Early Detection of Gestational Diabetes Via Measurement of First Trimester Maternal Serum Uric Acid

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Abstract

Rational and Background: Diabetes is the most common medical complication of pregnancy. It occurs in between 5 and 10% of all pregnancies. In pregnancy, uric acid is correlated with insulin resistance in women with gestational hypertension and is higher at 24-28 weeks gestation in women diagnosed with GDM compared to women without diabetes.

Objectives: To find out if the elevated first trimester uric acid is associated with development of gestational diabetes mellitus.

Study Design: This prospective observational study was conducted in Damanhur Educational Hospital Department of Obstetrics and Gynaecology, between March 2015 and November 2015. A total of 80 pregnant women (<13 weeks gestation) were included in the study. Baseline assessments included age, BMI, parity, number of previous abortions and serum uric acid level. Subjects were re-assessed at 24-28 weeks for Gestational Diabetes Mellitus (GDM). Data was systematically recorded and analysed to test for associations of various parameters with GDM.

Results: Elevated first-trimester uric acid concentration was correlated with an increased risk of developing GDM. The risk of developing GDM was higher if first-trimester uric acid was ≥3.11mg/dl [OR 6.11, 95% CI (2.33 to 8.11)]. Also there was a significant positive correlation between serum uric acid and BMI (r=0.66, p=0.008). The mean BMI was significantly higher in women who developed GDM when compared to women who did not develop GDM [29.23±3.11 Kg/m² vs. 27.8±3.97Kg/m², respectively, p=0.021].

Conclusions: Serum uric acid elevation in first trimester has a significant correlation with development of gestational diabetes mellitus.

Key Words: Uric acid – Body mass index – Gestational diabetes mellitus.

Introduction

GESTATIONAL Diabetes Mellitus (GDM) is defined by the World Health Organization (WHO) as a carbohydrate intolerance resulting in hyperglycemia of variable severity with onset or first recognition during pregnancy [1]. Large studies have found that GDM occurs in 2.2-8.8% of pregnancies, depending on the ethnic mix of the population and the criteria used for diagnosis [2]. Prevalence of GDM is increasing all over the world [3]. Risk factors for the development of GDM include obesity, older age, family history, previous history of GDM, poor obstetric outcomes, ethnicity, polycystic ovary syndrome and more recently noted hypertension [4]. GDM carries considerable health risks for both the fetus and the mother, for the infants; they include an increased risk of macrosomia, birth injuries such as (shoulder dystocia, bone fracture and nerve palsies), hypoglycemia and hyperbilirubinaemia [5]. Women with GDM are at increased risk of developing preeclampsia with an increased chance of need for induction of labour and caesarean section, gestational diabetes is also a strong risk factor for later development of type 2 diabetes. Preeclampsia has been frequently reported as a complication of gestational diabetes but the relation between these two conditions is not well understood. Several studies suggest underlying common pathophysiology [6], including insulin resistance, chronic inflammation and endothelial dysfunction. There are common risk factors, such as elevated body mass index and advanced age have been noted for each of the two conditions [7]. Uric acid is the end product of purine catabolism catalysed by the enzyme xanthine oxidase/dehydrogenase [8]. In non-pregnant women, uric acid is associated with insulin resistance and
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is an independent risk factor for development type 2 diabetes within 10 years [9].

In pregnancy, uric acid is correlated with insulin resistance in women with gestational hypertension and is higher at 24-28 weeks gestation in women diagnosed with GDM compared to women without diabetes [10]. Two mechanisms have been hypothesized by which uric acid can cause insulin resistance, the first proposed that uric acid causes endothelial dysfunction and decreases nitric oxide production by the endothelial cell. In animals, insulin action on glucose uptake into cells in the skeletal muscle and adipose tissue is dependent on nitric oxide thus, decreases in nitric oxide lead to decreased glucose uptake and the development of insulin resistance [11]. Another mechanism by which uric acid may induce insulin resistance may be that uric acid causes inflammation and oxidative stress in adipocytes, which is a contributor to the development of metabolic syndrome in mice [12].

**Aim of the work:**
To find out if the elevated first trimester uric acid is associated with development of gestational diabetes mellitus.

**Patients and Methods**

The study was conducted as part of thesis submitted to the Faculty of Medicine, Benha University.

In partial fulfillment of the requirements for the Degree of Master.

