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High Insulin Resistance Cause Risk of Gestational Diabetes During Pregnancy

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Abstract

We prospectively evaluated differences in fasting- and oral glucose tolerance test (OGTT)-derived indices of insulin action in Caucasian and African-American pregnant women and compared them with obstetric outcomes. Pregnancy is a diabetogenic condition characterized by insulin resistance with a compensatory increase in β-cell response and hyperinsulinemia. Normal women compensate for insulin resistance by increasing insulin secretion to maintain normal glucose tolerance, pregnancy, occurs when insulin secretion is insufficient to compensate for the insulin resistance of pregnancy. Women with GDM have a limited ability to increase their insulin secretion. They have very blunt glucose-stimulated insulin responses compared with the augmented insulin responses of normal pregnant women.

Keywords: Oral glucose tolerance test; Insulin action; Hyperinsulinemia; GDM

Introduction

Women with GDM are at an increased risk of caesarean delivery, while their infants tend to experience higher rates of macrocosmic and shoulder dystocia. Abnormal fatal growth in diabetic pregnancy seems to occur with any elevation in the maternal glucose level. Pregnant women with elevated glucose levels have a higher risk of delivering increased birth weight infants [1], even when their glucose levels are below those diagnostic of GDM. Pregnant women with impaired glucose tolerance exhibit insulin resistance comparable to women with GDM, and have an increased risk of macrocosmic infants and other morbidities. It has been suggested that even minor degrees of increased glucose intolerance during pregnancy in women without GDM are related in a continuous and graded pattern with a significantly increased incidence of microsomal, caesarean section, pre-eclampsia and an increased need for neonatal intensive care unit admission, as well as greater length of maternal and neonatal hospital stay. Women of ethnic minority populations are at a greater risk for developing GDM. Found that the risk of GDM increased among non-Caucasian women in the Nurses' Health Study Cohort II. A significant interaction between glucose status and race was identified by Saldana et al. so their analyses were stratified by race looking at African-American and Caucasian mothers separately. Obesity-related risks during pregnancy were also found to vary by race [2], with obese AA women more likely to have adverse outcomes than obese Cau women. Other researchers report the racially disparate effects of impaired glucose tolerance and glucose levels on birth outcomes, with these conditions leading to higher levels of macrosomic babies among AA women, but not among cau women. Gravid as with GDM generally demonstrate higher degrees of post-pregnancy insulin resistance, β-cell dysfunction, higher BMI, central obesity, Notably, the diagnosis of GDM, based on glucose values from an antepartum OGTT, identifies a population of young women at elevated risk of developing diabetes later in life Reported that insulin sensitivity estimated from glucose and insulin levels during an OGTT was significantly improved compared with fasting values in pregnant women with normal glucose tolerance and GDM. This study examined the use of fasting- and OGTT-derived indices to measure insulin sensitivity and secretion in pregnant women in southern Louisiana with varying degrees of glucose tolerance. We further explored the potential use of these measures to define racially diverse risk profiles for these pregnant women and compare them with obstetric and perinatal outcomes [3]. The Institutional Review Board of the Woman's Hospital Foundation approved the protocol, and all participants gave written informed consent.

Method

The HOMA-IR generally provides a partial estimate of body insulin sensitivity because it mainly correlates with basal hepatic insulin resistance. This is why we also evaluated dynamic insulin sensitivity using the OGTT insulin sensitivity model of Matsuda and De Fronzo, which correlates with total glucose disposal, as extensively validated vs. the glucose clamp in various pathophysiological conditions. In pregnant women, ISOGTT exhibits better correlation with insulin sensitivity, derived using the glucose clamp, than did the HOMA-IR model. Insulin secretion was estimated after oral glucose loading by two methods; the corrected insulin response at glucose peak and the insulinogenic index divided by HOMA-IR which have been applied previously in pregnant women with and without GDM. The insulinogenic index was calculated as the ratio of change in insulin concentration to change in glucose during the first 30 min of the OGTT. The early-phase insulin release calculated by the IGI is used as a surrogate marker of first-phase insulin secretion measured during the glucose clamp. β-Cell compensatory capacity was calculated using the insulin sensitivity-secretion index defined as the product of SIOGTT and first-phase insulin release index [4]. The IS-SI expresses the overall ability of the β-cell to increase its release rate relative to insulin resistance in response to a glucose stimulus and reveals the progressive loss of β-cell function in individuals with IGT and GDM that was originally demonstrated using the disposition index calculated from the glucose clamp. An analogous mathematically derived measure; the insulin sensitivity secretion index

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has been utilized previously in both pregnant diabetic and non-diabetic women.

Obstetrical outcome information was obtained from a database that tracks labor and delivery data for all deliveries at the Woman's Hospital. Each woman's demographic information such as age and race was obtained from the computerized hospitalization record and confirmed with self-reported information. Neonatal data were abstracted by the review of maternal and new-born medical records. We recorded maternal pre pregnancy weight, parity, age, race, maternal drug or tobacco use, delivery mode, and obstetric history and infant's weight and height, gestational age at delivery, and birth weight for gestational age. According to the gestational age-specific weight distribution of the study population, infants were considered large-for-gestational age if their sex-specific birth weight for gestational age exceeded the 90th percentile of the US population fatal growth curves, or small for gestational age if their birth weight was below the 10th percentile [5]. Comparisons of clinical measures and insulin action indices between glucose tolerance groups were performed using one-way ANOVA for continuous parameters with means following normal distribution. Nonparametric Kruskal–Wallis test was carried out if appropriate. To evaluate differences in the impact of maternal race on glucose tolerance status category, data were further analyzed using a two-way ANOVA with race and glucose tolerance diagnosis as independent variables, with both main and interaction effects calculated. Regression analysis between fasting and OGTT-derived measures of insulin action was performed using Pearson product-moment correlation test. We further explored categorical differences in the distributions of family history of GDM, gestational age-specific weight, caesarean delivery rates and Apgar scores by glucose tolerance group using χ^2 tests for comparison. All *P* values were two-tailed.

