ABSTRACT

Objectives: Estimation of serum resistin and TNF-α at time of pregnancy diagnosis and 6<sup>th</sup> gestational week (GW) to investigate its relation to development of gestational insulin resistance (GIR) and diabetes mellitus (GDM).

Patients & Methods: All enrolled pregnant women underwent 75-Oral glucose tolerance test (OGTT) and ELISA estimation of serum resistin and TNF-α at baseline time and at the 6<sup>th</sup> GW. GIR was diagnosed using the homeostasis model assessment of IR (HOMA-IR) at score of >2 and GDM was diagnosed according to the results of the 75-OGTT if fasting blood glucose (FBG) was ≥92 mg/dl, 1-h post-prandial BG (PPBG) ≥180 mg/dl and 2-h BG ≥153 mg/dl. Study outcomes included the frequency of GIR and GDM, changes in serum levels of TNF-α and resistin throughout pregnancy and its relation to frequency and severity of GIR and GDM.

Results: At the 30<sup>th</sup> GW, 112 women (34.9%) were IR and 39 women (12.1%) progressed to GDM. Serum resistin and TNF-α levels were significantly increased at 6<sup>th</sup> GW than baseline measures in all pregnant women with significant variance among study groups. Statistical analyses defined high baseline serum resistin as significant predictor for development of GIR and the hazard for being IR rises sharply with serum resistin above 5.5 ng/ml and high 6-GW serum TNF-α levels at cutoff points of serum TNF-α 10.79 ng/ml and 15.9 ng/ml as significant early predictors for development of GIR and GDM, respectively.

Conclusion: Estimated serum resistin and TNF-α levels were positively associated with the risk for development of GIR and/or GDM later in pregnancy. Baseline serum resistin and 6-GW serum TNF-α levels could differentiate women susceptible to develop GIR and GDM, so could be used as early predictors.

INTRODUCTION

Insulin resistance (IR) is typically defined as decreased sensitivity to metabolic actions of insulin, such as insulin-mediated glucose disposal and inhibition of hepatic glucose production (Gutch et al., 2015). Pregnancy involves progressive increase in IR, so the β-cells must
adapt to maintain normal glucose metabolism and blood levels during pregnancy (Retnakaran et al., 2016), and prevent development of gestational diabetes mellitus (Vejrazkova et al., 2014). Gestational diabetes mellitus (GDM) can cause short- and long-term complications to the mother and fetus, imparts a high risk of developing postpartum diabetes and is considered to be an early stage of type-2 DM (Reece, 2010; Ornoy et al., 2015).

Various hormones, cytokines and growth factors have been implicated or associated with GDM risk in humans, including adipokines, prolactin, (Thagaard et al., 2017) vascular endothelial growth factor, (Troncoso et al., 2017), tumor necrosis factor-α (TNF-α) and receptor activator of nuclear factor-kappa B ligand (Panthan et al., 2015). Adipokines are substances of hormonal character that are secreted by adipose tissue (Mierzwicka & Bolanowski, 2016) and play a major role in carbohydrate metabolism, so its disturbed secretion may induce metabolic disorders especially affecting glucose homeostasis (Vejrazkova et al., 2017).

Human resistin is a 12.5 kDa peptide characterized by the presence of 10 to 11 cysteine residues (Patel et al., 2004) and is expressed in peripheral blood mononuclear cells (PBMC), macrophages and bone marrow cells (Steppan & Lazar, 2004). Human resistin plays a major regulatory role in the inflammatory response through macrophages, PBMC which are the primary targets of resistin (Filková et al., 2009). Also, resistin up-regulates the expression of proinflammatory cytokines such as TNF-α, IL-6 and IL-12, (Araki et al., 2006).

Human resistin is present in placental tissue, mainly in trophoblastic cells and its gene expression was more prominent in term than in 1st trimester chorionic tissue (Yura et al., 2003), so resistin is considered as an isolated placental hormone which in humans, may modulate insulin sensitivity during pregnancy (Lappas et al., 2005). Moreover, resistin is involved in IR via affecting glucose metabolism, inhibiting fatty acid uptake and metabolism by affecting acetyl-CoA carboxylase, and AMP-activated protein kinase (Ikeda et al., 2013).

