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URIC ACID AND BODY MASS INDEX (BMI) IN FIRST TRIMESTER AS PREDICTOR FOR GESTATIONAL DIABETES MELLITUS

By

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ABSTRACT

Background: Gestational diabetes mellitus (GDM) is defined as glucose intolerance of variable degree with onset or first recognition during pregnancy. The precise mechanisms underlying gestational diabetes remain unknown. The hallmark of GDM is increased insulin resistance. Pregnancy hormones and other factors are thought to interfere with the action of insulin as they bind to the insulin receptors. Uric acid is the end product of purine catabolism catalyzed by the enzyme xanthine oxidase/dehydrogenase.

Objective: The aim of the study was to determine whether uric acid level in the first trimester of pregnancy can be used as a predictor of subsequent development of GDM or not.

Patients and methods: This was a prospective observational cohort study which included 520 pregnant women in their first trimester who regularly attended the outpatient clinic for routine antenatal care. This study was conducted at Alexandria Police Authority Hospital from November 2018 to May 2020.

Results: Elevated first-trimester uric acid concentration was correlated with an increased risk of developing GDM. The risk of developing GDM was 4-fold higher if first-trimester uric acid was ≥ 3.05 mg/dl [OR 3.8, 95% CI (1.99 to 9.15)]. There was a significant positive correlation between serum uric acid and BMI (r = 0.378). The mean BMI was significantly higher in women who developed GDM when compared to women who did not develop GDM [29.02 ± 4.39 Kg/m2 vs. 26.04 ± 4.16 Kg/m2, respectively]. There was no statistically significant relationship between age and development of GDM. Serum uric acid at recruitment was insignificantly associated with a fasting blood glucose ≥ 105 mg/dl among women who developed GDM [OR 0.833, 95% CI (0.493 to 1.47)].

Conclusion: 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.

Keywords: Gestational diabetes mellitus, Uric acid, Body mass index.

INTRODUCTION

Gestational diabetes mellitus (GDM) is defined as glucose intolerance that was not present or recognized prior to pregnancy and it is diagnosed when the pancreatic function in women is not sufficient to control the diabetogenic environment that pregnancy confers *(Gilmartin et al., 2012)*. The diagnosis of GDM also identifies pregnancies at increased risk of perinatal morbidity *(Reece, 2014)*.

Several risk factors are associated with the development of GDM. The most

common is obesity (body mass index over 30) diagnosed before pregnancy *(Torloni et al., 2013)*. Being a member of an ethnic group with a higher rate of type II diabetes (as mentioned above), polycystic ovarian syndrome *(Toulis et al., 2013)*, essential hypertension, or pregnancy-related hypertension, strong family history of diabetes in first-degree relatives and a history of GDM in a previous pregnancy are other important risk factors *(Baptiste-Roberts et al., 2013)*. Nevertheless, no risk factors are known in around 50% of patients with GDM.

There is enough evidence to assert that T2D has a strong genetic component. The concordance of T2D in monozygotic twins is approximately 70%, compared with 20%–30% in dizygotic twins *(Lehtovirta et al., 2010)*.

Uric acid is the main product of purine metabolism and is formed from xanthine by the action of xanthine oxidase. Normal serum uric acid levels are generally 3-7 mg/100 ml for men, and 2-6 mg/100 ml for women, frequently expressed as mg %. The reason is that estrogen promotes excretion of uric acid during the reproductive period. The limit of uric acid solubility in extracellular fluids is 7.0 mg/dl, and patients with higher serum concentrations are considered hyperuricemic *(Anton et al., 2010)*.

Uric acid is primarily excreted via the kidney, where it is completely filtered at the glomerulus, fully reabsorbed into the proximal tubule, and then secreted (about 50% of the filtered load) and again reabsorbed. Elevated serum uric acid concentrations can result from an overproduction of uric acid but are usually the consequence of its low excretion.

High serum uric acid levels are associated with alcohol intake, a purine-rich diet, compromised renal function, and obesity. Insulin increases both sodium and uric acid reabsorption *(Alderman, 2010)*. Therefore, increased serum uric acid levels may be an expression of an insulin-resistant state and metabolic syndrome. This proposition is supported by evidence that higher serum uric acid levels correlate with a lower insulin-stimulated glucose uptake and a higher plasma insulin response to oral glucose loading *(Pacifico et al., 2013)*.

