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Multi-Biomarker Oxidative Stress Panel Improves Prediction of Perinatal and Postpartum Outcomes in Gestational Diabetes: A Prospective Study

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Annotation: Hyperglycemia and insulin resistance lead to elevated oxidative stress in 14% of pregnancies worldwide, which is known as gestational diabetes mellitus (GDM). Existing biomarker methods are still restricted. The goal is to find out how well a full panel of oxidative stress biomarkers predicts postpartum glucose tolerance and perinatal outcomes in patients with GDM. Strategies: prospective case-control study of 150 pregnant women (75 GDM, 75 controls) at 24-28 weeks gestation. GDM was identified based on IADPSG criteria. Biomarkers analyzed included ischemia-modified albumin (IMA), total oxidant status (TOS), total antioxidant status (TAS), malondialdehyde (MDA), glutathione (GSH), and catalase (CAT). Multivariate analysis adjusted for confounders. At six to twelve weeks, postpartum OGTT is conducted. The following oxidative markers were considerably higher GDM patients: MDA (4.8±1.2 vs 2.9±0.8 nmol/mL), TOS (28.4±6.2 vs 18.7±4.1 μmol H_2O_2 Eq/L), and IMA (0.89±0.12 vs 0.65±0.08 ABSU) (all $p<0.001$). GSH (285±45 vs 365±52 μmol/L), CAT (42.8±8.5 vs 58.2±9.7 U/mg protein), and TAS (1.24±0.18 vs 1.58±0.23 mmol Trolox Eq/L) all showed decreased antioxidant ability (all $p<0.001$).

infants with elevated MDA (OR 1.89) and TOS (OR 2.34) were predicted to be big for gestational age. GSH (AUC 0.72) was lower and prenatal MDA (AUC 0.78) was higher, which indicated postpartum glucose intolerance. Conclusions: Beyond a single biomarker, GDM entails a total oxidative imbalance. Improved screening methods for better maternal-fetal outcomes are supported by multi-dimensional biomarker panels, which offer superior risk assessment for perinatal problems and postpartum metabolic dysfunction.

Keywords: ischemia-modified albumin, malondialdehyde, oxidative stress, and gestational diabetes mellitus.

Introduction

One of the most common pregnancy complications is gestational diabetes mellitus (GDM), which affects roughly 14% of pregnancies globally, with significant regional variations ranging from 5.8% in Europe to 25.0% in southeast Asia, according to the international Diabetes Federation (IDF) [1,2]. Since the world Health Organization (WHO) approved the International Association of Diabetes and Pregnancy study Groups (IADPSG) criteria in 2010, the use of the 75-g oral glucose tolerance test (OGTT) with thresholds of fasting ≥5.1 mmol/L, 1-hour ≥10.0 mmol/L, and 2-hour ≥8.5 mmol/L has increased the recognition of GDM [3,4]. Pregnancy-induced insulin resistance, pancreatic β-cell dysfunction, and elevated oxidative stress interact intricately in the pathogenesis of GDM [5]. The formation of reactive oxygen species (ROS) is naturally increased during a normal pregnancy, and the mother's antioxidant stores are also depleted. Through a variety of processes, including as mitochondrial failure, protein glycation, and glucose auto-oxidation, persistent hyperglycemia in GDM aggravates this oxidative imbalance [6,7]. Recent studies have shown that pregnant women with GDM develop pathological insulin resistance and β-cell dysfunction; decreased insulin sensitivity index and increased insulin resistance are linked to their diminished capacity to compensate for oxidative stress [8]. Oxidative stress markers are consistently higher in pregnant women with GDM than in those with normal pregnancies, according to studies [9]. Research on GDM has showed promise with individual biomarkers. One byproduct of lipid peroxidation that indicates oxidative stress-induced damage to cellular membranes is malondialdehyde (MDA) [10]. The main intracellular antioxidant, glutathione (GSH), is diminished in diabetics [11]. As oxidative stress increases, catalase (CAT) activity falls, jeopardizing cellular defenses [12]. Integrated indicators of oxidative balance are provided by total oxidant status (TOS) and total antioxidant status (TAS) [13]. With studies demonstrating higher levels in GDM compared to a typical pregnancy, ischemia-modified albumin (IMA) has become a viable biomarker for assessing oxidative stress in pre-diabetes and gestational diabetes [14,15]. But as there is still a lack of methodological consistency, the usefulness of quantifying circulating IMA needs to be verified in prospective studies looking at clinical objectives [14]. The majority of investigations have concentrated on individual biomarkers with small sample sizes and brief follow-up periods, despite mounting evidence of oxidative stress involvement in GDM. Despite recommendations from the American college of Obstetricians and Gynecologists (ACOG) and the American Diabetes Association (ADA) for postpartum glucose test at 4-12 weeks, adherence is still below ideal [16,17]. Prenatal oxidative stress indicators may predict postpartum glucose intolerance, according to emerging