The current study was undertaken to study the relation between development of gestational insulin resistance (GIR) and GDM on one hand and the complementary action between disturbed levels of adipocytokines and inflammatory markers on the other hand.

**Design**

Prospective comparative clinical trial

**Setting**

Tertiary referral hospital, KSA

**PATIENTS & METHODS**

After approval of the study protocol by the Local Ethical Committee, all pregnant women who attended the Antenatal Outpatient Clinics (OPC) for assurance of being pregnant and signed written fully informed consent to participate in the study were evaluated for eligibility for study inclusion and time of pregnancy diagnosis was considered as baseline time. All women underwent evaluation for demographic data including age, weight and height, and for baseline clinical and obstetric data. Exclusion criteria include manifest DM, previous GDM in multipara women, morbid obesity with...
body mass index (BMI) >35 kg/m² (WHO, 1995), patients with cardiac, hepatic, renal diseases or endocrinopathy especially obesity-inducing diseases and multiple gestational sacs on follow-up US examination.

All enrolled women were asked to attend the OPC, fasting for at least 8-hr, next morning to diagnosis of pregnancy to give blood sample for baseline laboratory investigations and 75-Oral glucose tolerance test (OGTT), which entails estimation of fasting blood glucose (FBG) and 1-hr and 2-hr postprandial blood glucose (PPBG) levels after taking a 75-gm oral glucose diet.

Investigations

Sampling: Venous blood samples (5 ml) were collected from the antecubital vein under complete aseptic conditions and were divided into two parts:

The first part was put in a tube containing sodium fluoride (2 mg sodium fluoride/ ml blood) to prevent glycolysis for estimation of blood glucose levels using glucose oxidase method (Tinder, 1969).

The second part was collected in plain tube, allowed to clot, centrifuged at 1500×g for 15 min and the serum samples were divided into two parts one for estimation of serum insulin and the other part was collected in clean dry Eppendorf tube to be stored at −70°C until being assayed for serum resistin and TNF-α.

Laboratory investigations

Serum levels of studied parameters were measured using enzyme linked immunosorbent assay (ELISA) kits according to the manufacturer’s instructions and were read using a 96 well microplate ELISA reader (Dynatech MR 7000).

Human insulin was measured with ELISA kit provided by abcam Inc., San Francisco, USA (Gordon et al., 1985). Fasting serum insulin (FSI) was measured at baseline, 6th, 18th and 30th GW for calculation of the homeostasis model assessment of IR (HOMA-IR) score.

Human resistin was measured with ELISA kit provided by R&D systems Inc., Minneapolis, USA, (Haluzik & Haluzikova, 2006). Serum resistin was measured at baseline and 6th GW.

Human TNF-α was measured with ELISA kit provided by abcam Inc., San Francisco, USA (Coughlan et al., 2001). Serum TNF-α was measured at baseline and 6th GW.

Evaluated clinical variables:

Baseline BMI (kg/m²) was calculated as weight (kg)/ height (m²) (Bray, 1992) and patients were classified according to BMI using the World Health Organization (WHO, 2004) ranges as underweight <18.5 kg/m², normal weight 18.5-24.9 kg/m², overweight 25-29.9 kg/m² and obese ≥30 kg/m².

Baseline IR was evaluated using HOMA-IR score which was calculated according to the formula proposed by Matthews et al., (1985) and HOMA-IR score of >2 is considered abnormal.

GDM was diagnosed according to the results of the 75-OGTT which were interpreted according to the recommendations of the International association of diabetes and pregnancy study groups and woman was considered to have GDM if FBG ≥92 mg/dl, 1-h BG ≥180 mg/dl and 2-h BG ≥153 mg/dl (IADPSG, 2010).
Follow-up and grouping

All women were asked to re-attend the OPC at start of the 6th, 18th, and 30th GW to give blood samples for follow-up investigations and evaluation. All women developed GIR only without progress to GDM were collected in GIR group and women who developed GIR and progressed to GDM were collected in GDM group. For comparative purposes, 20 women were randomly chosen from those who completed their pregnancy course with HOMA-IR score <2 and gave normal OGTT on all sessions of estimations were collected as Control group.

