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ESCME

Original Article

Searching for Optimal Serum Marker for Early Prediction of Gestational Diabetes

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ABSTRACT

Aim of work: To examine which serum marker (sex hormone binding globulin [SHBG], C- reactive protein [CRP], insulin, glucose) is accurate in early predicting the occurrence of GDM.

Patients and Methods: One hundred and fifty six pregnant mothers high risk to develop gestational diabetes mellitus (GDM) were included in the study. When GDM was excluded at booking setting (using OGTT), mothers are candidate for assaying fasting and non fasting sex hormone binding globulin (SHBG), fasting and non fasting quantitative C-reactive protein (CRP) and fasting insulin levels (from the same blood samples withdrawn during performing the OGTT). OGTT was repeated at 28 weeks and 36 weeks of gestation to diagnose GDM. According to the results of follow up OGTT, participant were divided into cases who developed GDM and those who did not develop GDM to assess the accuracy of each of the studied markers in predicting the occurrence of GDM in high risk mothers.

Results: sex hormone binding globulin levels (fasting and non fasting) were significantly lower among women who subsequently developed GDM compared with the control group ([276.9±78.7nmol/L vs 322.4 ± 71.6nmol/L, P=0.001], [261.5 ± 66.7 nmol/L vs 299 ± 59.7 nmol/L, P=0.001], respectively). No difference was detected in C-reactive protein levels (fasting and non fasting) (P=0.33, 0.349), fasting insulin (P=0.082), fasting glucose levels (P=0.119), between the study group who subsequently developed GDM and the control group.

Conclusion: SHBG can be used as an early marker to identify the group at highest risk for subsequent GDM allowing earlier intervention and possible benefits to the mothers and their offspring.

Key Words: Sex Hormone-Binding Globulin, C-reactive protein, Insulin, Gestational- Diabetes .

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INTRODUCTION

Gestational diabetes (GDM) complicates 3-5% of gestations and is a risk for pregnancy related maternal and neonatal morbidity, including an increased need for cesarean delivery, increased rates of fetal macrosomia, and an increased incidence of birth trauma *(Kjos, et al. 1999)*. Furthermore, although glucose control improves immediately after delivery, mothers who have had GDM are at risk for having type 2 diabetes develop later in life *(Damm, et al. 1995)*. Recognizing these associations, the American College of Obstetricians and Gynecologists (ACOG) and the American Diabetes Association (ADA) have each recommended routine screening for GDM during pregnancy *(American College of Obstetrics and Gynecol-*

ogy 2001; American Diabetes Association 2006). As currently recommended by the ACOG and the ADA, serum screening for GDM typically begins between 26 and 30 weeks of gestation but, as many use a 2-step process (a 50 g glucose loading test [GLT], followed by a 100 g glucose tolerance test for those with a positive GLT), testing for GDM may not be completed until 32 weeks. This timing leaves only a brief window for implementing interventions designed to improve outcome. Because a first step in treating GDM is usually to instruct patients to follow a special diet and monitor their blood sugars closely, the window for intervention may be further narrowed if these first steps prove ineffective, and insulin or oral agents to control blood sugar are ultimately necessary. Some patients with GDM will not start on these medications until the last 4-6 weeks of their pregnancy. Earlier detection of women at risk for GDM might allow earlier intervention, interventions potentially reducing either later diagnosis of GDM or its associated morbidities. GDM results from a combination of increased insulin resistance and impaired pancreatic insulin secretion and women with a history of GDM are at significantly increased risk of developing type 2 diabetes in the future *(Wolf, et al. 2003)*. Type 2 diabetes is an important cause of cardiovascular disease, and the latter is a leading cause of death among patients with diabetes. Inflammation, marked by increased serum levels of C-reactive protein (CRP), is an important independent risk factor for cardiovascular disease. Inflammation is also associated with insulin resistance, and prospective studies indicate that increased inflammation at baseline is an independent risk factor for the future development of type 2 diabetes *(Wolf , et al. 2003). Thadhani et al. (2003)* and others tested the hypothesis that increased CRP levels measured early in pregnancy, are associated with the subsequent development of GDM. Insulin is an important regulator of serum sex hormone-binding globulin (SHBG) concentration which works by inhibiting its production in hepatocytes. Low SHBG level is associated with increased insulin resistance and hyperinsulinemia. *Bartha et al. (2000)* compare maternal serum SHBG level between normal and gestational diabetic pregnant women and studied the relationships between SHBG, SHBG/insulin and SHBG/glucose ratio and several endocrine, metabolic and clinical parameters *(Bartha, et al. 2000)*. Also, *Thadhani et al. (2003)* examined the association between early pregnancy levels of sex hormone binding globulin and subsequent gestational diabetes as they demonstrated that sex hormone binding globulin offers a potential early marker to target women who are at risk for gestational diabetes. *Smirankis et al. (2005 a)* and others have demonstrated that a variety of serum markers of both insulin resistance (e.g. , sex hormone-binding globulin [SHBG], homeostasis model assessment index [HOMA]) and inflammation (e.g. , high sensitive C-reactive protein [hsCRP]) measured in the first and early second trimester are associated with the later diagnosis of GDM *(Smirnakis, et al. 2005 a)*. *Smirnakis et al. (2007)* performed comparison of different serum markers as predictors for GDM to choose the optimal one and they found that first-trimester sex hormone-binding globulin appeared to be the optimal marker. In this current study, we thought to determine which serum marker (2 non-fasting measures [SHBG,CRP] 4 fasting measures [SHBG, CRP, insulin, glucose]) is accurate in early predicting the occurrence of GDM in high risk mothers to allow earlier intervention.