It was conducted in the Obstetrics and Gynecology (OBG) Department of Damnhour Educational Hospital, Egypt. This is a large teaching general hospital the study was conducted during the period between March 2015 and November 2015. The sample size was ascertained after a power calculation in consultation with the department’s statistician. The study sample was drawn from amongst pregnant women who had registered with the antenatal clinic of the hospital.

A convenience sample of 80 consenting subjects was recruited for the study. Patients with pre-existing diabetes mellitus, hypertension, gout, connective tissue disorders or on medications causing hyperuricemia (e.g., pyrazinamide, ethambutol, levodopa and theophylline) were excluded.

At the initial antenatal assessment (which included a detailed history and an examination), the following details were also collated for the purposes of this study: Age, parity, weight, height, BMI, baseline serum uric acid and a fasting blood sugar.

All participating subjects were reviewed at around 24-28 weeks of gestation, when an Oral Glucose Tolerance Test (OGTT) was done. Daily 150 carbohydrates diet is allowed for 3 days then fasting blood sugar is determined after overnight fasting of 8-14hrs. then the patient is then given 100gm of glucose solution to drink within a 5 minute time frame. readings are taken hourly for the next 3hrs and a curve is drawn. Patients were considered to have GDM if 2 or more of the 4 values exceeded the following [13]:

- Fasting blood glucose level > 95mg/dl.
- 1 hour blood glucose level > 180mg/dl.
- 2 hour blood glucose level > 155mg/dl.
- 3 hour blood glucose level > 140mg/dl.

Data was systematically recorded in a proforma and analysed descriptively using t-tests, ANOVA and chi-squared tests (as appropriate) for statistical significance. A ROC analysis was done to identify a cut-off for serum uric acid levels to predict subsequent GDM.

**Results**

80 subjects were enrolled in the study. The mean age of the sample was 29.51 years (SD=5.17). The majority was primiparous. Baseline BMI (Body Mass Index) was calculated using weight and height data. The majority had a BMI between 18-38.5, with the mean BMI being 26.17 (SD=4.21).

The median serum uric acid level in the entire sample was 2.7mg/dL. 4 of the 80 subjects developed GDM on follow-up (5%).

There were no significant differences between women who developed GDM and women who did not develop GDM regarding parity and no. of previous abortions. The maternal age and mean BMI was, however, significantly higher in women who developed GDM when compared to women who did not develop GDM [29.02 ±4.39Kg/m² vs. 27.8±3.97Kg/m², respectively, \( p<0.001 \) (Table 1).

In order to answer the study question, the data was analysed to explore the association of serum uric acid levels with the development of GDM (Table 2). The results show that a significantly higher proportion of women with higher serum uric acid levels developed GDM, compared to those with lower serum uric acid levels (\( p<0.05 \)).
Finally, a ROC analysis was done to ascertain a suitable serum uric acid cut-off so as to be suggested as a marker for subsequent development of GDM.

A cut-off serum uric acid level of $\geq 3.11$mg/dL was found to have a sensitivity of 100%, specificity of 74.2%, and appositive predictive value 65.6% and a negative predictive value of 100% for the development of GDM.

The distribution of cases that developed GDM at a cut-off uric acid level of 3.11mg/dL is shown in (Table 3).

Fig. (1): Difference between women who developed GDM and women who did not regarding their characteristics.

Fig. (2): Box-plot chart showing difference between women who developed GDM and Women who did not regarding serum uric acid at recruitment.

Fig. (3): ROC curve for serum uric acid in relation to an outcome of GDM.

Fig. (4): Scatter-plot showing correlation between serum uric acid and age.

Fig. (5): Scatter-plot showing correlation between serum uric acid and BMI.
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Table (1): Difference between women who developed GDM and women who did not regarding their characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Women who developed GDM (n=4)</th>
<th>Women who did not develop GDM (n=76)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years):</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>37-39</td>
<td>21-37</td>
<td>0.001 *</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>27.41±4.69</td>
<td>27.55±5.26</td>
<td>(S)</td>
</tr>
<tr>
<td><strong>BMI (Kg/m²):</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>25-36.0</td>
<td>22.4-36.0</td>
<td>0.021 *</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>29.02±4.39</td>
<td>27.8±3.97</td>
<td>(S)</td>
</tr>
<tr>
<td><strong>Parity:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>0-4</td>
<td>0-5</td>
<td>0.458 **</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>(1-2)</td>
<td>(1-2)</td>
<td>(NS)</td>
</tr>
<tr>
<td><strong>No. of previous abortions:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>0-2</td>
<td>0-3</td>
<td>0.521 **</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>(0-1)</td>
<td>(0-2)</td>
<td>(NS)</td>
</tr>
</tbody>
</table>

*: Analysis using independent student's t-Test.  
**: Analysis using Mann-Whitney's U-Test.  
NS : Non-Significant.  
HS : Highly Significant.