Study outcomes:

Study outcomes included determination of the frequency of women developed GIR and GDM and its relations to estimated levels of TNF-α and resistin.

Statistical analysis

Obtained data were presented as mean±SD. Results were analyzed using paired t-test and One-way ANOVA Test. Sensitivity & specificity of serum parameters as predictors for development of GIR and GDM were evaluated using the receiver operating characteristic (ROC) curve analysis judged by the area under the curve (AUC). Kaplan-Meier regression analysis was used to determine a cutoff point for serum parameters to predict GIR and GDM development. Statistical analysis was conducted using the IBM SPSS (Version 23, 2015) for Windows statistical package. P value <0.05 was considered statistically significant.

RESULTS

The study included 359 women eligible for evaluation, 38 women were excluded and 321 women were included in the study. At the 18th GW, 19 women (5.9%) were IR with mean HOMA-IR score of 2.24±0.56; range: 2-2.93 and 12 developed GDM (3.7%) with mean FBG of 123.5±12.1; range: 110-145 mg/dl. At the 30th GW, 112 women (34.9%) were IR; with mean HOMA-IR score of 2.39±0.37; range: 2.14-3.66 and 39 women progressed to GDM for a frequency of 12.1% with mean FBG of 119.8±22.3; range: 109-166 mg/dl. The remaining 209 women (65.1%) achieved the 30th wk free of both IR and GDM and 20 of them were randomly chosen as Control group. Baseline data of women included in the three groups showed non-significant (p>0.05) difference (Table 1).

Baseline FBG, FSI and HOMA-IR score showed non-significant (p>0.05) differences between patients of studied groups. At the 6th GW, despite of non-significant (p>0.05) difference between patients of studied groups concerning FBG and FSI, calculated HOMA-IR score showed significant (p=0.018) variance between studied groups. OGTT 1-hr and 2-hr PPBG levels showed significant variance (p=0.008 & 0.009, respectively) between studied groups. Estimated BG levels on OGTT and FSI and HOMA-IR score at 18-GW and 30-GW showed significant variance between women of the three groups and in women of GDM group compared to corresponding measures of control and IR women. Women of GIR group showed significantly higher 2-hr PPBG, FSI and HOMA-IR score at both 18-GW and 30-
GW compared to corresponding measures of control women (Table 2).

Mean baseline serum resistin and TNF-α levels showed non-significant (p>0.05) difference between studied groups. At 6-GW, mean serum resistin and TNF-α were significantly higher in all women compared to their respective baseline levels with significant (p=0.0001) variance between women of all groups. Moreover, 6-GW serum levels of resistin and TNF-α were significantly higher in GDM women than GIR women (Table 3).

The 6-GW serum TNF-α levels showed positive significant correlation with the possibility for development of GIR (Rho=0.326, p=0.0001) and GDM (Rho=0.291, p=0.001), while the possibility for development of GDM was positively correlated with baseline serum resistin (Rho=0.173, p=0.047). Severity of GIR as judged by HOMA-IR score showed positive (Rho=0.306, p=0.0001) association with 6-GW serum TNF-α levels, while FBG levels estimated at 30-GW showed positive association with 6-GW serum resistin (Rho=0.192, p=0.028) and TNF-α (Rho=0.303, p=0.0001).

ROC curve analysis defined high 6-GW TNF-α and high baseline serum resistin levels as the significant (p=0.0005 & 0.008, respectively) predictors for development of GIR with AUC of 0.762 (95th CI: 0.663-0.861) and 0.638 (95th CI: 0.514-0.761), respectively. However, high 6-GW serum TNF-α level was defined as the significant (p=0.001) predictor for development of GDM with AUC of 0.684 (95th CI: 0.568-0.800).

Kaplan-Meier regression analysis defined baseline serum resistin at cutoff point of 5.5 (95% CI: 5.28-5.72) and 6-GW serum TNF-α levels at cutoff points of 10.79 (95th CI: 9.69-11.89) for early prediction of upcoming GIR and 6-GW serum TNF-α level at cutoff points of 15.9 (95% CI: 13.19-18.661) for early prediction of later development of GDM.

