the levels of sCD163 and Gal-3 can be used to predict treatment outcomes of Liraglutide in obese patients.

Thirty-four obese patients (58.8% female; 44.1% diabetic) were enrolled for 12-week Liraglutide therapy. Anthropometric parameters were assessed before and after. Serum levels for sCD163 and Gal-3 were measured using ELISA.

Pre-treatment age (mean ± SD) was 52.41 ± 10.74y, BMI was 44.97 ± 7.71 kg/m^2, HbA1c was 47.18 ± 15.96 mmol/mol, sCD163 was 284059.20 ± 71859.88 pg/ml and Gal-3 was 6584.83 ± 3477.59 pg/ml. Post-treatment, BMI reduced to 43.19 ± 7.92 kg/m^2 (***p < 0.001***), HbA1c to 43.59 ± 16.00 mmol/mol (***p<0.001***), sCD163 to 249130.45 ± 57972.85 pg/ml (***p<0.001***) and Gal-3 to to 6254.23 ± 3282.66 pg/ml (***p<0.03***). We found that pre-treatment sCD163 levels correlate weakly with BMI and HbA1c (r=0.3 & 0.4) while Gal-3 correlates moderately with age only (r=0.36). Percentage of changes in HbA1c (ΔHbA1c) correlates strongly with ΔsCD163 (r=0.6). Levels of pre-treatment sCD163 correlates strongly with higher ΔsCD163 (r=0.7). Changes in BMI post-treatment (ΔBMI) is negatively correlated with initial sCD163 levels (r=-0.3) and is not correlated with ΔsCD163.

Liraglutide treatment leads to significant improvement in sCD163 and Gal-3 levels in obese patients. Patients with high HbA1c have high levels of sCD163. Reduction in sCD163 predicts reduction in HbA1c. Higher initial sCD163 levels predicts poor weight improvement. Patients most likely to have reduction in sCD163 are the ones with higher initial sCD163 levels. We conclude that sCD163 but not Gal-3 levels can predict response to liraglutide in obese patients.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Ectopic ACTH Syndrome Caused by Adenocarcinoma of Lung - a Rare Association with Rare Complication

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INTRODUCTION Ectopic ACTH constitutes 5-10% of ACTH dependent Cushing's syndrome. Evolution of symptoms can be rapid. Cortisol & ACTH is usually higher as compared to Cushing's disease. Imaging studies should be obtained for localization of the source of ACTH. Here, we present a case of Cushing's syndrome caused by ectopic ACTH production caused by adenocarcinoma lung, which has very rarely been associated with this syndrome **CASE** A 52-year-old male presented with 3-month H/O bilateral pedal edema & puffiness of face. He noticed bruises over his trunk & darkening of face for 2 months. There was a recent worsening of his HTN (requiring 3 antihypertensives) & recently diagnosed with DM. On examination patient had

cushingoid features, acne and ecchymosis. BP - 166/110 mm Hg. Proximal myopathy, Pedal edema, clubbing & cervical lymphadenopathy was present (largest node - 2.5cm). Fasting & postprandial blood sugars were 190 & 285 mg/dl respectively. Serum K^+ was 3.0 meq/L (3-5-5.0meq/L), 11pm serum cortisol was 51.6 mcg/dl (cutoff < 7,5mcg/dl), 8 am cortisol after overnight 1mg dexamethasone was 60.7 mcg/dl (cutoff<1.8mcg/dl). Serum ACTH(8am) - 178 pg/ml.(>20 pg/ml-ACTH dependent Cushing's) Biopsy of neck node revealed poorly differentiated adenocarcinoma. PET scan showed left lung upper & lower lobe masses. A diagnosis of ectopic ACTH syndrome was made, the source of which was adenocarcinoma of lung, which has been very rarely reported to be associated with it. Oral ketoconazole was started followed by Chemotherapy with paclitaxel & carboplatin. Within the next 7 days patient developed pleural effusion, neutropenia & worsened rapidly. BAL revealed Nocardia species, known to be associated with hypercortisolism. He was treated with appropriate antibiotics & supportive treatment but succumbed to septic shock. **CONCLUSION** If a patient presents with rapidly evolving symptoms of Cushing's syndrome, ectopic ACTH syndrome should be considered. The presence of wasting and weight loss, hypokalemic alkalosis, pedal edema & marked hyperpigmentation should also alert towards the diagnosis. Histopathological confirmation of malignancy is important, as in our case with ectopic ACTH where the source was an adenocarcinoma of the lung, of which only 5 cases have been reported till now (Ectopic ACTH more commonly associated with SCLC). Finally, in cases of severe hypercortisolemia, there should be a high index of suspicion for opportunistic infections including invasive fungal infections, Nocardiosis etc, so that specific antibiotic therapy can be initiated. Typical features like fever, leukocytosis can be absent. Treatment of underlying hypercortisolism with surgical/medical management prior to initiation of chemotherapy has been shown to reduce the frequency of infections.

