fluid administration. CXR was negative for acute pulmonary disease. All oral T2D agents were held and our patient was initiated on a DKA protocol based on ADA guidelines. Her EuDKA subsequently resolved with successful transition to a weight-based basal-bolus insulin regimen. **Conclusions:** There are no published case reports identifying patients with T2D developing euglycemic DKA precipitated only by a low carbohydrate diet and ertugliflozin initiation. We hypothesize that our patient's ketogenic diet lowered the threshold for a euglycemic ketoacid crisis resulting directly from the new addition of the SGLT2 inhibitor in the setting of pre-existing glucose toxicity. In patients considering, starting and being maintained on ertugliflozin or other SGLT2 inhibitors, the importance of effective, early and frequent dietary counseling with close follow-up cannot be overstated. Further, this report of EuDKA in a patient starting ertugliflozin supports that EuDKA is an SGLT2 inhibitor class risk.

Neuroendocrinology and Pituitary

ADVANCES IN NEUROENDOCRINOLOGY

The Protective Effects of Hepatocyte GH Receptor (GHR) Signaling Against Steatosis and Liver Injury Is Sexually Dimorphic and Autonomous of IGF1

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GH dysregulation contributes to the development of non-alcoholic fatty liver disease (NAFLD), however debate remains as to the relative contribution of the direct vs indirect effects of GH, via IGF1. Mouse models with congenital, liver-specific knockout of the GHR, JAK2 or STAT5, as adults exhibit steatosis, glucose intolerance, insulin resistance and white adipose tissue (WAT) lipolysis. It is believed that fatty liver is due to the dramatic reduction in circulating IGF1 altering systemic metabolism, due to loss of the insulin-like effects of IGF1 and the loss of IGF1 negative feedback to the pituitary leading to a rise in GH that promotes systemic insulin resistance and WAT lipolysis shifting the flux of fatty acids to the liver. In addition, low IGF1/high GH alters the development of other metabolically relevant tissues, which could indirectly contribute to the liver phenotype observed with congenital loss of hepatic GH signaling. To directly test the actions of GH on adult hepatocyte function, we developed a mouse model of adult-onset, hepatocyte-specific knockdown of the GHR (aHepGHRkd; 12 week-old, $GHR^{fl/fl}$ mice treated with AAV8-TBGp-Cre). aHepGHRkd enhanced hepatic *de novo* lipogenesis (DNL), rapidly leading to steatosis in males, but not females. In males, enhanced DNL and steatosis was sustained with age and associated with hepatocyte ballooning, inflammation and mild fibrosis. These changes occurred independent of severe systemic insulin resistance and WAT lipolysis, although the aHepGHRkd mice exhibit low IGF1/high GH similar to that of congenital models. To directly test the role of hepatocyte GHR signaling, independent of changes in IGF1, aHepGHRkd mice were treated with a vector expressing rat IGF1 targeted specifically to hepatocytes (AAV8-TBGp-rIGF1). Mice were fed standard chow diet and tissues collected 8m post-AAV. IGF1 replacement elevated plasma IGF1 in aHepGHRkd mice, resulting in a reduction in plasma GH and pituitary expression of Gh, Ghrhr and Ghsr, indicating negative feedback of IGF1 was restored. In male aHepGHRkd mice, IGF1 replacement reduced insulin and whole body lipid utilization and increased WAT, however it did not reduce steatosis or alter hepatic fatty acid composition indicative of DNL and had minimal effects on liver injury markers. RNAseq analysis of liver extracts showed IGF1 replacement also had no major impact on the differentially expressed genes observed after aHepGHRkd. These results demonstrate that steatosis, DNL and liver injury observed in male aHepGHRkd mice are autonomous of IGF1. Despite the fact that hepatic GHR protein levels were not detectable in both female and male aHepGHRkd mice, females maintained moderate levels of IGF1 and were protected from steatosis. The mechanism by which female mice are protected remains to be elucidated, however is consistent with clinical data indicating pre-menopausal women are resistance to NAFLD.

Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS II

The Importance of Early Diagnosis and Treatment of Primary Aldosteronism on the Progression of Chronic Kidney Disease, Compared With Essential Hypertension: A Retrospective Cohort Study

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<META NAME="author" CONTENT="Windows 사용자">Introduction: Primary aldosteronism(PA) has few clinical phenotypes and features, compared with other endocrine hypertension(HTN). Even though hypokalemia is a typical sign of PA, most of PA reveals normal potassium concentration. For that reason, PA is likely to undetected and underestimated and it may account for larger proportion of total HTN than we expected. However, it has known that PA has higher risk of renal complications than essential hypertension(EH) and has been controversy which treatment between medication and operation is better for renal protection of PA. Methods: We retrospectively reviewed the medial records of patients with PA and EH of a single medical center from January, 2009 to December, 2019. PA patients were divided into medical and surgical treatment groups. EH patients were distinguished from one that satisfied with case detection test, called non-confirmed PA. We excluded cases with other secondary HTN and baseline eGFR < 60 mL/min/1.73m^2. Results: Patients with PA(N=66) and patients with EH(N=514) were selected for analysis.