During pregnancy, maternal serum uric acid levels initially fall, with a subsequent rise to prepregnancy levels near term *(Lind et al., 2010)*. The third-trimester rise in uric acid levels may be related to an increase in fetal uric acid production or a decrease in uric acid clearance *(Dunlop and Davison, 2010)*.

In pregnancy, uric acid is correlated with insulin resistance in women with gestational hypertension. Serum uric acid is higher at 24-28 weeks gestation in women diagnosed with gestational diabetes compared to women without diabetes. Uric acid is also higher in nonpregnant women with a history of gestational diabetes, independent of body mass index *(Laughon et al., 2011)*.

The aim of the present study was to test the hypothesis that increased uric acid levels, and body mass index (BMI) measured in the first trimester of pregnancy, are associated with the subsequent development of GDM.

PATIENTS AND METHODS

This was a prospective observational cohort study which included 520 pregnant women in their first trimester who regularly attended the outpatient clinic for routine antenatal care. This study was conducted at Alexandria Police Authority Hospital from November 2018 to May 2020.

Inclusion criteria: Pregnant women with gestational age < 13 weeks.

Exclusion criteria: Diabetes mellitus before pregnancy, multiple pregnancies, renal diseases, liver diseases, cardiovascular diseases, thyroid diseases, gout, smoking, and drugs known to increase uric acid level in the blood such as aspirin, caffeine, diuretics and phenothiazines.

All subjects that included in this study were fully counseled for their approval to be included in this study and they all agreed to undergo this study.

All patients subjected to:

1. History:

Personal history, menstrual history, obstetric history, past history, medical history: especially of hormonal treatment and drugs known to increase uric acid level in the blood such as phenothiazines, and family history: of diabetes mellitus, hypertension….etc.

2. Clinical examination:

General examination: pulse, temperature, blood pressure, body weight, height, body mass index and chest and heart examination.

Abdominal examination: Inspection, palpation, and auscultation.

3. Ultrasonography:

a. Date the pregnancy (gestational age). The most accurate measurement for dating is the crown-rump length of the fetus, which can be done between 7 and 13 weeks of gestation. After 13 weeks gestation, the gestational age may be estimated by the bi-parietal diameter, head circumference, or femur length.

b. Confirm fetal viability.

c. Determine the site of the fetus (intrauterine or extrauterine).

d. Check the site of the placenta. e) Check the number of fetuses. f) Check for any abnormalities. g) Assess fetal growth.

e Check for fetal movement.

4. Investigations:

Maternal serum uric acid: Was measured before 13 weeks gestation.

Collection of blood samples:

Venous blood sample of 3ml was collected from each subject after placement in a dry, clean, non-contaminated glass tube and left for half an hour to allow clotting of the blood and separation of the serum to measure uric acid level.

The specimens were centrifuged immediately thereafter for 5 minutes at 4.000 rpm, and the supernatant serum was transferred into another dry, clean, non-contaminated glass tube, immediately frozen and stored at -30C until assayed.

Serum uric acid was measured in Alexandria Police Authority Hospital main clinical laboratories chemistry department by enzymatic photometric test using a diagnostic kits (Dia Sys "Diagnostic Systems International" from

Lab Top) using TBHBA (2,4.6-tribromo-3-hydroxy benzoic acid) by uricase method (in uric acid) .

Screening for GDM:

All patients underwent routine GDM screening with 50gm oral glucose-loading test (GLT) between 24-28 weeks gestation. When plasma glucose level after 1hr was >140mg/dl, the patient was considered to be at increased risk for developing GDM and underwent 3hrs oral glucose tolerance test (OGTT).

Measurements of OGTT:

Preparation:

The patient is instructed not to restrict carbohydrate intake in the days or weeks before the test. The test should not be done during an illness, as it may not reflect the patient's glucose metabolism when healthy. A full adult dose should not be given to a person weighing less than 43 kg, or exaggerated glucoses may produce a false positive result. Usually the OGTT is performed in the morning as glucose tolerance can exhibit a diurnal rhythm with a significant decrease in the afternoon. The patient is instructed to fast (except for water) for 8–12 hours prior to the tests.