### Table (1): Demographic and clinical data determined at the 6th week GA

<table>
<thead>
<tr>
<th>Data</th>
<th>Control (n=20)</th>
<th>GIR (n=73)</th>
<th>GDM (n=39)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>27.1±3.7</td>
<td>28.3±2.4</td>
<td>28.7±3.1</td>
<td>0.112</td>
</tr>
<tr>
<td><strong>BMI data</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.3±8.8</td>
<td>85.4±6.8</td>
<td>87.5±7.5</td>
<td>0.059</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170.9±4.2</td>
<td>169.4±4</td>
<td>170.3±3.2</td>
<td>0.212</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.6±3.3</td>
<td>29.7±2.6</td>
<td>30.2±1.6</td>
<td>0.067</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.2±0.5</td>
<td>2±0.7</td>
<td>1.9±0.9</td>
<td>0.484</td>
</tr>
<tr>
<td>Parity</td>
<td>1.15±0.7</td>
<td>1±0.9</td>
<td>1.1±0.8</td>
<td>0.732</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>114.7±3.5</td>
<td>115.1±7.3</td>
<td>113.2±5.5</td>
<td>0.376</td>
</tr>
<tr>
<td>Diastolic</td>
<td>71.8±7.2</td>
<td>72.1±4.3</td>
<td>74±3.7</td>
<td>0.298</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; BMI: Body mass index; *: indicates significant difference
Table (2): HOMA-IR and OGTT data of women of the studied group

<table>
<thead>
<tr>
<th>Time</th>
<th>Parameter</th>
<th>Control (n=20)</th>
<th>GIR (n=73)</th>
<th>GDM (n=39)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>FBG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
<td>85.1±12.5</td>
<td>85.5±11.5</td>
<td>88.2±15.5</td>
<td>0.538</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FSI</td>
<td>6.48±0.46</td>
<td>6.61±0.87</td>
<td>0.125</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HOMA-IR score</td>
<td>1.43±0.3</td>
<td>1.41±0.2</td>
<td>0.871</td>
</tr>
<tr>
<td></td>
<td>OGGT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-GW</td>
<td></td>
<td>0-hr</td>
<td>87.8±3.1</td>
<td>88.8±3.3</td>
<td>89.9±4.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-hr</td>
<td>155.6±8.7</td>
<td>162.9±10.5*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-hr</td>
<td>106.8±15.1</td>
<td>121.2±6.2*</td>
<td>123.4±4.4*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FSI</td>
<td>6.39±0.56</td>
<td>6.64±0.73</td>
<td>6.8±0.63*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HOMA-IR score</td>
<td>1.4±0.11</td>
<td>1.48±0.17</td>
<td>1.53±0.14*</td>
</tr>
<tr>
<td>18-GW</td>
<td>OGGT</td>
<td>0-hr</td>
<td>90.9±4.8</td>
<td>91.7±4.4</td>
<td>103.9±22.1*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-hr</td>
<td>168.9±5.7</td>
<td>169.4±5.7</td>
<td>176±10*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-hr</td>
<td>106.8±15.1</td>
<td>122.1±5.8*</td>
<td>146.4±11.6*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FSI</td>
<td>6.78±0.82</td>
<td>7.18±0.73*</td>
<td>7.88±0.64*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HOMA-IR score</td>
<td>1.54±0.18</td>
<td>1.64±0.16*</td>
<td>2.04±0.27*†</td>
</tr>
<tr>
<td>30-GW</td>
<td>OGGT</td>
<td>0-hr</td>
<td>91.3±7.9</td>
<td>91.8±4.1</td>
<td>119.8±11.9*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-hr</td>
<td>169.6±6.3</td>
<td>179.5±8.1</td>
<td>204.8±11.6*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-hr</td>
<td>91.8±5.3</td>
<td>125.8±7.7*</td>
<td>164.3±13.3*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FSI</td>
<td>6.78±0.82</td>
<td>9.15±0.88*</td>
<td>9.57±0.28*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HOMA-IR score</td>
<td>1.55±0.24</td>
<td>2.19±0.14*</td>
<td>2.74±0.41*†</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; P value indicates significance of variance between studied groups; *: indicates significance of difference versus control group; †: indicates significance of difference versus IR group.
DISCUSSION