MATERIALS AND METHODS

Participants:

During the period from May 2007 to December 2007, one hundred and fifty six pregnant mothers high risk to develop gestational diabetes mellitus (GDM) were included in the study.

All participants were attending the routine antenatal clinics, Maternity Hospital, Kasr El-Aini Teaching hospital, Cairo University, Cairo, Egypt. The inclusion criteria were gestational age from 8 – 16 weeks, past history of GDM, family history of diabetes mellitus, maternal age over 30 years, multiparous mothers and multifetal pregnancy. All cases with bleeding in early pregnancy, bad obstetric history, fetal congenital anomalies, overt diabetes mellitus (gestational or pre gestational), polycystic ovarian disease, body mass index > 35kg/m^2, present steroid therapy, irritable bowel disease, and women with septic focus were excluded from the study. Six women refused to participate at booking and 22 women were lost for follow up so were excluded from the study (thus the original pool was 178 mothers).

Study protocol:

After explanation of the whole procedure, all participants were investigated clinically, then a 2 hours glucose tolerance test (OGTT) was done as described by the American diabetes association *(American Diabetes association, 2006).* After 8 hours fasting, Ten ml of fasting venous blood samples were taken from each subject participating in the study, the serum were left to clot and fasting blood glucose was carried out immediately by glucose oxidase method *(Siest, 1981)* then 75g glucose load was given orally followed by with drawing another 2 blood samples one and two hours later for assaying blood glucose (About 2 ml of blood was taken on fluoride from each subject in the study for determination of blood glucose-by-glucose oxidase) *(Siest, 1981)*. The maximum normal values were 95 mg/dl for fasting sample and 180 mg/dl and 155 mg/dl for the one and two hours samples. If any two values were abnormal, GDM is diagnosed. When GDM was excluded at booking setting, the mother is now candidate for assaying fasting and non-fasting sex hormone binding globulin (SHBG), fasting and non-fasting quantitative C-reactive protein (CRP) and fasting insulin levels (from the same blood samples withdrawn during performing the OGTT). Candidate samples were stored at -20°C till the time of assay.

Follow up :

All participants were followed up throughout their pregnancy and managed according to the local protocols set be the corresponding unit. OGTT was repeated at 28 weeks and 36 weeks of gestation to diagnose GDM. Only samples from participants who completed the study were assayed. Serum SHBG was done on Immulite 2000 analyzer (DPC, Cirrus Inc., Los Angeles, USA), using solid-phase enzyme chemiluminescent immunoassay *(Jayagopal, et al. 2004)*. The kit was supplied from DPC (96th street, Los Angeles). C-reactive protein (CRP) was determined immediately by rapid latex agglutination procedure *(Saxtad, et al. 1970).* Insulin was determined using radio immunoassay *(Westgard, et al. 1981)* and the kit was supplied from Linco Diagnostic (Linco Research Inc., 6 research park, Missouri, USA). According to the results of follow up OGTT, participant were divided into cases who developed GDM and those who didn't develop GDM (control group) to assess the accuracy of each of the studied markers in predicting the occurrence of GDM in high risk mothers.

