can be misleading. In the present case, we describe a patient with Type 1 Diabetes (T1D) on SGLT2 who underwent a strict low carb diet. **Case Report** A 70-year-old female with past medical history of unspecified diabetes mellitus and primary hypothyroidism presented to emergency room complaining of nausea and dizziness of four days with decreased oral intake. She was alert and oriented, normal weight (52 kg, BMI 20 kg/m²) with stable vital signs, except for mild tachypnea (22/min). Initial labs showed serum glucose 136 mg/dL, bicarbonate 10 mmol/L (normal 20-31), anion gap of 27, venous blood gas pH 7.1, B-hydroxybutyrate 8.8 mmol/L (normal 0.02-0.27), glucosuria > 500 mg/dL, and moderate ketonuria. Screening for ethyl alcohol and ethylene glycol was negative. Lactic acid, cardiac enzymes, renal and liver function tests were normal. She was diagnosed with diabetes mellitus at age 37, on insulin since then. No alcohol use. Her new primary care physician found an A1C of 9.0% for which metformin 1000mg oral twice a day and empagliflozin 12.5 mg oral daily were added and aspart insulin was discontinued. Daily glargine remained at 20 units daily. She was advised to lose weight for which she started a keto-diet 4 weeks prior to this presentation. She had lost 15 pounds since then accompanied by polyuria and polydipsia. Upon admission, she received IV insulin and IV fluids. An endocrinology consultation was requested for euglycemic DKA secondary to SGLT2 complication by starvation ketosis. Antibodies against glutamic acid decarboxylase were positive at 250 IU/mL (normal < 5). She was discharged on glargine, aspart insulins and oral medications were discontinued. **Conclusion** This case shows the importance of identifying the specific type of diabetes for appropriate individualization of therapy. Following a keto-diet in unrecognized T1D can trigger ketoacidosis in the setting of SGLT2 inhibitors leading to euglycemic diabetes ketoacidosis.

**Diabetes Mellitus and Glucose Metabolism**

**DIABETES COMPLICATIONS I**

*Nutritional Influences on One Carbon Metabolism Exacerbate Diabetic Cardiomyopathy and Nephropathy*

Ahmed W. Al-Humadi, MD1, Athina Strilakou, MD, PHD1, Hussam W. Al-Humadi, Assoc Professor2, Rafal Al-Saigh, Assistant Professor3, Charis Liapi, MD1.

1Medical School of Athens, Athens, Greece, 2University of Babylon, Babylon, Iraq, 3College of Pharmacy, Babylon, Iraq.

**SAT-630**

Choline (Ch) exerts a key role as methyl donor in the one carbon pathway and is an essential nutrient for the optimal development and function of a number of biological systems including the cardiovascular and urinary system. Ch-deprivation has been associated with heart function impairment, insulin resistance, abnormal fat metabolism and acute kidney injury. Diabetes mellitus is a common metabolic disorder with increased prevalence in aging and diabetic patients are of higher risk to develop heart and kidney failure. This study aims to investigate the impact of dietary Ch-deprivation on cardiac and renal function in a streptozotocin (STZ) experimentally induced diabetic setting. Twenty-four male adult Wistar rats, were randomly separated into four groups: control, choline deficient through choline deficient diet (CD), STZ induced diabetic (DM) and diabetic-choline deficient (DM+CD) group. After 5 weeks of dietary intervention, echocardiographic measurements, myocardium and kidney histological examination along with Vascular Endothelial Growth Factor-A (VEGF-A165) and Kidney Injury Molecule-1 (KIM-1) immunohistochemistry expression were performed. DM+CD rats demonstrated an exacerbation of myocardial inflammation and fibrosis accompanied by preserved ejection fraction but with an increased left ventricular (LV) wall tension index and velocity and a decreased LV posterior wall thickness compared to DM group. VEGF-A165 expression both in heart and kidneys was abruptly upregulated in the CD rats with a downward trend under the diabetes mellitus entity reaching significant downregulation in the renal tissue. KIM-1 expression was significantly increased under the insult of both choline deficiency and diabetes mellitus depicting a possible synergistic, though detrimental, effect compared to each condition alone. In conclusion, five weeks of dietary choline deprivation aggravates the inflammation and fibrosis in the heart and kidneys of diabetic rats leading to organ dysfunction. The structural impairment of the choline deprived diabetic heart with evidence of stiffness and dilation of the left ventricular cavity with preserved systolic function indicates the emergence of a new distinct phenotype of cardiomyopathy that combines features of the restrictive and dilated type at the same time. Moreover, in this setting the kidney injury gets worse implying that diabetic nephropathy might establish earlier and accelerate more quickly in choline deficiency conditions.