The current study detected a frequency of GIR and GDM of 34.9% and 12.1%, respectively among studied population of pregnant women. These data indicated a fact that IR detected early in pregnancy could predict the oncoming GDM. In line with these findings, Alptekin et al., (2016) found calculated HOMA-IR was significantly associated with GDM and remained independently associated with it with an AUC of 0.809 and sensitivity and specificity rates of 90 and 61%, respectively at cutoff point of 2.08. The reported frequency of GDM go in hand with Ma et al., (2017), Karcaaltincaba et al., (2017) and Arbib et al., (2017) who reported frequencies of 12.2%, 11.1% and 9.9%, respectively for GDM. Moreover, Huhn et al., (2017) and Brown & Wyckoff, (2017) documented that using the IADPSG criteria, there was an absolute increase of GDM prevalence of 8.5% (Huhn et al., 2017) and a 1.03-3.78-fold rise versus baseline criteria (Brown & Wyckoff, 2017).

Estimated serum resistin levels showed progressive increase with progress of pregnancy, but the pattern of changes of estimated levels differed between studied groups and showed positive correlation with frequency and severity of GIR and GDM. Moreover, statistical analyses defined high baseline serum resistin as significant predictor for development of GIR and the hazard for being insulin resistant rises sharply with serum resistin above the cutoff point of 5.5 ng/ml. Similarly, Kelly et al., (2017) detected elevated serum resistin in normotensive diabetic versus non-diabetic women and concluded that 1st trimester adipocytokines profiles suggest IR, and its persistence in 2nd and 3rd trimesters. Moreover, Guelfi et al., (2017) reported that in non-diabetic women early estimated maternal adiponectin, resistin and leptin levels progressively changed with advancing pregnancy, where adiponectin level decreased, while leptin and resistin levels increased, till time of GDM diagnosis and concluded that adipokine profile is altered early in pregnancy in women who develop GDM. These data signified that early estimated serum resistin could be used as early predictor for upcoming GIR and GDM.

Table (3): Serum resistin and TNF-α levels estimated throughout duration of pregnancy in women of the studied group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Time</th>
<th>Control (n=20)</th>
<th>GIR (n=73)</th>
<th>GDM (n=39)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum resistin (ng/ml)</td>
<td>Baseline</td>
<td>4.11±1</td>
<td>4.49±0.95</td>
<td>4.57±0.92</td>
<td>0.195</td>
</tr>
<tr>
<td></td>
<td>6-GW</td>
<td>5.32±0.45*</td>
<td>5.91±0.92*</td>
<td>6.83±0.96*</td>
<td>0.0001</td>
</tr>
<tr>
<td>Serum TNF-α (ng/ml)</td>
<td>Baseline</td>
<td>5.63±2.23</td>
<td>5.78±2.55</td>
<td>5.65±2.71</td>
<td>0.938</td>
</tr>
<tr>
<td></td>
<td>6-GW</td>
<td>7.67±3.13*</td>
<td>10.9±3*</td>
<td>12±4.96*</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD; TNF-α: Tumor necrosis factor-α P value indicates significance of variance between studied groups; *: indicates significance of difference versus baseline measures of studied groups.
The results of the current study and findings of the reviewed recent works refute that documented in older literature that serum resistin levels showed no difference between GDM and normoglycemic pregnant women (Akdeniz et al., 2011; Karatas et al., 2014), were not associated with the degree of glucose tolerance in pregnancy (Skvarca et al., 2012) and showed no association with GDM (Lobo et al., 2013).

As regards TNF-α, estimated serum TNF-α levels were significantly higher at the 6th GW than baseline measures in all studied women with significant variance between women of studied groups. ROC curve analysis defined high 6-GW serum TNF-α levels at cutoff points of 10.79 ng/ml and 15.9 ng/ml as significant early predictors for development of GIR and GDM, respectively. These data can illustrate the relationship between altered glucose and lipid metabolism and progressively increasing serum levels of TNF-α. Also, Ozler et al., (2015) reported that TNF-α was found to be predictive for GDM development at 24-28 GW and higher TNF-α levels were independent predictor of the need for insulin treatment in GDM patients.