Statistical analysis:

Quantitative data were statistically described in terms of mean ± standard deviation (± SD) while qualitative data were described using frequencies (number of cases) and percentages. Comparison of quantitative variables between women who developed GDM and those who didn't was done using Student t test for independent samples. For comparing categorical data, Chi square (χ^2) test was performed. Yates correction was used instead when the expected frequency is less than 5. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using computer programs Microsoft Excel version 7 (Microsoft Corporation, NY, USA) and SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) statistical program.

RESULTS

Baseline characteristics of the study groups are presented in table (1) The average gestational age at the time of booking when serum was sampled for CRP, SHBG, insulin, glucose was taken was between 8-16 weeks. There was no difference in maternal age, parity, BMI, blood pressure among case and control subjects. Also there was no difference in gestational age at delivery, mode of delivery, neonatal birth weight, apgar score among case and control subjects

Table (2) presented measured biomarkers of the study groups at 8-16 gestational age. Serum levels of SHBG (fasting and non-fasting) were significantly lower in women who subsequently developed GDM compared with controls (P value = 0.001).

There was no difference in serum levels of CRP (fasting and non-fasting), insulin levels (fasting), fasting glucose level, fasting glucose: insulin ratio among case and control subjects

Table 1: Baseline characteristics of the study groups

	GDM (n = 47)	Control (n = 109)	P value
Maternal age (years)*	31.16 ± 5.2	29.67 ± 5.6	0.121
Gestational age at booking (weeks)*	11.92 ± 3.12	11.27 ± 3.27	0.250
Parity:†			0.902
- Primiparas	16 (34.04)	36 (33.03)	
- Multiparas	31 (65.96)	73 (66.97)	
BMI (kg/m^2)*	31.54 ± 4.1	30.88 ± 3.9	0.341
Systolic blood pressure (mmHg)*	116.76 ± 17.62	117.01 ± 18.22	0.937
Diastolic blood pressure (mmHg)*	71.53 ± 14.73	69.94 ± 15.12	0.545
Gestational age at delivery (weeks)*	38.77 ± 1.42	39.24 ± 1.61	0.085
Mode of delivery: †			0.745
- Vaginal	39 (82.98)	91 (83.49)	
- Cesarean	8 (17.02)	18 (16.51)	
Neonatal birth weight (g)*	3256.4 ± 357.6	3316.7± 389.1	0.365
Apgar score at one minute*	7.86 ± 0.94	7.88 ± 0.99	0.907
Apgar score at 5 minutes*	9.05 ± 0.71	9.06 ± 0.64	0.931

GDM: gestational diabetes mellitus; SHBG: sex hormone binding globulin; CRP: c-reactive protein
* Data are described in mean ± SD
† Statistically significant difference

Table 2: Measured biomarkers of the study groups at 8–16 gestational weeks

	GDM (n = 47)	Control (n = 109)	P value
Fasting serum SHBG (nmol/L)*	276.9 ± 78.7	322.4 ± 71.6	0.001†
Non fasting serum SHBG (nmol/L)*	261.5 ± 66.7	299 ± 59.7	0.001†
Fasting serum CRP (mg/L)*	11.2 ± 6.1	10.1 ± 6.6	0.330
Non fasting serum CRP (mg/L)*	11.1 ± 6.3	10.02 ± 6.7	0.349
Fasting serum insulin (uU/ml)*	13.49 ± 4.8	11.96 ± 5.1	0.082
Fasting glucose (mg/dl)*	76.8 ± 7.9	74.6 ± 8.1	0.119
Fasting glucose : insulin ratio*	5.53 ± 0.67	5.74 ± 0.7	0.084

- GDM: gestational diabetes mellitus; BMI: body mass index
* Data are described in mean ± SD

† Data are described in number of cases (%)

DISCUSSION

In the present study, we found that serum levels of fasting and non-fasting sex hormone binding globulin (SHBG) measured at 8-16 weeks of gestation were lower in high risk mothers who subsequently developed GDM.

