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SUN-003

Introduction: Although potential risk of DKA, ketogenic diet became popular among T1DM patients as proven to reduce the incidence of hypoglycemia, glycemic variability, and HbA1c (1).

Severe carbohydrate restriction had not been included among DKA precipitating factors in pregnancy since a minimum consumption of 175 g of carbohydrates a day is recommended for pregnant women by the IOM and ADA.

Proband: A 35-year-old pregnant woman (G2P1 at 29 weeks' gestation) was admitted to the ICU because of a 12-hour history of severe nausea, 4 vomiting episodes (stomach contain), weakness, and metabolic acidosis accompanied by low normal BG values for pregnancy. She had a history of bulimia nervosa since 15 years of age that was well-controlled by severe carbohydrate restriction prior to pregnancy, and T1DM since 19 years of age. Her pre-pregnancy carbohydrate daily intake was ~ 20–30 g and BMI 24.1 kg/m^2. Good glycemic control was established before conception and continued until delivery.

Upon admission, she was tachypneic and suffering from diffuse abdominal pain. Fetal monitoring and abdominal ultrasonography showed no significant changes, and the sonographic fetal weight was appropriate for gestational age.

The laboratory results revealed metabolic acidosis pH 7.23, bicarbonate 11.7 mmol/L, anion gap 22.5, and BG 78 mg/dL. Lactate and salicylate blood levels were normal. The blood β-hydroxybutyrate was highly positive and the patient was diagnosed with euglycemic DKA. She was managed with a continuous intravenous insulin infusion and fluid and electrolytes repletion.

The patient was discharged after three days of hospitalization only to return four days later because of the similar complaints with findings of blood β-hydroxybutyrate 7.7 mmol/L and BG levels of 61–65 mg/dL. Because of persistent ketonemia with low normal BG levels, patient was questioning about her dietary habits and revealed to maintain severe carbohydrate-restricted pre-conception eating manners. After repeated consultations by specialists for eating disorder, patient agreed to increase her carbohydrate intake to 120 gr per day.

Fetal brain MRI, fetal heart and brain ultrasound were performed to evaluate potential negative effects of ketones, and all were found normal. No major malformations were observed in the newborn, and a normal growth pattern was observed at 5 months of age.

Conclusion: This is the first report of a pregnant T1DM patient with long lasting bulimia nervosa excellently controlled by severe carbohydrate restriction prior pregnancy and occultly continued against medical advice through the pregnancy, causing repeatedly hypoglycemic-euglycemic DKA.

References: 1. Schmidt S, Christensen MB, Serifovski N, et al. Low versus high carbohydrate diet in type 1 diabetes: A 12-week randomized open-label crossover study. *Diabetes Obes Metab* 2019; 21:1680- 1688

Reproductive Endocrinology

REPRODUCTIVE ENDOCRINOLOGY: REPRODUCTIVE FUNCTION AND DYSFUNCTION ON DEVELOPMENT

Excessive Ovarian Sympathetic Activity Impairs Embryonic Development and Causes Reproductive and Metabolic Dysfunction

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MON-032

Nerve growth factor is a member of the neurotrophin family and within the ovary, it plays an important role in sympathetic innervation and in the development and maintenance of folliculogenesis. Despite its critical role, excessive levels of ovarian NGF may lead to ovarian pathology and to the development of features of polycystic ovary syndrome (PCOS), which is the most common endocrine disorder among women of reproductive age. Here, using a transgenic mouse model overexpressing NGF selectively in the ovary (17NF mice), we studied how ovarian sympathetic hyperactivity affects embryonic development and reproductive and metabolic function in adulthood. Firstly, we showed that ovarian NGF excess caused growth restriction in the developing female fetuses, which was driven by defects in the placenta function. Moreover, the 17NF fetuses experienced a reduction of germ cell number along with delayed gonocyte and primary oocyte maturation. The adult 17NF mice displayed irregular cyclicity and aberrant ovarian expression of steroidogenic genes and epigenetic markers. The ovarian sympathetic hyperactivity also led to increased systemic sympathetic outflow, indicated by increased circulating dopamine levels, and to metabolic dysfunction in adulthood. The 17NF mice had increased adiposity, impaired glucose metabolism and decreased energy expenditure. The subcutaneous and parametrial fat depots displayed impaired function due to ovarian NGF excess, wherein the subcutaneous fat increased mass by enhanced preadipocyte differentiation and enlarged adipocyte size, while the parametrial fat had smaller adipocyte size and a modest increase in stimulated lipolysis. These defects also led to hepatic steatosis. Overall, our findings indicate that ovarian sympathetic hyperactivity has deleterious effects on whole-body homeostasis and leads to impaired embryonic development and to reproductive and metabolic defects in adult life.

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID II

A Prospective Clinical Trial on the Efficacy of Lithium as Adjuvant Therapy to Radioiodine in the Treatment of Hyperthyroidism (Railit Study)

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