Results

The consented participants, five pregnant women were excluded because of vomiting after the glucose load. The remaining patients completing the OGTT. In current clinical practice, women with GDM are identified on the basis of hyperglycemia on routine glucose tolerance testing in pregnancy. We report our institutional experience with the 100-g OGTT in which glucose and insulin levels were evaluated in the fasting state and after an oral glucose load in a cohort of pregnant women across the Glycemic spectrum. Adding insulin levels to the OGTT provided a clearer picture of the subtle metabolic abnormalities in both insulin sensitivity and β-cell function in this at-risk pregnant population. Other investigators suggested the use of fasting measure, such as HOMA-IR as an alternative but sensitive screening test for GDM, which avoids oral administration of glucose-containing solutions. While the HOMA-IR was correlated with the OGTT-derived insulin sensitivity assessment, we found the HOMA-IR provided a weaker predictive index compared to the glucose-stimulated ISOGTT measure. Furthermore, based on OGTT-derived indices of insulin secretion, it was quite clear that β-cell function progressively deteriorates with worsening of glucose tolerance, consistent with results obtained in other studies. Similar to the ISSI index first reported we calculated an IS-SI for each pregnant patient as a novel integrated measure of insulin sensitivity relative to insulin secretion. Specifically, we found that compared with NGT-abnGCT, women with GDM and GIGT had lower insulin sensitivity, poorer insulin secretion, and greater glycemia [6, 7]. Poor β-cell compensation for insulin resistance was evident in all pregnant women with GDM. Assessment and therefore all the pregnant women studied had some subtle impairment in insulin action. A further limitation is that the estimates of insulin action have been made on calculations based on the OGTT, and not by a "gold standard" test, euglycemic clamp study. The indices are being used in population studies as the clamp studies are not feasible in large numbers.

Discussion

Racial differences in basal and post-stimulation glucose homeostatic regulation were present over the spectrum of glucose tolerance. Non diabetic pregnant AA women were more insulin resistant but had higher baseline and glucose-stimulated insulin levels compared to Cau counterparts, findings that are consistent with earlier observations. One limitation to this finding of ethnic differences is the modest size of the AA pregnant group compared to the Cau group studied. However, these differences are not unique to pregnancy; AA children have higher fasting insulin, greater glucose-stimulated insulin levels and lower insulin sensitivity as assessed by a variety of methods. As was observed by other investigators, we found that pregnant AA women were more obese than. Even so [8], with further adjustments for obesity and body fat distribution, non-diabetic African-Americans continued to have lower insulin sensitivity but higher fasting and 2-h insulin levels and acute insulin response to glucose than whites. Although maternal GDM has long been associated with fetal macrosomia, our data support a growing number of studies that report a continuous effect of maternal glucose levels, even in the absence of GDM, on offspring birth weight and pregnancy outcomes observed among women with both normal and abnormal GDM screenings, increasing level of maternal glucose was linearly related to macrosomia risk. Undetectable glucose intolerance and a resistance to insulin are supposed mechanisms for this subgroup of patients [9]. Demonstrated that women with NGT-abnGCT are clearly distinct from those with NGT-normal GCT on the basis of lower insulin sensitivity and greater glycemia. We found that pregnant women who failed only the GCT showed a higher prevalence of delivering high birth weight infants and were significantly more often delivered by caesarean section than GDM and GIGT mothers [10]. Found that the new borns of false positives screening test mothers were heavier than those with NGT or GDM. Others have also reported that increasing maternal glucose levels within the normal non diabetic range were consistently related to larger offspring birth size and increased risk of interventional deliveries. In a prospective study of greater than 6000 women, Yogev et al. found a gradual increase in the rate of macrosomia, LGA and caesarean section in relation to increasing GCT severity categories in women without GDM. Moreover, they demonstrated that increased fetal weight and caesarean section rates in both obese and non-obese women are associated with higher degrees of carbohydrate intolerance. However, all pregnant women with GDM and GIGT at our institution receive interventional glucose-lowering therapy to improve obstetrical outcome. Standard obstetrical practice at our institution generally does not precipitate any specific intervention or treatment recommendations for women with normal glucose tolerance results after an abnormal GCT [11, 12].

Conclusions

In summary, antepartum OGTT screening identifies carbohydrate intolerance that occurs when insulin secretion is insufficient to compensate for the insulin resistance of pregnancy. The work presented here argues that insulin measures along with glucose determinations during oral glucose tolerance testing provide valuable screening test information which is racially disparate and future work should examine the predictive value of derived insulin action indices for the diagnosis of GDM risk in a large prospective ethnically-diverse

cohort. Hyperglycemia during pregnancy, less severe than overt DM, is associated with increased risk of adverse maternal and fetal outcome that is independently related to the degree of metabolic disturbance.

Acknowledgement

None

Conflict of Interest

None

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