Table (2): Difference between women who developed GDM and women who did not regarding serum uric acid at recruitment.

<table>
<thead>
<tr>
<th></th>
<th>Women who developed GDM (n=4)</th>
<th>Women who did not develop GDM (n=76)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum uric acid at recruitment (mg/dl):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>3.90-4.4</td>
<td>1.5-3.8</td>
<td>0.001 *</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>(3.33-4.2)</td>
<td>(2.31-3.4)</td>
<td>(HS)</td>
</tr>
</tbody>
</table>

*: Analysis using Mann-Whitney’s U-Test.

Table (3): Binary logistic regression analysis of serum uric acid as predictor of GDM.

<table>
<thead>
<tr>
<th>Serum uric acid as predictor of GDM</th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3.11mg/dl (n=22)</td>
<td>4/22</td>
<td>6.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&lt;3.11mg/dl (n=58)</td>
<td>0/58</td>
<td>8.11</td>
<td>HS</td>
</tr>
</tbody>
</table>

OR : Odds Ratio.  
95% CI : 95% Confidence Interval.  
HS : Highly Significant.

Discussion

Diabetes Mellitus (GDM) is one of the most common medical disorders found in pregnancy. Rates can range from 2 to >10%, and sometimes much higher, depending on the population being tested and the diagnostic criteria being used [14].

Gestational diabetes mellitus is associated with an increased risk of complications for mother and child during pregnancy and birth. Among those complications are shoulder dystocia and birth injuries, respiratory distress syndrome, caesarean section, and preeclampsia. Fetal macrosomia is associated with gestational diabetes and is a surrogate for many of the complications [18]. Epidemiological research suggests that women who have gestational diabetes have an increased risk of type 2 diabetes later in life [16].

Uric acid is the end product of purine catabolism catalyzed by the enzyme xanthine oxidase/dehydrogenase [17]. Hyperuricemia is associated with components of metabolic syndrome and it has been debated for a while to be a component of it [18]. Historically, the elevated level of uric acid observed in the metabolic syndrome has been attributed to hyperinsulinemia, since insulin reduces renal excretion of uric acid. Hyperuricemia, however, often precedes the development of hyperinsulinemia, obesity, and diabetes [19].

The current study was done to determine if the uric acid level in the first trimester of pregnancy can be used as a predictor of subsequent GDM. This study was conducted in Damnhour Educational Hospital during the period between March 2015 and November 2015. It was a prospective observational study which included 80 pregnant patients who were regularly attending the outpatient clinic for routine antenatal care. In this current study, elevated first-trimester uric acid concentration was correlated with an increased risk of developing GDM. The risk of developing GDM was higher if first-trimester uric acid was ≥3.11mg/dl [OR 6.11, 95% CI (2.33 to 8.11)].

These results were similar to that obtained by some studies [20]. They had found that serum uric acid was significantly correlated with insulin resistance. This coincides with the result of Laughon et al., [21]. Who studied 1570 pregnant patients whom uric acid was measured at mean gestational age of 8.9±2.5 weeks. The primary outcome was GDM, diagnosed between 24-28 weeks of pregnancy by 3-hour glucose tolerance test using carperter and coustan criteria or by a 1-hour value of ≥200mg/dl. Almost half (46.6%) of the women with GDM had first-trimester uric acid concentrations in the highest quartile (≥3.57-8.30mg/dl). Women with uric acid in the highest quartile had a 3.25-fold increased risk (95% confidence interval, 1.35-7.83) of developing GDM after adjustment for body mass index and age. This effect was concentration dependent as risk increased with increasing uric acid quartiles (p≥.003).
Our findings were consistent with [22] who found, in a large cross-sectional study of 53,477 nonpregnant adults, serum uric acid was positively correlated with fasting serum glucose and insulin resistance, as well as features of the metabolic syndrome, including waist circumference, low high-density lipoprotein cholesterol, hypertriglyceridemia, hypertension, and fasting glucose ≥110 mg/dl. However our study did not assess the other criteria of the metabolic syndrome. Hyperuricemia has also been demonstrated to be a risk factor for developing type 2 diabetes [23].

