The relationship between pregnancy weight gain and impaired glucose tolerance test

Gita Hatamizadeh 1*, Golaleh Farhad 2

1. Department of Obstetrics and Gynecology, Islamic Azad University-Tehran Medical Branch (IAUTMU), Tehran, Iran
2. Booali hospital, Islamic Azad University-Tehran Medical Branch (IAUTMU), Tehran, Iran

ABSTRACT

Impaired glucose tolerance has several adverse effects on growing fetus. In this study we evaluated the effect of excessive weight gain during pregnancy on the risk of glucose intolerance in pregnant women. A case-control study was conducted through which the glucose tolerance status after 100 gram oral glucose intake was compared between 60 pregnant women with maximum 10 weeks of gestation and excessive weight gain between gestational age 10 and 28 weeks and 60 pregnant women with excessive weight gain. Impaired glucose tolerance was defined as one high level in glucose tolerance test after 100 g oral glucose intake, and two high levels were considered gestational diabetes. In women their weight gain ratio exceeded unity, the chance of glucose intolerance was four times higher than that in the women with normal weight gain (%13 versus %3.3, P-value = 0.05). It can therefore be concluded that excessive weight gain during pregnancy elevates the risk of glucose intolerance in pregnant women.

Key words: Diabetes mellitus, impaired glucose tolerance, weight gain ratio, pregnancy

*Corresponding Author : Dr. Gita Hatamizadeh, Department of Obstetrics and Gynecology, Islamic Azad University-Tehran Medical Branch, Tehran, Iran. P.O.Box: 1663938641
Tel: +98 21 33798113 Fax: +98 21 22850731
e-mail: gita_hatamizadeh@yahoo.com
1. Introduction

Gestational diabetes mellitus (GDM) is known to have negative consequences for both mother and infant, including excessive fetal growth, intra-uterine fetal death (IUFD), intra-uterine growth restriction (IUGR), increased risk of birth injuries, and cesarean delivery with its related risks (Williams obstetrics 2010). Impaired glucose tolerance (IGT), a state of glucose intolerance of less severity than GDM, has also been shown to have similar risks in some populations (Tallarigo et al., 1986; Tan YY et al., 1986).

Irrespective of the pregnancy, weight gain is a recognized risk factor in the development of type 2 diabetes (Bray et al., 1992; Chan et al., 1994). Even the modest weight gain influences the progression along the continuum of glucose intolerance, increasing its severity (Colditz et al., 1995; Saldana et al., 2006). Several studies have shown the relationship between the increased adiposity and the increase of insulin resistance resulting in higher risk for type 2 diabetes (Olefsky, 1998; Bray et al., 1998).

Weight gain in early pregnancy, has been shown to be mostly composed of adipose tissue, with the majority of fat mass gained during the first 20-23 weeks of gestation (Dornhost et al., 1996; van Raaij et al., 1998; Saldana et al., 2006). Therefore, it is plausible that excessive weight gained during the first two trimesters of pregnancy can increase the risk of development of glucose intolerance (Saldana et al., 2006).

Previous studies have established a relationship between increasing pre-pregnancy weight and the GDM (Solomon et al., 1997; Rodrigues et al., 1999), however only limited studies have examined the relationship between weight gain during pregnancy and the risk of developing GDM (Saldana et al., 2006; Savitz et al., 1999). Moreover, to our knowledge there is only a single study to examine the relationship between pregnancy weight gain and the risk of developing IGT during pregnancy (Saldana et al., 2006). Understand the impact of excessive weight gained on glucose tolerance status allows for preventing excessive weight gain through extending the current recommendations of IGT and GDM prevention (Lu et al., 2001; Taffel et al., 1993).

This study aimed at examining the independent association of pre-pregnancy body mass index (BMI) and the amount of weight gain during pregnancy (prior to diagnosis of GDM) with glucose intolerance in a population of pregnant women who came to Booali hospital clinic for prenatal care.

2. Material and Method

2.1. Study population recruitment

The population study was selected among pregnant women coming to Booali hospital for prenatal care during March 2010 to March 2011, with a gestational age (GA) of below 10 weeks at their first visit. We excluded women who had overt diabetes mellitus, but women with history of GDM in their previous pregnancies were included. Data acquiring was done using a questionnaire asking patients the age, the parity, the smoking behavior and the history of GDM in prior pregnancies. Data on patient weight and height was locally measured and recorded at first visit and used to calculate the patient BMI (Kg/m2). All women were visited every four weeks on a regular basis. The patient BMI was updated at 28th gestational age.

2.2. Weight gain and weight gain ratio calculation

Maternal weight and height were measured in the clinic at the first visit and BMI was calculated accordingly. The calculated BMI data corresponding to the first visit (like pre-pregnancy BMI) (kg/m2) was categorized according to the Institute of Medicine (IOM) guidelines: underweight <19.8 kg/m2, normal weight= 19.8-26 kg/m2, overweight >26-29 kg/m2, and obese >30kg/m2 (Ogata, 1995; Institute of Medicine, 1990).

Regarding the small size of the underweight and normal-weight groups the related data was combined and used as a single category. Observed maternal weight gain was calculated by subtracting maternal weight at the GA of 10 weeks or less from the weight at the GA=28 weeks which corresponds to the end of the second trimester. Weight gain ratio was considered as the ratio of the observed weight gain to the recommended weight gain within the same period. The recommended weight gains were based on IOM BMI-specific recommendations (Table 1; Institute of Medicine 1990; Siega- Riz et al., 1994).