Ethical consent:

An approval of the study was obtained from Al- Azhar University academic and ethical committee. Every patient signed an informed written consent for acceptance of the operation.

Statistical analysis:

Analysis of data was done by SPSS® for Windows® version "20" as follows: Description of quantitative variables as mean, SD and range. Description of qualitative variables as number and percentage. Unpaired t-test was used to compare two groups as regard quantitative variable in parametric data (SD<50% mean). Mann Whitney Willcoxon test was used to compare two groups as regard non parametric data (SD>50% Mean). Sensitivity = true +ve /true +ve + false –ve = ability of the test to detect +ve cases. Specificity = true -ve/true-ve+ false +ve = ability of the test to exclude negative cases. PPV (positive predictive value) = true+/true+ve +false. +ve = % of true +ve cases to all positive. NPV = true-ve/true-ve + false –ve = % of the true –ve to all negative cases. Accuracy = true+ve/true+ve + true –ve. P value < 0.05 was considered significant.

RESULTS

Included women were recruited at a mean gestational age of 11.01 ± 0.87 weeks (range: 10 – 13 weeks). Serum samples were taken from all included women at recruitment for checking serum uric acid. The mean serum uric acid was 3.4 mg/dl. All included women were screened for gestational diabetes mellitus (GDM) at a mean gestational age of 25.88 ± 1.09 weeks (range: 24 – 28 weeks). Of the recruited 520 women, 25 (4.8%) had a positive 1 hr glucose test (blood glucose ≥ 140 mg/dl after oral 50 g glucose); of them 23 (4.4%) were diagnosed to have GDM **(Table 1** and **Figure 1)**.

Table (1): Characteristics of and gestational age of included women at Recruitment and at Screening for GDM serum uric acid at recruitment

Age (Years)	
Range:	16 – 43
Mean ± SD:	27.55 ± 5.24
BMI (Kg/m^2)	
Range:	18 – 38.5
Mean ± SD:	26.17 ± 4.21
Parity	
Range:	0 – 8
Median (IQR):	1 (1 – 2)
No. of Previous Abortions	
Range:	0 – 8
Median (IQR):	0 (0 – 1)
Gestational Age at Recruitment(Weeks)	
Range:	10 – 13
Mean ± SD:	11.01 ± 0.87
Serum Uric Acid at Recruitment (mg/dl)	
Range:	0.4 – 8.2
Mean ± SD :	3.4 ± 0.5
Gestational Age at Screening for GDM (Weeks)	
Range:	24 – 28
Mean ± SD:	25.88 ± 1.09