**Diabetes Mellitus and Glucose Metabolism**

**DIABETES TECHNOLOGY**

*The Effectiveness of Insulin Pump Therapy Compared to Multiple Daily Insulin Injections in Type 1 and Type 2 Diabetes Mellitus in a Predominantly African American Population*

Ahmad Imam, MD1, Hussam Alim, MD1, Bayan Chaker, MD1, Danaa Abushananab, MD1, Mohammad Talha Rauf, MD1, Berhane Seyoum, MD2.

1Wayne State University, Detroit, MI, USA, 2Wayne State Univ, Detroit, MI, USA.

**SAT-647**

Compared to multiple daily insulin injections (MDI), continuous subcutaneous insulin infusion (CSII) has proven to reach target HbA1c level with less frequent hypoglycemia, be more cost-effective, and improve quality of life. However, data on the effectiveness of CSII therapy in the African American population remain limited. The primary objective of our study was to compare the effectiveness of CSII therapy in lowering HbA1c levels in patients with type 1 diabetes (T1D) and type 2 diabetes (T2D) in a predominantly African American population. The secondary objective was to identify factors that affect the effectiveness of CSII. Participants were selected randomly from a list of patients currently receiving CSII at our institution’s diabetic clinic.
Each patient’s consent was obtained over the phone or during a visit to the clinic. Primary data were collected with a questionnaire, whereas additional data, including HbA1c levels before and after starting CSII, were collected from medical records. A total of 57 participants were enrolled in the study. African Americans represented 79% of the participants; 43% of the participants were unemployed, and 56% had an annual income of less than 20,000 USD. Since commencing CSII therapy, all participants achieved a decrease in mean HbA1c level from 9.7% to 8.0% (P = 0.001), and that of African American participants decreased from 9.8% to 8.2%. Increase number of individuals at home was associated with less reduction in HbA1c levels after starting CSII therapy (P = 0.02). Overall, satisfaction with CSII therapy was high, and 63% of participants reported being very satisfied with the treatment. The mean BMI among participants while using MDI was 32.6 kg/m² but significantly increased to 33.9 kg/m² (P = 0.01) while using CSII. The increase in mean BMI after starting CSII therapy was significantly higher in participants with T2D than in ones with T1D (P = 0.001). While receiving MDI, female participants had a significantly higher mean BMI than their male counterparts (P = 0.02); however, that difference became nonsignificant after they began CSII therapy (P = 0.06). The level of physical activity after starting CSII therapy did not alter the risk of increased BMI. The results of our interim analysis indicate the significant effect of CSII in lowering HbA1c levels in all diabetic patients regardless of sex, race, BMI, type of diabetes, marital status, employment status, level of education, adherence to diabetic diet, physical activity, duration on CSII, and use of other antidiabetic medications. The significant increase in BMI once CSII therapy commenced may reflect the increase in insulin dose among patients who were not adherent to insulin while receiving MDI. Patients need to be aware of that side effect, and additional interventions for weight management may be considered for overweight and obese patients planning to start treatment with CSII.