data, opening the door to early risk stratification [18]. In comparison to healthy controls, this study sought to: (1) analyze a comprehensive oxidative stress biomarker panel in women with GDM; (2) identify relationships with perinatal outcomes; and (3) evaluate predictive value for postpartum glucose tolerance at a follow-up of 6-12 weeks.

METHODS AND PATIENTS

Following the standards of the Helsinki Declaration and ethical approval, this prospective case-control study was carried out at three teaching hospitals in Mosul, Iraq, between April and December 2021. 150 pregnant women, age 18 to 40, with singleton pregnancies (75 GDM, 75 controls) during 24-28 weeks gestation were included in the study. The estimated 1.5 nmol/ml MDA variations between groups were used to calculate the sample size, which called for 64 per group with 15% attrition allowance. The 75-g OGTT was used to diagnose GDM in accordance with IADPSG guidelines, and abnormal values were determined as follows: fasting ≥5.1 mmol/L, 1-hour ≥10.0 mmol/L, or 2-hour ≥8.5 mmol/L. the glucose tolerance of the controls was normal for both maternal and gestational age. Multiple pregnancies, pre-gestational diabetes, chronic illnesses, and drugs that interfere with glucose metabolism were among the exclusions. Ten milliliters of fasting venous blood were drawn, placed in serum and EDTA tubes, centrifuged at 3000 rpm, and kept at -80°C. Oxidative indicators, such as albumin-cobalt binding (IMA), thiobarbituric acid method (MDA), and Erel's method (TOS), were assessed using standardized assays. TAS (Erel's method), GSH (spectrophotometric with Ellman's reagent), and CAT (hydrogen peroxide decomposition monitoring) were examples of antioxidant markers. Coefficients of variance were kept below 5% by quality control. Demographics, physical examinations, BMI calculations, dietary evaluations, and supplement documentation were all part of the clinical data gathering process. The following perinatal outcomes were tracked: birthweight, delivery problems, LGA babies, and macrosomia. Using a75-g OGTT, postpartum glucose tolerance was evaluated at 6-12 weeks and categorized as normal (<7.8 mmol/L), impaired (7.8–11.0 mmol/L), or diabetes (≥11.1 mmol/L). strict quality control procedures and established techniques were used in all studies.

STATISTICAL ANALYSIS

A statistical examination IBM Corp., Armonk, NY's SPSS version 28,0 was used for the statistical analysis. Mean ± standard deviation was used to express continuous variables, and independent t-test were used for comparison. Using chi-square tests, categorical variables were compared and displayed as frequencies and percentages. Multivariate Analysis: Logistic regression models that took into account dietary determinants, maternal age, parity, BMI, and family history of diabetes. Predictive modeling: Analysis of receiver operating characteristic (ROC) curves yielded the best cut-off values and area under the curve (AUC) for predicting postpartum glucose intolerance. Application of Bonferroni correction for multiple biomarker comparisons ($\alpha = 0.05/6 = 0.008$) is known as multiple testing correction. For primary analyses, $p<0.05$ and for multiple comparison, $p<0.008$ were considered statistically significant.

RESULTS

3.1 Participant Characteristics

150 pregnant women in all were enrolled (75 with GDM and 75 as controls). Table 1 displays the baseline attributes. The pre-pregnancy BMI was higher in women with GDM (28.4 ± 4.1 vs 25.2 ± 3.6 kg/m^2, $p<0.001$), they were considerably older (29.8 ± 4.2 vs 27.3 ± 3.8 years, $p<0.001$), and they had a higher likelihood of having a family history of diabetes (68% vs 23%, $p<0.001$).

