patients (mean age 28.5years, range 24–31) presented to us only in third trimester though two of them had deranged OGTT during first trimester screening elsewhere. The average HbA1c at the time of presentation was 5.425 (Range 4.8 to6.3). Only one of them was on insulin for a short term, which she stopped on her own as the readings continued to be in range even after stopping insulin. Of the 529 last trimester readings shared by the patients, 91.3% were within the target range of FBS< 92mg/dl, 1and 2 post meals <140 and 120 respectively, and pre meals < 95mg/dl. Three of them were induced (Two at 40 weeks and one at 38.4 weeks) for labour. The obstetrical outcomes were as follows: one had LSCS due to non-descent of head, second had vacuum assisted delivery, third had shoulder dystocia and fourth had normal delivery. The neonatal outcomes in respective cases were large for date baby with hypocalcaemia, transient HOCM with hypocalcaemia, shoulder dystocia with perinatal depression, PDA with respiratory distress. **Conclusion:** GDM should be intervened before the establishment of diabetic fetopathy. Missing the initial spikes in maternal glucose and the consequent surreptitious transfer of glucose to fetus can initiate such fetopathy. There is rerouting or detour of the maternal glucose, to the fetal system, for its disposal. Hence low or normal blood glucose readings in a known GDM patient should alert the clinician about the possibility of fetal glucose steal.[1] Highly suspicious cases can be monitored for polyhydramnios and fetal and maternal weight gain. The best ways to prevent this are pre-conceptional screening for IGT and universal screening for GDM with OGTT in each trimester.

Reference: 1.Desoye, Gernot & Nolan, Christopher. (2016). The fetal glucose steal: an underappreciated phenomenon in diabetic pregnancy. Diabetologia. 59. 10.1007/s00125-016-3931-6.aq`

Diabetes Mellitus and Glucose Metabolism

DIABETES IN WOMEN AND DURING PREGNANCY

Insulin Resistant Gestational Glucose Intolerance Is Associated With Adverse Perinatal Outcomes

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Background: Women with gestational diabetes mellitus (GDM) and gestational glucose intolerance (GGI, abnormal initial GDM screening test) and their infants have an increased risk of adverse perinatal outcomes including large for gestational age birth weight (LGA), pregnancy-related hypertension, neonatal intensive care unit (NICU) admission, and cesarean delivery. We expanded a prior analysis defining physiologic subtypes of GGI categorized by insulin resistance, insulin deficiency, or mixed pathophysiology. We aimed to determine if GGI subtypes are at differential risk for adverse outcomes. **Methods:** We applied homeostasis model assessment (HOMA2) to fasting glucose and insulin levels at 16–20 weeks' gestation to assess insulin resistance and deficiency, defined using the 50th percentile in 220 women with a normal glucose loading test (GLT) at 24–30 weeks' gestation. We defined GGI as GLT 1-hr glucose ≥140 mg/dL (n=245) and normal glucose tolerance (NGT) as GLT 1-hr glucose <140 mg/dL (n=1538). We classified women with GGI into subtypes according to the presence of insulin resistance and/or deficiency. We compared odds of adverse outcomes in each subtype to odds in women with NGT using logistic regression with adjustment for age, race/ethnicity, marital status, and 1st trimester BMI, plus infant sex in LGA models. **Results:** Of women with GGI, 49.0% had the insulin resistant subtype (IR, n=120), 30.6% had the insulin deficient subtype (ID, n=75), 15.9% had mixed pathophysiology (MP, n=39), and 4.5% had no evidence of IR or ID (n=11). GLT results and GDM diagnosis were similar among GGI subtypes. We found increased odds of LGA (primary outcome) in women with IR compared to women with NGT (OR 1.97 [1.17–3.32], p=0.01) in an unadjusted model; this was attenuated in an adjusted model with BMI (adjusted OR 1.43 [0.82–2.49], p=0.21). There was a trend toward increased odds of LGA in women with ID (adjusted OR 1.87 [0.92–3.80], p=0.09) and no increased odds in women with MP (adjusted OR 1.33 [0.50–3.57], p=0.57) compared to NGT. The odds of pregnancy-related hypertension in the IR subtype were increased (adjusted OR 1.68 [1.02–2.77], p=0.04) compared to women with NGT; women with ID (adjusted OR 0.91 [0.44–1.88], p=0.79) or MP (adjusted OR 1.13 [0.48–2.67], p=0.78) did not have increased odds. Neither infants of women with IR nor ID had increased odds of NICU admission overall, yet among women with BMI <25, infants of those with IR had increased odds of NICU admission compared to those of women with NGT (adjusted OR 3.37 [1.04–10.96], p=0.02); odds of NICU admission were not increased in infants of women with ID and BMI <25 (adjusted OR 0.50 [0.07–3.83], p=0.50). There was no difference in cesarean delivery across subtypes. **Conclusion:** Insulin resistant GGI is a high-risk subtype for adverse perinatal outcomes. Using HOMA2 to delineate subtypes may provide opportunities for a personalized approach to GGI/GDM.

Diabetes Mellitus and Glucose Metabolism

DIABETES IN WOMEN AND DURING PREGNANCY

Maternal Chronotype and Pregnancy Outcomes in Gestational Diabetes

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Introduction: Gestational diabetes mellitus (GDM) is an increasingly frequent complication of pregnancy and its presence is related to the development of adverse maternal and fetal outcomes. Pregnancy is deeply influenced by the circadian rhythm and the misalignment of the maternal