Cardiovascular Endocrinology
HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

Familial Homozygous Lipoprotein Lipase Defect Presenting with Recurrent Chylomicronemia Syndrome: Making a Case for Elective Plasmapheresis as an Adjuvant Treatment Modality.
Hafeez Shaka, MD, Ebizogie Edigin, MD, Sara Elizabeth Yap, MD, Iriagbonse Asemota, MD.
John H. Stroger Jr. Hospital of Cook County, Chicago, IL, USA.

SAT-565
Background
The chylomicronemia syndrome is a disorder characterized by severe hypertriglyceridemia and fasting chylomicronemia. Type Ia hyperlipoproteinemia is an extremely rare genetic disorder that results from homozygous deficiency in LPL activity. It is characterized by eruptive xanthomas, lipemia retinalis, memory disturbances, hepatosplenomegaly and frequent episodes of pancreatitis. Pharmacologic agents including fibrates, fish oils and statins have been used for treatment. Patients who fail pharmacologic therapy are usually treated with plasmapheresis. This case showed a patient in which joint decision making led to elective plasmapheresis to avoid chylomicronemia syndrome as an adjunct to medical therapy.

Clinical case
A 35-year-old lady without known medical co-morbidity presented with 2 weeks of dull abdominal pain radiating to the back. She developed nausea and vomiting which led to her presentation. There was no history of alcohol use, gall stones, diabetes mellitus or thyroid dysfunction. She was not on any medications. Physical examination was significant for BMI of 18, moderate abdominal tenderness, splenomegaly and an indurated rash in her legs. Laboratory investigation showed elevated lipase and triglyceride level of 3313 (normal 30-50) mg/dL. CT of the abdomen was consistent with acute interstitial pancreatitis. She was managed with insulin drip and fenofibrate and discharged 3 days later on fenofibrate, atorvastatin and long acting insulin. She developed another episode of acute pancreatitis while on medical therapy requiring readmission and initiation of insulin drip 2 months later. However, triglycerides trended upwards when the drip was stopped and symptoms of acute pancreatitis worsened. She subsequently underwent plasmapheresis which led to resolution of symptoms. Despite maximally tolerated pharmacologic therapy, she persistently has triglycerides above 4000s and persistent abdominal discomfort. Genetic testing confirmed homozygous defect in LPL gene. Following an outpatient Endocrinology visit, a decision was made to pursue elective plasmapheresis as an adjunct to therapy. She had on average 2 sessions monthly for 3 months with overall improvement in abdominal discomfort as well as significant improvement in triglyceride levels.

Conclusion
Familial chylomicronemia syndromes often require multimodal therapeutic approaches to prevent morbidity and complications. These include diet and pharmacologic therapy. Although plasmapheresis is often used during hospitalizations for hypertriglyceridemia induced pancreatitis refractory to diet and pharmacologic therapy, it was used in our patient electively and efficaciously to control hypertriglyceridemia and improve symptoms of chylomicronemia syndrome.

Reproductive Endocrinology
SEX, GENDER, AND HORMONES
Combining Clinical and Genetic Approaches in Diagnosing a Large Brazilian Cohort of Patients with 46,XY Differences/Disorders of Sex Development (DSD)
NATHALIA LISBOA ROSA ALMEIDA GOMES, PhD1, Rafael Loch Batista, MD PhD1, Mirian Yumie Nishi, pHD1, Antonio Marcondes Lerario, MD1, Thaisiana Evilen Silva, MSc1, Anna Flavia Figueredo Benedetti, MD1, Mariana Ferreira de Assis Funari, PhD1, José Antonio Diniz Paria Júnior, MD1, Daniela Moraes Silva, MD1, Luciana Ribeiro Montenegro, BSc1, Maria Tereza Martins Ferrari, MD2, Alexander Augusto Lima Jorge, MD,PHD1, Elaine Maria Frede Costa, MD,PHD1, SORAHIA DOMENICE, PhD1, Berenice Bilharinho Mendonca, MD1.