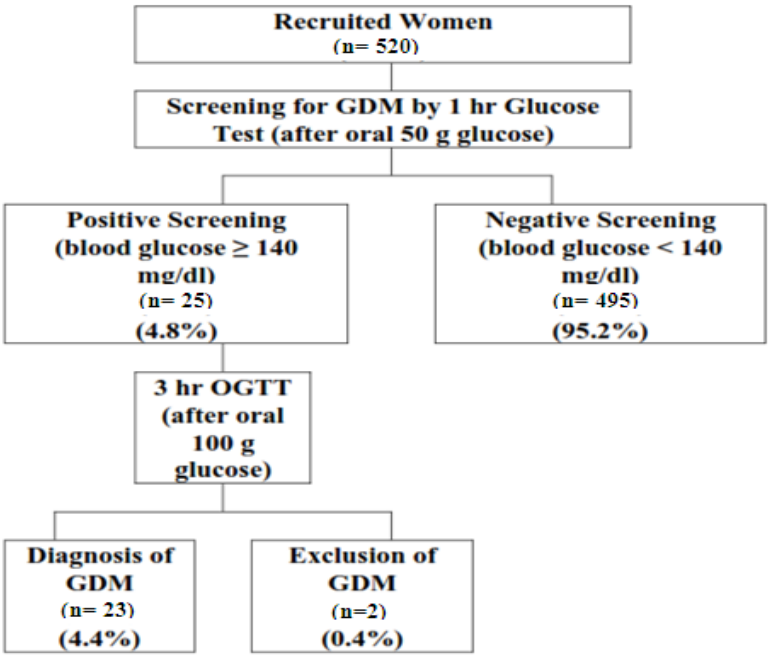


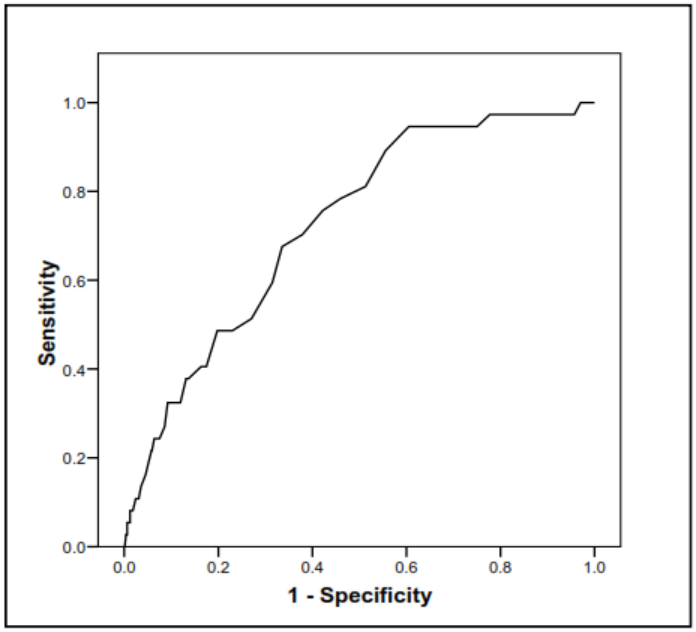
Figure (1): Study Course

There were no significant differences between women who developed GDM and women who did not develop GDM regarding maternal age, parity and no. of previous abortions. The mean BMI was, however, significantly higher in women who developed GDM when compared to women who did not develop GDM [29.02 ± 4.39 Kg/m2 vs. 26.04 ± 4.16 Kg/m2, respectively, $p<0.001$]. There were no significant differences between women who developed GDM and women who did not regarding gestational age at recruitment and at screening. The mean serum uric acid at recruitment was significantly higher in women who developed GDM when compared to women who did not develop GDM **(Table 2)**.

Table (2): Difference between women who developed GDM and women who did not regarding their characteristics gestational age at recruitment and screening and serum uric acid at recruitment

	Women who developed GDM (n=23)	**Women who did not develop GDM (n=497)**	***P***
Age (Years) Range: Mean ± SD:	 21 – 39 27.41 ± 4.69	 16 – 43 27.55 ± 5.26	>0.05
BMI (Kg/m^2) Range: Mean ± SD:	 22 – 36 29.02 ± 4.39	 18 – 38.5 26.04 ± 4.16	<0.001
Parity Range: Median (IQR):	 0 – 4 2 (1 – 2)	 0 – 8 1 (1 – 2)	>0.05
No. of previous abortions Range: Median (IQR):	 0 – 4 0 (0 – 2)	 0 – 8 0 (0 – 1)	>0.05
Gestational age at recruitment (Weeks) Range: Mean ± SD:	 10 – 13 10.86 ± 0.79	 10 – 13 11.02 ± 0.87	>0.05
Gestational age at screening for GDM (Weeks) Range: Mean ± SD:	 24 – 28 25.89 ± 1.24	 24 – 28 25.88 ± 1.08	>0.05
Serum Uric Acid at Recruitment (mg/dl) Range: Mean ± SD	 1.2 – 7.3 3.8 ± 0.8	 0.4 – 8.2 2.9 ± 0.4	<0.001

Receiver operator characteristics (ROC) curve was constructed for serum uric acid as predictor of GDM and showed a significant good predictability [AUC =0.725, 95% CI (0.638 to 0.801), p<0.001] **(Figure 2)**.



AUC = 0.725, 95% CI (0.638 to 0.801), p<0.001

Figure (2): ROC Curve for Serum Uric Acid as Predictor of GDM

The best cutoff point for serum uric acid as predictor of GDM was ≥ 3.05 mg/dl [sensitivity 75.7%, specificity 57.8%, PPV 7.5%, NPV 89.1%, positive likelihood ratio 1.8 and negative likelihood ratio 0.42]. This obviously low PPV is explained by the low prevalence of women who developed GDM among included women [23/520 (4.4%)]. The relatively high sensitivity and NPV (75.7% and 89.1%, respectively) makes serum uric acid useful as a 'screening' test for GDM **(Table 3)**.