This finding was independent of the influence of maternal age, parity, weight which in previous studies have been associated with GDM. SHBG, a glycoprotein synthesized by the liver, binds circulating estradiol and testosterone *(Anderson, 1974)*.

Estradiol and thyroid hormone are the principal stimuli for hepatic SHBG secretion whereas insulin, prolactin ,androgens, and growth hormone suppress hepatic SHBG secretion *(Hampl, et al. 1996)*.

Increasing insulin levels suppress hepatic SHBG secretion ,even in the face of increasing estradiol levels; among non pregnant women, SHBG correlate inversely with glucose tolerance, insulin levels, and insulin resistance as determined by the hyperinsulinemic clamp *(Sherif, et al. 1996)*.

Given that insulin resistance precedes the onset of type 2 diabetes mellitus by several years, it is not surprising that reduced SHBG levels are associated significantly with the development of type 2 diabetes mellitus *(Buchanan, 2001)*. In addition, when insulin sensitivity is increased pharmacologically, SHBG levels rise.

Importantly, unlike other markers of insulin resistance, SHBG is reliable in the non fasting state and exhibits no diurnal variation which renders SHBG a unique marker of insulin resistance *(Hamilton, et al. 1995)*.

Bartha et al. (2000) examined SHBG levels in the third trimester and found lower levels among women with GDM compared with control subjects, but these investigators did not examine first-trimester levels.

Thadhani et al. (2003) based on the fact that women with history of GDM have evidence of chronic insulin resistance and that insulin resistance and its surrogate markers are evident early in pregnancy, suggests that lower levels of SHBG can be detected in the first trimester, well before the clinical diagnosis of GDM and at a time when intervention may alter clinical outcomes.

Smirnakis et al. (2007) showed that SHBG measured in first trimester was more strongly associated with GDM than SHBG measured in the second trimester.

Our study agree with the studies conducted by *Thadhani et al. (2003) ; Smirnakis et al. (2007)* regarding the lower levels of SHBG measured at 8-16 weeks of gestation in mothers who subsequently developed GDM.

In our study fasting SHBG levels were 276.9 ± 78.7 nmol/L in the study group (n=47) who subsequently developed GDM, compared to serum levels of 322.4 ± 71.6 nmol/L in the control group (n=109).

Regarding our results of non-fasting SHBG levels, among the study group who subsequently developed GDM levels were 261.5 ± 66.7 compared to levels of 299 ± 59.7 in the control group.

We provide evidence that SHBG levels (fasting and non-fasting) were significantly lower in mothers who subsequently developed GDM (P value=0.001)

In this current study we found no difference in serum levels of C -reactive protein (fasting and non-fasting levels) between the study and control subjects, fasting CRP levels in the group who subsequently developed GDM were 11.2 ± 6.1 mg/L, whereas the fasting levels in the control group were 10.1 ± 6.6 mg/L with P value = 0.33, non-fasting levels in the study and control groups were 11.1 ± 6.3,10.02 ± 6.7 mg/L ,respectively with P value = 0.349.

Our finding disagree with several studies *(Thadani, et al. 2003; Smirnakis, et al. 2005a, 2007).*

One potential explanation for this difference is that most of our patient whether at risk for developing GDM or not have one or more factor increasing CRP levels such as irritable bowel syndrome, hidden septic foci.

Thadhani et al. (2003) identified an association between first-trimester inflammation, marked by increased CRP levels, and subsequent risk of GDM, this effect was independent of established risk factors for GDM such as age, multi parity, smoking. The increased risk of GDM was attenuated, however, when they adjusted for BMI, which was highly correlated with CRP. These observations highlight an important association between obesity, inflammation, and risk of developing GDM that previously has not been studied in detail.

Increased serum CRP is a sensitive index of systemic inflammation that has emerged as an independent risk factor for cardiovascular disease *(Rost, et al. 2001)*.

Growing evidence similarly implicates inflammation in the pathogenesis of type 2 diabetes.

