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/m², 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 ± 7.82 kg/m² (\(p < 0.001\)), 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 6254.23 ± 9282.66 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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**MON-914**

**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. 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...