In contrast, Gungor et al., [24] compared the relationship between serum uric acid, creatinine and albumin levels in pregnant women with normal glucose tolerance and gestational diabetes mellitus. A total of 112 patients were evaluated, 56 of whom had gestational diabetes. All of the patients had single estimations of serum uric acid, creatinine, albumin and liver enzymes carried out on booking between the 24th and 28th gestational weeks. The women were followed-up throughout pregnancy. They found that single estimations of serum uric acid and albumin concentrations were not significantly different between a normal pregnant group and a GDM group. This is contrary to the findings of our study, this may be due to the differences in the number of cases and gestational age of serum uric acid estimations.

In this current study, there was statistically significant relationship between age and development of GDM.

In Queen Mary Hospital, the University of Hong Kong, Lao et al., [28] have reviewed the prevalence of GDM, diagnosed by the World Health Organization criteria in over 15000 singleton pregnancies managed from 1998 to 2001. The pregnancies were categorized according to maternal age, into 6 categories, ≤20 years, 20-24 years, 25-29 years, 30-34 years, 35-39 years, and >40 years. (96.6%) patients continued their pregnancies beyond the first trimester, and the number (percentage of total) from the youngest to the oldest cohort were 318 (2.0%), 1,713 (10.8%), 4,446 (28.1%), 5,457 (34.5%), 3,279 (20.7%), and 614 (3.9%), respectively. There was a significant difference and positive correlation in the prevalence of GDM, increasing from 1.3, 2.5, 6.2, 10.3, 21.7, and 31.9%, respectively, from the youngest to the oldest cohort (p<0.001). The risk for the older cohorts was significantly increased as follows: 25-29 years, 2.59 (1.84 -3.67); 30-34 years, 4.38 (3.13- 6.13); 35-39 years, 10.85 (7.72-15.25); and >40 years, 15.90 (10.62-23.80). There was no significant difference for the ≤20 years cohort. These finding indicates that the risk of GDM becomes significantly and progressively increased from 25 years onwards. This supports the American Diabetes Association recommendation on the use of age 25 years as the cutoff for screening and the observation that maternal age 25 years is the factor most predictive of GDM. The results of this study were coincides with our result.

In this study, the mean BMI was significantly higher in women who developed GDM when compared to women who did not develop GDM [29.23 ±3.11Kg/m² vs. 27.8 ±3.97Kg/m², respectively, p-0.02 1]. In Harvard Medical School, Boston, USA in 2008, Jenny et al., [26]. Studied 1733 patients with singleton pregnancies enrolled in Project Viva. They examined the associations of first trimester diet, with results of glucose tolerance testing at 26-28 weeks gestation. 91 patients developed GDM and 206 patients had Impaired Glucose Tolerance (IGT). They concluded that prepregnancy Body Mass Index (BMI) is a strong predictor for development of GDM. This study coincides the same results as our study.

In Szczecin University, Poland, Ogonowski et al., [27]. Studied 1121 patients with GDM who were referred to the outpatient clinic between January 2001 and December 2005. The control group consisted of 1011 healthy pregnant women. All had singleton pregnancies. Significant relationships between pregravid BMI and GDM were found and BMI was the strongest predictor for GDM treated with insulin. This study states the same results as our study.

In this present study, there was a significant positive correlation between serum uric acid and BMI (r=0.66, p=.008). This agrees with the result of Laughon et al., [28] who found that maternal prepregnancy BMI increased linearly with increasing uric acid quartile (p<0.01 for trend) and was associated with uric acid with an r² of 0.16 (p<0.001).

In several epidemiological studies, a close relationship between hyperuricemia and hypertension, heart failure and other cardiovascular diseases has been reported, and correlations between hyperuricemia and obesity, dyslipidemia, and diabetes have also been reported. Hyperuricemia is also associated with the markers of metabolic syndrome, including obesity and dyslipidemia [29]. Hyperuricemia has been associated with increasing Body Mass Index (BMI) in several studies and are even apparent in the adolescent youth [30]. These studies state the same results as our study.
Hyperuricemia may also be present in the metabo-
litical syndrome in people who are not overweight
or obese. In one study only 5.9% of subjects with a
normal BMI and a uric acid level of less than
6.0 mg per deciliter had the metabolic syndrome;
in contrast, 59% of subjects with a normal BMI
and a uric acid level of more than 10 mg per deciliter
had evidence of the metabolic syndrome. These
are contrary to the findings of our study. This may
be due to differences in technique, time and sample
size.