Table 1. Baseline Characteristics of Study Participants

Variable	GDM (n=75)	Controls (n=75)	p-value
Maternal age (years)	29.8 ± 4.2	27.3 ± 3.8	<0.001
Pre-pregnancy BMI (kg/m²)	28.4 ± 4.1	25.2 ± 3.6	<0.001
Gestational age at sampling (weeks)	26.2 ± 1.8	26.0 ± 1.9	0.523
Nulliparous, n (%)	28 (37.3)	32 (42.7)	0.521
Family history of T2DM, n (%)	51 (68.0)	17 (22.7)	<0.001
Systolic BP (mmHg)	118 ± 12	112 ± 10	0.002
Diastolic BP (mmHg)	74 ± 8	70 ± 7	0.004

3.2 Glucose Metabolism Parameters

Women with GDM had significantly higher fasting glucose (5.8 ± 0.9 vs 4.3 ± 0.4 mmol/L, p<0.001), 1-hour OGTT values (11.2 ± 1.8 vs 7.1 ± 1.2 mmol/L, p<0.001), 2-hour OGTT values (9.4 ± 1.6 vs 6.2 ± 0.8 mmol/L, p<0.001), and HbA1c levels (6.8 ± 0.7 vs 5.2 ± 0.3%, p<0.001).

3.3 Oxidative Stress Biomarkers

All oxidative stress markers differed significantly between groups (Table 2). Women with GDM demonstrated elevated oxidative markers and decreased antioxidant capacity.

Table 2. Oxidative Stress Biomarkers in GDM vs Controls

Biomarker	Adjusted OR (95% CI)	p-value
IMA (per 0.1 ABSU increase)	**3.45 (2.18-5.46)**	**<0.001**
TOS (per 5 µmol/L increase)	**2.12 (1.52-2.96)**	**<0.001**
MDA (per 1 nmol/mL increase)	**2.78 (1.89-4.09)**	**<0.001**
TAS (per 0.2 mmol/L decrease)	**2.34 (1.61-3.40)**	**<0.001**
GSH (per 50 µmol/L decrease)	**1.89 (1.34-2.67)**	**<0.001**
CAT (per 10 U/mg decrease)	**2.01 (1.43-2.82)**	**<0.001**

3.4 Perinatal Outcomes

GDM was associated with increased rates of adverse perinatal outcomes (Table 3). Elevated oxidative stress markers predicted specific complications.

Table 3. Perinatal Outcomes by GDM Status

Outcome	GDM (n=75)	Controls (n=75)	OR (95% CI)	p-value
Preterm delivery (<37 weeks)	12 (16.0%)	5 (6.7%)	2.67 (0.92-7.74)	0.067
Macrosomia (>4000g)	18 (24.0%)	3 (4.0%)	7.50 (2.11-26.67)	<0.001
LGA infants	22 (29.3%)	6 (8.0%)	4.80 (1.84-12.52)	0.001
Cesarean delivery	35 (46.7%)	18 (24.0%)	2.78 (1.43-5.40)	0.002
NICU admission	14 (18.7%)	4 (5.3%)	4.06 (1.28-12.87)	0.015

Predictive Analysis for LGA Infants:

TOS >25 μmol H_2O_2 Eq/L: OR 2.34 (95% CI 1.45-3.78), p<0.001

MDA >4.0 nmol/mL: OR 1.89 (95% CI 1.23-2.91), p=0.004

TAS <1.3 mmol Trolox Eq/L: OR 2.12 (95% CI 1.38-3.26), p=0.001

3.5 Postpartum Follow-up Results

Of 150 participants, 127 (84.7%) completed postpartum OGTT (65 GDM, 62 controls). Among women with GDM, 23 (35.4%) developed impaired glucose tolerance and 8 (12.3%) developed type 2 diabetes. All controls maintained normal glucose tolerance.

Table 4. Postpartum Glucose Tolerance

Category	GDM group (n=65)	Control group (n=62)
Normal glucose tolerance	34 (52.3%)	62 (100%)
Impaired glucose tolerance	23 (35.4%)	0 (0%)
Type 2 diabetes	8 (12.3%)	0 (0%)

3.6 Predictive Value for Postpartum Glucose Intolerance

ROC curve analysis identified optimal cut-off values for predicting postpartum glucose intolerance (Table 5).

Table 5. ROC Analysis for Postpartum Glucose Intolerance Prediction

Biomarker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)	p-value
MDA	0.78 (0.65-0.91)	>4.2 nmol/mL	74.2	76.5	<0.001
TOS	0.72 (0.58-0.86)	>26 μmol/L	71.0	70.6	0.002
GSH	0.72 (0.58-0.86)	<300 μmol/L	67.7	73.5	0.003
IMA	0.69 (0.54-0.84)	>0.85 ABSU	64.5	70.6	0.008
TAS	0.67 (0.52-0.82)	<1.3 mmol/L	61.3	68.7	0.016
CAT	0.64 (0.48-0.80)	<45 U/mg	58.1	64.7	0.045

Composite Score: A composite oxidative stress score combining MDA, TOS, and GSH achieved AUC 0.83 (95% CI 0.72-0.94), with 80.6% sensitivity and 79.4% specificity for predicting postpartum glucose intolerance.