Table (3): Accuracy of serum uric acid in prediction of GDM

Serum uric acid as predictor of GDM	Sensitivity	specificity	PPV	NPV	LR+	LR-
≥ 3.05 mg/dl	75.7%	57.8%	7.5%	89.1%	1.8	0.42

PPV positive predictive value , NPV negative predictive value
LR+ positive likelihood ratio, LR- negative likelihood ratio

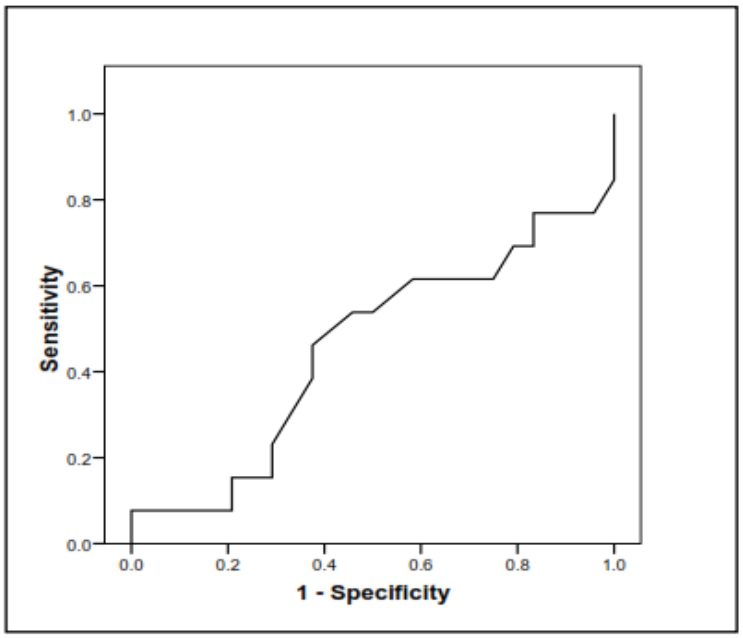
There were no significant correlation between serum uric acid and each of age, parity or fasting blood sugar. There was, however, a significant positive correlation between serum uric acid and BMI (r=0.378, p<0.001) **(Table 4)**.

Table (4): Correlation between Serum Uric Acid and Other Measured Variables

		Age	BMI	Parity	Fasting Blood Sugar
Serum Uric Acid	***r****	0.18	0.378	0.007	0.091
	P	> 0.05	< 0.001	> 0.05	> 0.05

* Pearson's Correlation Coefficient
NS non-significant – HS highly significant

ROC curve was constructed for serum uric acid as predictor of fasting blood glucose ≥ 105 mg/dl among women who developed GDM and showed an insignificant poor predictability [AUC =0.455, 95% CI (0.249 to 0.661), p>0.05] **(Figure 3)**.



AUC = 0.455, 95% CI (0.249 to 0.661), p>0.05

Figure (3): ROC Curve for Serum Uric Acid as Predictor of Fasting Blood Glucose ≥105 among women with GDM

Of the included 520 women, 227 (43.6%) had a serum uric acid above this cutoff value (≥ 3.05 mg/dl); of them 17 (7.5%) developed GDM, in contrast to 6 (1.9%) of the 293 women who had a serum uric acid < 3.05 mg/dl. Binary logistic regression analysis of measured variables showed that a level of serum uric acid at recruitment ≥ 3.05 mg/dl was independently associated with a significantly 4-fold higher risk of developing GDM [OR 3.8, 95% CI (1.99 to 9.15)].

Binary logistic regression analysis of measured variables showed that serum uric acid at recruitment was

insignificantly associated with a fasting blood glucose ≥ 105 mg/dl among women who developed GDM [OR 0.833, 95% CI (0.493 to 1.47)] **(Table 5)**.

