

SAT-568**Hypertriglyceridemia-Induced Pancreatitis in a Pregnant Female Treated with Plasmapheresis**

Background: Gestational hypertriglyceridemia can lead to critical and even life-threatening consequences to both mother and fetus. A well-known consequence is hypertriglyceridemia-induced acute pancreatitis. Few case reports described the successful management of triglyceride (TG) induced pancreatitis in pregnant women using plasmapheresis.

Clinical Case:

A 28-year-old primigravida patient, in the 29th week of gestation, was admitted with acute onset of epigastric pain and nausea for 24 hours. Laboratory findings were remarkable for an elevated serum lipase of 505 U/L (ref 23–300) and an abnormal lipid profile. Her total cholesterol was 1651 mg/dl and triglycerides (TG) from an undiluted sample was 1361 mg/dl. When a 1:5 dilution was performed the result was higher at >4000 mg/dl. She was transferred to the ICU for treatment of acute pancreatitis. She has no family history of hypertriglyceridemia. No MRI was obtained. Gemfibrozil, LovazaTM (omega-3-acid ethyl esters), and an insulin infusion were started but serum TG levels did not improve. On hospital day 2 she developed worsening tachycardia, tachypnea with laboratory findings of metabolic acidosis and hypocalcemia. As there was no reduction in triglyceride levels with medical therapy and her clinical status was deteriorating, the treating multidisciplinary team decided to initiate plasmapheresis. After one session, TG levels decreased from >4000 mg/dl to 1829 mg/dl and continued to decline to 721 mg/dl. Hospital day 6 her TG level rose to 1245 mg/dl prompting a second plasmapheresis. TG levels decreased to 770 mg/dl shortly after but rose the next day to 1365 mg/dl. She underwent a 3rd plasmapheresis after which her TG ranged from 400–700 mg/dl for the remainder of her hospitalization. On the day of discharge, her TG level was 733 mg/dl. She was advised to restrict fat intake and continue both gemfibrozil and LovazaTM but despite this her TGs again increased to 1693 mg/dl. From that point she started weekly sessions of plasmapheresis for a total of 8 sessions prior to an uneventful vaginal delivery at 36 weeks of gestation. One month later her lipid profile dramatically improved. Total cholesterol was 233 mg/dl and triglycerides were 304 mg/dl while on lipid lowering therapy.

Conclusion: Pancreatitis during pregnancy is associated with a high maternal and fetal death rate. Early treatment is important for the survival of the mother and fetus. Plasmapheresis is an alternative and safe treatment for cases that are not responsive to medical therapy. It can be administered safely to reduce triglyceride levels and diminish the systemic inflammatory response leading to a shortened hospital stay and better outcomes.

Adrenal**ADRENAL - TUMORS*****Dehydroepiandrosterone Sulfate (DHEAS) Levels Predict Weight Gain in Women with Anorexia Nervosa***

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SAT-167

Introduction: Anorexia nervosa (AN) and atypical AN (defined as weight loss and all the psychological features of AN but BMI>18.5 kg/m²) are serious disorders characterized by undernutrition and complicated by endocrine dysregulation. Predictors of recovery, including serum biomarkers, are lacking. Prior studies have suggested that higher urinary free cortisol (UFC) may predict weight gain in women with AN, but 24-hour urine collections are not feasible in a real-world setting. Like cortisol, the adrenal androgen dehydroepiandrosterone (DHEA) and its sulfated form DHEAS, which has a longer half-life, are stimulated by ACTH. We hypothesized that DHEAS levels would correlate with UFC and be a predictor of weight gain in women with AN.

Methods: We prospectively studied 34 women with AN and atypical AN, mean age 27.4 ± 7.7 years (mean ± SD), who received placebo in a randomized trial. AN and atypical AN were diagnosed by SCID. Baseline DHEAS and 24-hour UFC were measured by LC-MS/MS (Endocrine Sciences, Calabasas Hills, CA). Weight and body composition were assessed at baseline and 6 months later by DXA and cross-sectional abdominal CT at L4.

Results: At baseline, mean weight was 51.3 ± 4.9 kg. Of the 18 subjects who gained weight (range 0.1–10.3 kg), 28% were eumenorrheic, 39% amenorrheic, and 33% on oral contraceptives at baseline; baseline reproductive status was similar for subjects who did not subsequently gain weight. In the group as a whole, mean baseline DHEAS level was 173 ± 70 µg/dL (0.7 ± 0.3 times the mean normal range for age) and mean baseline UFC for subjects who completed testing (n=15) was 20 ± 18 µg/24h (normal range 0–50 µg/24h). Higher DHEAS levels at baseline predicted weight gain over 6 months (r=0.61, p<0.001), which remained significant after controlling for age, baseline BMI, OCP use, and SSRI/SNRI use (p<0.001); none of these covariates were predictors of weight gain. Baseline DHEAS levels predicted an increase in fat mass (r=0.40, p=0.03) and appendicular lean mass (r=0.38, p=0.04) by DXA, and abdominal fat by CT (r=0.60, p<0.001); the associations remained significant after controlling for the above factors. UFC did not predict change in weight (r=0.37, p=0.17) or body composition. DHEAS levels were positively associated with UFC (r=0.61, p=0.02).

Conclusion: In women with AN, higher DHEAS levels are a predictor of weight gain and increases in fat mass, skeletal muscle mass, and abdominal fat. Serum DHEAS correlates with UFC, a predictor of weight gain in prior studies. DHEAS may be a more practical biomarker of recovery, as 24-hour urine collections are challenging. Further studies are needed to determine whether higher DHEAS levels are a marker of global adrenal stress response and a reflection of higher cortisol levels, which may stimulate weight gain, or an independent predictor of weight gain in AN and atypical AN, perhaps through neuromodulation.