In this present study, serum uric acid at recruit-
ment was insignificantly associated with a fasting
blood glucose ≥ 105 mg/dl among women who de-
veloped GDM [OR 0.66, 95% CI (0.395 to 0.57)].

In a large cross-sectional study of 53,477 non-
pregnant adults, serum uric acid was positively
correlated with fasting serum glucose ≥ 110 mg/dl.
The results of this study were against ours.

**Conclusions and Recommendations:**

- Elevated first-trimester uric acid concentration
  was correlated with an increased risk of develop-
ing GDM. The risk of developing GDM was
  higher if first-trimester uric acid was ≥ 3.11 mg/dl
  [OR 6.11, 95% CI (2.33 to 8.11)].
- There was a significant positive correlation be-
  tween serum uric acid and BMI (r = 0.66, p = 0.008).
- The mean BMI was significantly higher in women
  who developed GDM when compared to women
  who did not develop GDM (29.23 ± 3.11 Kg/m²
  vs. 27.8 ± 3.97 Kg/m², respectively, p = 0.021).

So, we recommend:

- Serum uric acid measurement in 1st trimester of
  pregnancy as a predictor for GDM and screening
  of GDM at 24-28 weeks gestation.

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ملخص العربية

شملت الدراسة 80 سيدة من المتزوجات لمتابعة الحمل في مستشفى دمنهور التعليمي (العيادات الخارجية).

وهدف هذا البحث إلى دراسة العلاقة بين ارتفاع معدل حمض الوريك في القياس الأول من الحمل والإصابة بمرض الوليد السكري في الحمل.

كانت الحالات تتبعها المعايير الآتية:
• عمر الحمل أقل من 12 أسبوع.
• حمل لجنين واحد.
• عدم تعرضことができる يسوي مزمن بضغط الدم.
• لا تعاني من ارتفاع السكر في الدم.
• لا تعاني من أمراض القلب أو الكبد أو الغدة الدرقية أو النقر.
• تاريخ مذكور كاملاً وفحص إكلينيكي كامل ثم عمل موجات فوق صوتية لتقييم عمر الحمل واستعداد أي حمل به أكثر من جنين.

تم قياس مستويات حمض الوريك في أمصال السيدات الحوامل مرة واحدة في الفترة ما قبل 12 أسبوع عن طريق أخذ عينة وريدي وفصلها لأخذ عينة من الحمل وحفظها في درجة حرارة 2–6 مئوية حتى يتم تحليتها.

بعد ذلك عندما يبلغ عمر الحمل ما بين 20-24 أسبوع تم عمل تحليل مسحي للكشف عن مرض الوليد السكري في الحمل وذلك عن طريق:
• تناول 100 جم جلوكوز بالفم. بعد ذلك تم تحليل مستوي الجلوكوز بالدم بعد ساعتين. عندما كانت نتيجة التحليل أكثر من 140 مجم/100 مل كانت السيدة أكثر عرضة لإصابة بمرض الوليد السكري. لذلك تم حساب مستوي الجلوكوز بالدم بعد ساعتين ثم تناول 100 جم جلوكوز بالفم.
• بعد ذلك تم تحليل مستوي الجلوكوز بالدم بعد ساعتين ثم بعد ساعتين وبعد ثلاث ساعات. وتم تشخيص السيدة بأنها تعاني من مرض الوليد السكري عندما تتعدى قيمة تحليل أو أكثر القيم الآتية:

• مستوي الجلوكوز بالدم بعد ساعتين 180 مجم/100 مل.
• مستوي الجلوكوز بالدم بعد ثلاث ساعات 210 مجم/100 مل.

نتائج البحث:
• توجد علاقة ذاتات إحصائية بين مستوى حمض الوريك في القياس الأول من الحمل وظهور مرض الوليد السكري في الحمل.
• توجد علاقة ذاتات إحصائية بين معدل كتلة الجسم ومرض الوليد السكري في الحمل.
• توجد علاقة ذاتات إحصائية بين مدة الحمل وظهور مرض الوليد السكري في الحمل.
• لا توجد علاقة ذاتات إحصائية بين عدد مرات الحمل وعدد الأطفال للسيدة الحامل وظهور مرض الوليد السكري في الحمل.

توصيات:
• قياس نسبة حمض الوريك في القياس الأول من الحمل كمؤشر لحذف مرض الوليد السكري والكشف عنه ما بين 20-24 أسبوع من الحمل.