Discussion

Talking about the multifaceted oxidative imbalance involving increased oxidative markers (IMA, TOS, MDA) and diminished antioxidant defenses (TAS, GSH, CAT) is what this thorough analysis shows to be a hallmark of GDM[19]. Our results build on earlier studies by offering the most comprehensive biomarker panel analysis in GDM with prolonged postpartum follow-up, demonstrating clinically significant correlations with both postpartum glucose tolerance and perinatal outcomes. Our results support the use of IMA as a biomarker for oxidative stress[20-23], with a substantial effect size (Cohen's d= 2,31) confirming earlier findings that serum IMA levels are higher in GDM compared to normal pregnancy[24-26]. While IMA has the potential to detect the existence of oxidative stress, its clinical relevance needs to be confirmed in prospective studies that look at clinical endpoints, which is what our work does by evaluating perinatal and postpartum outcomes[27]. Significant lipid peroxidation is indicated by the observed MDA rise (4.8 ± 1.2 vs. 2.9 ± 0.8 nmol/mL, p<0.001) in GDM, which is in line with earlier studies that found higher oxidative stress indicators in pregnant GDM patients. Given that MDA contributes to cellular membrane damage and is linked to problems in diabetes, this finding is especially pertinent [28]. Our multivariate study supports the association between oxidative stress and insulin resistance in GDM by demonstrating that oxidative indicators are still strongly linked to GDM even after controlling for conventional risk variables. This shows

that oxidative stress might both contribute to and be a result of the pathophysiology of GDM. With lower TAS, GSH, and CAT levels, our GDM cohort showed widespread antioxidant depletion, which suggests that the mother's antioxidant defenses are overloaded. Since GSH is the main intracellular antioxidant and a cofactor for glutathione peroxidase activity, its depletion is especially important [29]. A vicious loop oxidative damage is created when hydrogen peroxide detoxification is compromised by catalase decrease [30]. These results imply that GDM is a condition of oxidative- antioxidant imbalance with possible treatment implications rather than only glucose intolerance. Pregnancy-related targeted antioxidant supplements may aid in reestablishing this equilibrium, but efficacy and safety must be established through randomized controlled trials. Mechanistic insights into GDM problems can be gained from the correlation between higher oxidative stress markers and unfavorable neonatal outcomes. The prediction of LAG newborns by TOS and MDA increase raises the possibility that maternal oxidative stress may effect fetal growth through altered nutrition transport and placental malfunction [31]. Our GDM cohort's 7,50- fold higher risk of macrosomia is consistent with previous research, but it also offers new biomarker linkages for risk stratification. Our study uses particular biomarker criteria to quantify the elevated oxidative stress associated with GDM, which has been linked to difficulties for both pregnant women and their unborn children. The discovery of MDA >4.0 nmol/mL and TOS >25 μmol H_2O_2 Eq/L as predictive thresholds may have therapeutic applications for improved monitoring. Our discovery that postpartum glucose intolerance (35.4% IGT, 12.3% T2DM) occurred in 47.7% of women with GDM highlights the urgent need for improved follow-up. An innovative method of risk classification is provided by the capacity of prenatal oxidative stress indicators to forecast postpartum glucose intolerance. Since MDA was the best predictor (AUC 0.78), it is possible that lipid peroxidation during pregnancy is a reflection of underlying metabolic dysfunction that continues after delivery. Personalized postpartum monitoring tactics may be guided by the composite score (AUC 0.83) that combines various indicators, which offers superior prediction performance.

Conclusions

According to this study, a thorough oxidative imbalance encompassing several pathways outside of glucose metabolism is hallmark of GDM. With clinical relevance for perinatal outcomes prediction and postpartum risk stratification, the multi-biomarker method offers better insights into metabolic risk than single markers. Personalized preventative measures are made possible by the capacity of antenatal oxidative stress markers to predict postpartum glucose intolerance. Evaluation of cost-effectiveness and validation across various groups are necessary for implementation, nevertheless. Structured postpartum screening with 75-g OGTT at 6-12 weeks remains essential for all women with GDM, with enhanced follow-up recommended for those with elevated oxidative stress markers during pregnancy.

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