Biological relevance of the weight data was verified, and all women gained over 2.3 kg/week or lost less than 1.1 kg/week were excluded from the study and referred to more work-up.

Table 1. IOM- BMI recommendation: weekly rate of maternal weight change (kg) during 2nd & 3rd trimesters of pregnancy

<table>
<thead>
<tr>
<th>Primary BMI (kg/m2)</th>
<th>IOM- BMI recommended weight gain during 2nd trimester of pregnancy (kg/week)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 19.8</td>
<td>0.485</td>
</tr>
<tr>
<td>19.8-26</td>
<td>0.440</td>
</tr>
<tr>
<td>26-29</td>
<td>0.304</td>
</tr>
<tr>
<td>&gt;30</td>
<td>0.304</td>
</tr>
</tbody>
</table>

2.3. Case and control groups

The study population was divided into a control group of consisting 60 pregnant women with normal weight gain and a case group of involving 60 pregnant women with weight gain higher than normal recommendations (weight gain ratio higher than 1).
2.4. Case and control groups
All members of the population study were undergone OGGTT with 100 g of oral glucose. Cut points of 105, 165, 145 mg/ml was also used for one, two and three hour(s) post-parandial blood sugar values respectively to define the abnormal values (Williams obstetrics, 2010). GDM was defined as having two or more abnormal values from the OGGTT. There is currently no broadly recognized definition for the classification of IGT during pregnancy. For these analyses we defined IGT as having only one abnormal value from the OGGTT. Normal glucose tolerance (NGT) was defined as having no high values on the OGGTT.

2.5. Statistical analysis
Data management was performed using the SPSS16. Sample weights were incorporated into all analyses. Bi-variate analysis was performed using x² test to examine the significance of the difference between the categorical variables. One-way analysis of variance (ANOVA) was also used for overall comparison of continuous variables. Potential confounders were identified from the literature which included maternal age, height, pre-pregnancy BMI, smoking and parity. These factors were matched in two groups. Finally the interaction of the weight gain ratios in pregnancy was evaluated and found to be a significant predictor of the likelihood of developing IGT. In our samples nobody had GDM at GA of 28 weeks.

3. Results
The case and control groups were matched for age and parity (Table 2) hence the IGT incidence difference in our study was not related to age and parity. Also nobody in our study population was smoker. Primary BMI in women in both case and control groups had not significant difference (P-value > 0.05) but weight gain amounts and weight gain ratio were significantly different between the two groups.

13% of the case group and 3.3% of the control group had IGT (4 times higher in the earlier) whose difference was found to be significant (P-value = 0.05). In this study we did not find significant difference between the pre-pregnancy BMI and IGT during pregnancy (P-value = 0.6) perhaps because of the limited scope of the study. In the case group 6 members (10%) had a history of GDM in their previous pregnancies (albeit the women having IGT in this study were not the same persons), whereas nobody in the control group had such a history.

Table 2. Specificities of women in case and control groups

<table>
<thead>
<tr>
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<th>Case (n=60)</th>
<th>Control (n=60)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age (years)</td>
<td>26.4±3.77</td>
<td>27.0±5.91</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Maternal BMI (kg/m²)</td>
<td>24.8±3.66</td>
<td>24.0±3.46</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Maternal parity</td>
<td>3.2±1.1</td>
<td>3.1±1.2</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Smoker mother</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Maternal weight gain (kg)</td>
<td>9.4±2.3</td>
<td>4.4±2.2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Weight gain ratio</td>
<td>1.65±0.42</td>
<td>0.75±0.23</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>History of GDM</td>
<td>6 (10%)</td>
<td>0</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>IGT</td>
<td>8 (13%)</td>
<td>2 (3.3%)</td>
<td>&lt; 0.05</td>
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4. Discussion
The association between obesity and impaired glucose tolerance (which is a sign of impaired glucose metabolism) in pregnant and non-pregnant women is well recognized. If the weight gain is not restricted, IGT can progress to overt diabetes. Diabetes has many adverse effects in pregnancy for mother and the fetus such as excessive fetal growth, intra-uterine fetal death (IUGD), intra-uterine growth restriction (IUGR), increased risk of birth injuries, and cesarean delivery with all related risks (Williams obstetrics, 2010). Impaired glucose tolerance (IGT), a state of glucose intolerance of less severity than GDM, has also been shown to have similar risks in some populations (Tallarigo et al., 1986; Tan YY et al., 1986). Our aim in this study was to evaluate the effects of the excessive weight gain during pregnancy on glucose tolerance status. Because any positive association between these two factors is indicative of the role of excessive weight gain as one of the risk factors for IGT in pregnancy and GDM, if such an association is observed, the excessive weight gain in pregnant women must be prevented by prescribing a suitable regimen to prevent IGT and GDM.

At first the two groups in our study were matched for mean age and parity. Finally we found that the frequency of impair glucose tolerance was four times higher in the women with excessive weight gain during pregnancy relative to those with normal weight gain.

Literature survey identified a couple of related studies to ours. In the first study conducted on 952 pregnant women at the University of North Carolina (August 1995-May 2000), an association was found between the excessive weight gain during pregnancy (in already overweight women) and the increased risk of IGT. Similar results also obtained in another study enrolling 75 pregnant women (at department of OB-GYN at Lenox Hill hospital, Hackmon et al., 2007). The result of our study is in agreement with the previous related finding.
providing more support for the hypothetical presence of a causative relationship between excessive weight gain during pregnancy and the risk of IGT. Our results invoke motivation for conducting large-scale studies for further confirmation of this hypothesis thereby seeking for effective IGT preventing approaches.

5. Acknowledgement

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