Diabetes Mellitus and Glucose Metabolism

METABOLIC INTERACTIONS IN DIABETES

Praliciguat, a Clinical-Stage Soluble Guanylate Cyclase Stimulator, Improves Lipid Handling and Insulin Sensitivity in Diet-Induced Obese Mice

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SUN-662

Praliciguat (PRL) is a soluble guanylate cyclase (sGC) stimulator which in animal models distributes broadly to tissues and elicits hemodynamic, anti-inflammatory, antifibrotic, and metabolic effects. Here, we assessed metabolic effects of PRL in a mouse diet-induced obesity model.

Six-week-old male C57BL/6N mice were maintained on a low-fat diet (LFD, lean mice) or placed on a 60% high-fat diet (HFD, obese mice). At age 14 weeks, one group of obese mice was maintained on HFD (obese controls) and one group of obese mice was switched to HFD formulated with PRL to achieve a C_{max} similar to a 6-mg/kg oral dose (PRL-treated mice). After 38 days of treatment, an oral lipid tolerance test (LTT) was conducted. In a 2nd study under the same dosing paradigm, overnight fasted blood and organs were collected on day 28.

As reported previously (1), compared to obese controls, PRL-treated mice had lower fasting insulin (-28%), HOMA-IR (-26%) and triglycerides (-16%) as well as lower plasma triglycerides AUC after LTT (-34%). Gene expression and phosphorylated proteins associated with insulin pathways were measured in liver, skeletal muscle and white adipose tissue. PRL treatment normalized expression of genes involved in lipid handling (liver *Pnpla3*, *Slc27a1*, *Lpl*; muscle *Lipe*; white adipose tissue *Fdft1*, *Ppara*). Expression of proinflammatory genes (liver *Tnf*; muscle *Ccl2*; white adipose tissue *Akt1, Icam1*) was lower in PRL-treated mice than in obese controls. Liver insulin signaling was assessed by determining pAKT (T308) and pAKT (S473), markers of PI3K pathway activity and pERK, a marker of MAPK pathway activity. Compared to lean mice, pAKT (T308) and pERK were lower in obese controls, whereas pAKT (S473) was similar; PRL-treated mice had higher pAKT (T308) and pAKT (S473) compared to obese controls while pERK was unchanged. In skeletal muscle and white adipose tissue, levels of pVASP, a key mediator of the sGC pathway, were higher in PRL-treated mice than in obese controls.

In summary, PRL improved insulin sensitivity and lipids in diet-induced obese mice by affecting mechanisms of lipid handling, inflammation, and insulin signaling in key tissues associated with metabolism.

1. Author information excluded, 1924-P: Praliciguat, a Clinical-Stage sGC Stimulator, Improves Insulin Sensitivity, Lipid Tolerance, and Energy Utilization in a Mouse Diet-Induced Obesity Model Housed at Thermoneutrality. Diabetes, 2019. **68**(Supplement 1): p. 1924-P.

Adipose Tissue, Appetite, and Obesity

CNS, INFLAMMATORY, AND THERMOGENIC INFLUENCES OF BODY WEIGHT

MYOD1 Is Associated with Eosinophil-Mediated Browning of Subcutaneous Adipose Tissue

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A novel role for adipose tissue (AT)-resident eosinophils (EOS) in metabolism has been suggested. These data were obtained using genetic animal models with either whole-body overexpression of Interleukin-5 (IL-5Tg) leading to eosinophilia or gene ablation resulting in complete lack of EOS. These models limit the specificity of the findings. We hypothesized that AT-resident EOS play a specific role in whole-body metabolism. To this end, we generated a transgenic mouse model overexpressing human eotaxin-2 (hE2) under the control of a fat-specific aP2 promoter, which would exclusively recruit circulating EOS into AT (hEo2Tg), without any changes in the overall EOS numbers. Compared