Table (5): Binary logistic regression analysis of serum uric acid as predictor of GDM and Fasting Blood Glucose ≥ 105 mg/dl

		OR	95% CI	P
Serum uric acid as predictor of GDM				
≥ 3.05 mg/dl (n=227) < 3.05 mg/dl (n=293)	17/210 6/287	3.8	1.99 to 9.15	<0.001
Serum Uric Acid as Predictor of FBG ≥ 105 mg/dl (among women who developed GDM)				
≥ 3.05 mg/dl (n=17) < 3.05 mg/dl (n=6)	5/12 2/4	0.833	0.293 to 1.47	>0.05

OR odds ratio, 95% CI 95% Confidence Interval

DISCUSSION

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 4-fold higher if first-trimester uric acid was ≥ 3.05 mg/dl [OR 3.8, 95% CI (1.99 to 9.15)].

These results were similar to that obtained by some studies *(Sautin et al., 2012, Ryo et al., 2011* and *Costa et al., 2010)*. They had found that serum uric acid was significantly correlated with insulin resistance.

This coincides with the result of *Laughon et al. (2011)* 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 Carpenter and Coustan criteria or by a 1-hour value of ≥200 mg/dl. Almost half (46.6%) of the women with GDM had first-trimester uric acid concentrations in the highest quartile (≥3.57-8.30 mg/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 also consistent with the association of uric acid with insulin resistance in the no pregnant population *(Yoo et al., 2010)*. In a large cross-sectional study of 53,477 no pregnant 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 *(Dehghan et al., 2012)*.

In this study, we found that uric acid ≥ 3.05 mg/dl was independently associated with a significantly 4-fold higher risk of developing GDM. These results were similar to those reported by others; however, there are no prior reports of the association between first-trimester uric acid and GDM among women at low risk *(Lind et al., 2010)*.

In contrast, *Güngör et al. (2011)* 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 24^{th} and 28^{th} 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 *(Gunger et al., 2011)*. 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 no statistically significant relationship between age and development of GDM.

Lao et al. (2011) have reviewed the prevalence of GDM, diagnosed by the World Health Organization criteria in over 15000 singleton pregnancies managed. 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 *(Lao et al., 2011)*. The results of this study were against ours, this may be due to the difference in the number of cases.

In this study, the mean BMI was significantly higher in women who developed GDM when compared to women who did not develop GDM [29.02 ± 4.39 Kg/m2 vs. 26.04 ± 4.16 Kg/m2, respectively, p <0.001].

Jenny et al. (2012) 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 pre-pregnancy body mass index (BMI) is a strong predictor for development of GDM. This study coincides the same results as our study.

Ogonowski et al. (2015) studied 1121 patients with GDM. The control group consisted of 1011 healthy pregnant women. All had singleton pregnancies. Significant relationships between pre-pregnancy 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 ($r=0.378$, $p<0.001$).

This agrees with the result of *Laughon et al. (2011)* who found that maternal pre-pregnancy 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 *(Nagahama et al., 2011* and *Yoo et al., 2005)*. Hyperuricemia has been associated with increasing body mass index (BMI) in several studies and are even apparent in the adolescent youth *(Conen et al., 2004)*. These studies state the same results as our study.

Hyperuricemia may also be present in the metabolic 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 *(Yoo et al., 2010)*. 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 recruitment was insignificantly associated with a fasting blood glucose ≥ 105 mg/dl among women who developed GDM [OR 0.833, 95% CI (0.493 to 1.47)].

In a large cross-sectional study of 53,477 nonpregnant adults, serum uric acid was positively correlated with fasting serum glucose ≥110 mg/dl *(Yoo et al., 2010)*. The results of this study were against ours.

CONCLUSION

The hallmark of GDM is increased insulin resistance. Patients with GDM having an insulin resistance cannot compensate with increased insulin production in the β-cells of the pancreas.

Uric acid is the end product of purine catabolism catalyzed by the enzyme xanthine oxidase/ dehydrogenase.

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.

In our study, it was found:

- Elevated first-trimester uric acid concentration was correlated with an increased risk of developing GDM. The risk of developing GDM was 4-fold higher if first-trimester uric acid was ≥ 3.05 mg/dl [OR 3.8, 95% CI (1.99 to 9.15)].
- There was a significant positive correlation between serum uric acid and BMI ($r = 0.378$, $p < 0.001$).
- The mean BMI was significantly higher in women who developed GDM when compared to women who did not develop GDM [29.02 ± 4.39 Kg/m2 vs. 26.04 ± 4.16 Kg/m2, respectively, $p<0.001$].

