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 euaglycemic DKA precipitated only by a low carbohydrate diet and ertugliflozin initiation. We hypothesize that our patient’s ketogenic diet lowered the threshold for a euaglycemic 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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SUN-LB52
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<sup>−/−</sup> 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 Ghrs, 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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SUN-LB92
<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 go 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 medical 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<sup>2</sup>. Results: Patients with PA(N=66) and patients with EH(N=514) were selected for analysis.
Each baseline mean eGFR of patients with PA and EH indicated 91.2 ± 74.5 and 87.1 ± 19.7 mL/min/1.73m² and statistically insignificant differences (P = 0.1688) as well as baseline SBP (P = 0.5403) and DBP (P = 0.8691). However, in spite of treatment of PA and controlled BP, mean eGFR of PA patients was lower than one of EH patients and its difference was statistically significant showing 66.5 ± 14.2 and 94.6 ± 195.9 mL/min/1.73m² (P < .0001) at 2~5 years, 52.4 ± 17.9 and 77.6 ± 20.6 mL/min/1.73m² (P < 0.0004) at 6~10 years. Baseline mean eGFR of PA with normokalemia and hypokalemia respectively were 77.7 ± 11.6 and 98.9 ± 92.5 mL/min/1.73m² (P = 0.0269). Baseline mean eGFR of non-confirmed PA and EH were 82.5 ± 13.2 and 88.4 ± 21.1 mL/min/1.73m² (P = 0.0240). Although baseline mean eGFR of PA with surgical treatment was better than one with medical treatment, it was reversal after 2~5 years indicating mean eGFR of PA patients treated with operation, 62.9 ± 16.1 mL/min/1.73m² and one treated with spironolactone, 70.5 ± 12.6 mL/min/1.73m² (P = 0.0010).

Conclusions: This study support PA has worse effects on renal function than EH. PA is frequently unsuspected and undiagnosed because it hardly shows symptoms and signs. Many cases do not reveal main characteristics such as uncontrolled HTN and hypokalemia, so that patients with PA maybe have longstanding exposure to risk of CKD. Therefore it is necessary to do case detection test and rule out PA in initial hypertensive patients. In addition, more longitudinal study and research should be performed to decide personalized and adequate treatments for PA patients.

### Diabetes Mellitus and Glucose Metabolism

#### DIABETES DIAGNOSIS, TREATMENT AND COMPLICATIONS

**Improved Family Medicine Resident Diabetes Care Through Participation in a Diabetes Clinic**

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**SUN-LB116**

As the population ages and the prevalence of diabetes increases, the demand for endocrinology services, especially in underserved areas, will continue to exceed availability. Primary care residency training programs must prepare residents to care for high risk patients with diabetes who cannot access specialists. We hypothesized that resident participation in an inter-professional diabetes clinic run by primary care physicians would lead to improved diabetes care in resident patient panels.

A diabetes clinic was created in an existing primary care practice at a Federally Qualified Health Center in Eastern Kentucky. All non-pregnant, adult, Type II diabetes patients with a HgbA1C of 8.0% or greater were invited to participate in the clinic. Initial visits included evaluations by a dentist, mental health counselor, social worker, nutritionist, primary care provider, and pharmacist. Four first-year and four second-year family medicine residents rotated through the diabetes clinic and followed the patients as they saw each member of the health care team. On follow-up visits, a resident served as the primary care provider for each patient and participated in post-clinic meetings of the entire healthcare team. Resident patient charts were reviewed 3 months prior to the year-long intervention and data collected was compared to resident patient charts 3 months following the intervention.

Ninety patients served as the pre-intervention sample and 108 were in the post-intervention sample. Chi-square analysis showed a statistically significant increase in patients with A1C less than 8.0% pre (57.7%) to post (71.3%) (P = 0.0468). Overall, there were significant increases in all health-associated behaviors. Patients receiving eye exams increased from pre (29%) to post (66%) intervention significantly; z = 5.2, P < .001. Patients receiving a urine microalbumin test increased from pre (61%) to post (82%) intervention; z = 3.2, P < .001. Patients receiving dietary counseling increased from pre (54%) to post (79%) intervention; z = 3.6, P < .001. Patients receiving foot exams increased from pre (54%) to post (48%) intervention, z = 1.9, P = .03.

Resident involvement in a multidisciplinary diabetes clinic led by primary care physicians resulted in a statistically significant increase in HgbA1Cs < 8 among patients in their regular clinic and resulted in a statistically significant increase in their diabetic patients receiving eye exams, dietary counseling, foot exams, and urine microalbumin tests. This study suggests that teaching family medicine residents important diabetes care skills with an inter-professional team approach through the use of a diabetes clinic may be superior to standard educational practices.

### Thyroid

#### THYROID NEOPLASIA AND CANCER

**Stratifying the Risk of Malignancy in Indeterminate Thyroid Nodules**

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**MON-LB75**

Background: Thyroid cancer is the most common endocrine cancer globally, and accounts for 3.4% of all new cancer cases in the US. Although several clinical guidelines to detect/manage thyroid nodules are available, a great deal of controversy still exists around the optimal approach for diagnosis. Approximately 20-30% of cytology results from thyroid fine-needle aspiration (FNA) fall into one of three indeterminate diagnostic categories. In recent years, a number of molecular and gene mutation diagnostic tests have been developed to diagnose the indeterminate thyroid nodules in FNA specimens. However, nearly half of patients recommended for surgery based on these tests were found to have a benign nodule. Therefore, there is a need for a more accurate predictive and prognostic test for thyroid cancer. In case of Euthyroid Hashimoto Thyroiditis condition (EHT), where patient required a partial thyroxin replacement dose, having about 48% risk of thyroid cancer. However, there is still not a very accurate predictive marker for early detection of thyroid cancer in EHT. Invention