The obtained results go in hand with Noureldeen et al., (2014) who detected elevated levels of TNF-α among pregnancy complicated with GDM at 2nd and 3rd trimesters and concluded that increased IR accompanies GDM is associated with increased resistin and TNF-α which might suggest their involvement in the development of GDM. Also, Duvnjak et al., (2016) reported that serum TNF-α concentration is associated with IR and correlates with its severity and Zhang et al., (2017) reported that serum and placental inflammatory biomarkers were significantly higher in women with GDM than women with healthy pregnancies.

The obtained results point to a pathogenic relationship between increased serum levels of resistin and TNF-α on one side and possibility of development and severity of GIR and GDM on the other side. In trial to explore the pathogenic role of adipocytokines and inflammatory cytokines and development of pregnancy-induced disturbed carbohydrate metabolism, Duvnjak et al., (2016) suggested that TNF-α leads to both β cell damage and also causes IR. Zhang et al., (2017) reported that distribution frequency of TNF-α-857CT single nucleotide polymorphisms was higher in women with GDM than in those with healthy pregnancies. Ning et al., (2016) observed a significant positive correlation between adipocytokines and overweight, IR and TNF-α in pregnant women with GDM. Recently, Cao et al., (2017) found the relative and absolute expression of plasma microRNA-16-5p, -17-5p, -20a-5p from GDM women were significantly upregulated compared with non-GDM women and the expression of those microRNAs was positively correlated with the risk factors of GDM; BMI, IR and TNF-α.

CONCLUSION

Estimated serum resistin and TNF-α levels were progressively increased with pregnancy progress and were positively associated with the risk for development of GIR and/or GDM later in pregnancy. Baseline serum resistin and 6-GW serum TNF-α levels could differentiate women susceptible to develop these
pregnancy-induced complications, so could be used as early predictors. Wider scale studies are mandatory to assure the diagnostic efficacy of the proposed cut-off points of resistin and TNF-α for increased risk of GIR and/or GDM development.

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قياس مستوى الريهستين وعامل التخثر الورمي-ألفا عند الأسبوع السادس من الحمل في مصل الأمهات لتلبية المبكر لمقاومة الإنسولين الحملي

هاني عبد الحليم الكلاف، عادل الخولي

أهداف البحث: قياس معدلات الريهستين وعامل التخثر الورمي-ألفا عند تشخيص الحمل والأسبوع السادس من الحمل وعلاقتها بمقاومة الإنسولين والبول السكري الحملي.

طرق البحث: خضعت كل السيدات الحوامل لاختبار تحميل الجلوكوز، قياس معدلات الريهستين وعامل التخثر الورمي-ألفا عند تشخيص الحمل والأسبوع السادس من الحمل، ثم تشخيص مقاومة الإنسولين الحملي عن طريق نموذج استناد مقاومة الإنسولين، والبول السكري الحملي عند انتهاء الحمل.

النتائج: في الأسبوع 30 من الحمل وجد أن 12% من السيدات مصابات بمقاومة الإنسولين الحملي، منهم 63% من السيدات مصابات بالبول السكري الحملي. ارتفعت قياسات الريهستين وعامل التخثر الورمي-ألفا عند ارتفاع مستويات الأكيما في مجموعات. أوضحت النتائج الإحصائية أن ارتفاع معدل الريهستين عند تشخيص الحمل يمثل مؤشر ذو دلالة إحصائية للتوقع يحدث مقاومة الإنسولين الحملي مع ارتفاع متوسط للعمر عند مرضى الريهستين-ألفا عند مرضى الريهستين-ألفا عند ارتفاع الأكيما تتراوح بين 1.29 و1.5 نانوجرام/مل عند مقابلة الإنسولين الحملي والبول السكري الحملي.

الخلاصة: قياس معدلات الريهستين وعامل التخثر الورمي-ألفا يرتبط إيجابياً بنسبة حديد مقاومة الإنسولين الحملي والبول السكري الحملي لاحقاً أثناء الحمل وقياس معدلات الريهستين عند الالتحاق بالدراسة وعامل التخثر الورمي-ألفا عند بدء الأسبوع 6 من الحمل ممكن من تميز هؤلاء الأكثر عرضة للإصابة.

3. المجلة المصرية للعلوم الطبية 38 (2) ديسمبر 2017 587-575-568

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