Recommendations

- Serum uric acid measurement in 1st trimester of pregnancy as a predictor for GDM and screening of GDM at 24-28 weeks gestation.
- As regard BMI should be less than 25kg/m for exclusion of obesity as a risk factor for occurrence of GDM.

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حمض البوليك فى الثلث الأول من الحمل وحساب كتلة الجسم كمؤشر لظهور مرض البول السكرى فى الحوامل

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خلفية البحث: يعرف مرض البول السكرى فى الحمل بأنه زيادة نسبة الجلوكوز فى دم السيدة الحامل والذى لم يكن موجودا قبل حدوث الحمل وهو يشخص عندما تكون وظائف البنكرياس فى السيدات الحوامل غير كافية لتتحكم فى البيئة المسببة للسكر والتى يسببها الحمل. إن تشخيص مرض البول السكرى فى الحمل مهم جدا إذ يبين لنا أيضا أنواع الحمل ذات الخطورة العالية من حيث مشاكل ما حول الولادة. يُعتقد أن هرمونات الحمل وعوامل أخرى تتداخل مع عمل الأنسولين لأنها ترتبط بمستقبلات الأنسولين. حمض اليوريك هو المنتج النهائي لتقويض البيورين المحفز بواسطة إنزيم أوكسيديز زانثين/ ديهيدروجينيز.

الهدف من البحث: دراسة العلاقة بين إرتفاع معدل حمض اليوريك فى القسم الأول من الحمل وكتلة الجسم والإصابة بمرض البول السكرى فى الحمل فيما بعد.

المريضات وطرق البحث: كانت هذه دراسة جماعية قائمة على الملاحظة شملت 520 امرأة حامل في الأشهر الثلاثة الأولى من الحمل اللائي يذهبن بانتظام إلى العيادة الخارجية للرعاية الروتينية السابقة للولادة. أجريت هذه الدراسة بمستشفى هيئة الشرطة بالإسكندرية خلال الفترة من نوفمبر 2018 إلى مايو 2020.

نتـــائج البحـــث: توجـــد علاقـــة ذات دلالـــة إحصـــائية بـــين مســـتوى حمـــض اليوريـــك فـــي القســـم الأول مـــن الحمـــل وظهـــور مـــرض البـــول الســـكري فـــي الحمـــل. توجـــد علاقـــة ذات دلالـــة إحصـــائية بـــين معـــدل كتلـــة الجســـم ومـــرض البـــول الســـكري فـــي الحمـــل. توجـــد علاقـــة ذات دلالـــة إحصـــائية بـــين عمـــر الســـيدة الحامـــل وظهـــور مـــرض البـــول الســـكري فـــي الحمـــل. توجـــد علاقـــة ذات دلالـــة إحصـــائية بـــين عـــدد مـــرات الحمـــل وعـــدد الأطفـــال للســـيدة الحامـــل وظهور مرض البول السكرى فى الحمل.

الاســـتنتاج: فـــي فتـــرة الحمـــل، يـــرتبط حمـــض اليوريـــك بمقاومـــة الأنســـولين لـــدى النســـاء المصـــابات بارتفـــاع ضـــغط الـــدم الحملـــي ويكـــون أعلـــى فـــي 24-28 أســـبوعًا مـــن الحمـــل عنـــد النســـاء المصـــابات بســـكري الحمـــل مقارنـــة بالنســـاء غيـــر المصـــابات بـــداء الســـكري. قيـــاس نســـبة حمـــض البوليـــك فـــى الثلـــث الأول مـــن الحمـــل كمؤشـــر لحـــدوث مـــرض البـــول الســـكرى والكشـــف عنـــه مـــا بـــين 24-28 أســـبوع مـــن الحمـــل. يجـــب أن تكـــون كتلـــة الجســـم أقـــل من 25 لاستبعاد السمنة كمسبب لحدوث مرض البول السكرى.

الكلمـــات الدالـــة: البـــول الســـكرى فـــي الحوامـــل، حمـــض اليوريـــك، مؤشـــر كتلة الجسم.