Inflammation is directly correlated with insulin resistance, as measured by the intravenous GTT or the euglycemic-hyperinsulinemic clamp ,and with other features of the insulin resistance syndrome, such as obesity, hypertensionand microalbuminuria *(Frohlich, et al. 2000)*.

Furthermore, women with the polycystic ovary syndrome, a disorder characterized by insulin resistance and increased risk of type 2 diabetes and cardiovascular disease, display significantly increased CRP levels compared with eugonadal women *(Kelly, et al. 2001).*

Our study shows no difference between the study and control groups regarding fasting serum insulin, fasting glucose, and fasting glucose: insulin ratio.

Fasting serum insulin levels were 13.49 ± 4.8 μU/ml in the study group who subsequently developed GDM compared to 11.96 ± 5.1 μU/ml in the control group (P value=0.082).

Fasting glucose levels were 76.8 ± 7.9 mg/dl and 74.6 ± 8.1 mg/dl in the study and control groups ,respectively (P value=0.119).

Our results disagree with the results of other studies *(Smirnakis, et al. 2007)* as they reported that the risk of subsequently diagnosed GDM increased significantly with increasing fasting glucose and fasting insulin (which is a proxy for insulin resistance). Also *Smirnakis et al. (2005b)* found that women in whom GDM was diagnosed at 24–28 weeks of gestation demonstrated higher levels of fasting glucose, fasting insulin, and HOMA at ~ 17 weeks in pregnancy compared with women who had normoglycemic pregnancies. Also they found that the risk of GDM increased significantly with increasing HOMA and fasting glucose levels, independent of other variables that are known to be associated with GDM. *Sacks at al. (2003)* reported similar findings to *Smirnakis et al. (2005b)*.

In conclusion SHBG can be used as an early accurate marker to identify the group at highest risk for subsequent GDM allowing earlier intervention and possible benefits to the mothers and their offspring.

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الملـخص العـربـــي

تحديد المصل الامثل فى التشخيص المبكر لحدوث مرض سكرالحمل

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الهدف من الدراسة : تحديد المصل الامثل فى التشخيص المبكر لحدوث مرض سكرالحمل.

تصميم الدراسة : اجرى البحث على ١٥٦ سيده حامل من الاكثر عرضه لحدوث مرض السكر اثناء حملهم.

عند التأكد من عدم وجود خلل فى منحنى السكر فى الدم اجرى قياس مستوى هرمون ملزمه الجلوبيولين رابطه الجنس(صائم ومفطر) وكذلك قياس مستوي تفاعل البروتين ج (صائم ومفطر) ومستوى الجلوكوز والانسولين(صائم) فى الدم وتم تقسيم الحالات بناء على قياسات منحنى السكر فى الدم عند الاسبوع الثامن والعشرون والاسبوع السادس الثلاثون من الحمل الى مجموعتين الاولى الامهات اللاتى اصبن بمرض سكر الحمل والثانيه الامهات اللاتى لم يصبن بمرض سكر الحمل و ذلك حتى يتثنى لنا تحديد التحليل الامثل بالمصل للتشخيص المبكر لحدوث مرض سكر الحمل .

النتائج : اثبتت الدراسه انخفاض فى مستوى هرمون ملزمه الجلوبيولين رابطه الجنس (صائم ومفطر) فى الامهات اللاتى اصبن بمرض السكر اثناء الحمل مقارنه بالامهات اللاتى لم يصبن بمرض السكر اثناء الحمل.

وهذه النتيجة توضح مدي أهمية قياس مستوى هرمون ملزمه الجلوبيولين رابطة الجنس فى التشخيص المبكر لحدوث مرض السكر اثناء الحمل فى الامهات الاكثر عرضه لهذا المرض وبذلك يمكن عن طريق التدخل المبكر حمايه الام والجنين من حدوث مضاعفات مرض السكر اثناء الحمل.

و أوضحت الدراسة عدم وجود اى اختلاف فى نسبه تفاعل البروتين ج (صائم ومفطر) ومستوى الجلوكوز والانسولين (صائم) فى الامهات اللاتى تعرضن لمرض السكر اثناء الحمل مقارنه بالامهات اللاتى لم يصبن بمرض السكر اثناء الحمل.