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Serum Zinc α-2-Glycoprotein Level and Related Factors in Gestational Diabetes Mellitus

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ABSTRACT

Background: Gestational diabetes mellitus (GDM), affecting up to 14% of pregnancies, is linked to obesity, sedentary lifestyle, and advanced maternal age. It is characterized by insulin resistance and adipose tissue inflammation. Zinc α-2-glycoprotein (ZAG) promotes lipid metabolism and glucose utilization, potentially linking it to GDM pathophysiology.
Objectives: To evaluate the association between serum ZAG levels and GDM.
Materials and methods: This case-control study was conducted at Al-Emamein Al-Kadhimein Medical City, Baghdad, Iraq, and included 100 pregnant women (24–28 weeks gestation), equally divided into GDM cases and healthy controls. Data collection included interviews, clinical exams, oral glucose tolerance tests, and serum ZAG level measurements.
Results: There were no statistically significant differences (P-value $>$ 0.05) in age, body mass index, or parity between the GDM group and the control group. However, GDM was significantly associated with a family history of diabetes and a history of macrosomia (P-value $<$ 0.05). Patients with GDM had significantly higher levels of fasting blood sugar, hemoglobin A1c, and two-hour postprandial glucose compared to the control group (P-value = 0.0001). Serum ZAG levels were significantly lower in the GDM group (40.49 $\pm$ 7.63 ng/mL) than in the control group (55.7 $\pm$ 10.96 ng/mL), with a statistically significant difference (P-value = 0.0001). A cutoff value of $\geq$ 50.5 ng/mL was identified for ZAG in distinguishing between the two groups.
Conclusion: This study suggests a potential role of ZAG in glucose metabolism during pregnancy and highlights its possible utility as a biomarker for metabolic alterations in GDM. However, as a single-center study with a relatively small sample size, further research is warranted to validate these findings and assess the clinical relevance of ZAG in GDM.
Keywords: Diabetes mellitus; Gestational diabetes mellitus; Serum zinc α-2-glycoprotein; Pregnant women.

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INTRODUCTION

Gestational diabetes mellitus (GDM) is the most common metabolic complication of pregnancy, affecting up to 14% of all pregnancies. GDM is defined as any degree of glucose intolerance that is first recognized during pregnancy, regardless of whether the condition persists after delivery [1]. If left untreated, GDM can lead to significant maternal and fetal complications, including pre-eclampsia, Cesarean-section delivery, birth trauma, macrosomia, neonatal hypoglycemia, and hyperbilirubinemia. In the long term, women with GDM face an increased risk of developing type 2 diabetes mellitus (T2DM), metabolic syndrome, and cardiovascular disease. One of the most common risk factors for GDM is excessive gestational weight gain, often due to overweight and obesity, which are prevalent in pregnancy [2–4].

Given these complications and the growing prevalence of obesity in pregnancy, identifying biomarkers that may contribute to the pathophysiology of GDM is crucial. Zinc α-2-glycoprotein (ZAG) is a protein primarily produced by the liver and adipose tissue, although it is also found in various other cell types, including specific cancer cells. It is secreted into the bloodstream and is involved in several physiological and pathological processes. Recent studies suggest that ZAG levels may be altered in women with GDM. Specifically, lower serum ZAG concentrations have been observed in pregnant

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women with GDM compared to their healthy counterparts, indicating a possible association between ZAG and glucose metabolism during pregnancy [5, 6].

The potential role of ZAG in GDM has been linked to two key mechanisms: insulin resistance and adipose tissue dysfunction. Insulin resistance is a central feature of GDM, and ZAG is believed to influence insulin sensitivity and glucose uptake. Additionally, since adipose tissue is a significant site of ZAG production, dysfunction in this tissue, which is common in GDM, may contribute to altered ZAG levels [4]. While these findings suggest a possible pathophysiological role for ZAG in GDM, further research is required to clarify the underlying mechanisms and evaluate its clinical significance [7].

In this context, the current study aims to investigate the association between serum ZAG levels and blood glucose concentrations in pregnant women diagnosed with GDM, thereby contributing to the growing body of evidence exploring ZAG's potential as a metabolic marker in pregnancy.

MATERIALS AND METHODS

This case-control study was conducted at the Department of Obstetrics and Gynecology, Al-Emamein Al-Kadhimein Medical City, Baghdad, Iraq, over one year from October 1, 2022, to September 30, 2023. Ethical approval was obtained from the Ethical Committee of the Iraqi Board for Medical Specialization in Obstetrics and Gynecology (Reference Number: 143, dated September 30, 2022). Informed consent was obtained from all participants prior to enrollment.

A total of 100 pregnant women between 24 and 28 weeks of gestation were included and equally divided into two groups. The GDM group consisted of 50 women diagnosed with GDM using the oral glucose tolerance test (OGTT). In comparison, the control group included 50 healthy pregnant women with normal OGTT results and uneventful antenatal progression. Eligible participants in the GDM group were women aged 18–45 years, with singleton viable pregnancies, at 24–28 weeks of gestation, and diagnosed with GDM. Exclusion criteria included pre-existing diabetes, hypertension, multiple gestation, or chronic illnesses. Healthy pregnant women were included as controls if they were between 24 and 28 weeks of gestation, had a normal OGTT, and had no history of diabetes mellitus or other metabolic or endocrine disorders. Exclusion criteria for the control group included pre-existing or gestational diabetes, hypertension, thyroid dysfunction, acute or chronic inflammatory conditions, multiple gestation, current use of corticosteroids or other medications affecting metabolism, or any obstetric complications. The required sample size was calculated based on the expected difference in mean ZAG levels between GDM cases and healthy controls, using the following equation:

$n = 2(Z\alpha/2 + Z\beta)^2\sigma^2/\Delta^2$

where Δ (difference in means) = 5 mg/L, σ (standard deviation) = 10 mg/L, and $\alpha = 0.05$, Power = 80% $\Rightarrow$ $Z_{\alpha/2} = 1.96$, $Z\beta = 0.84$. Accordingly, 63 participants were required per group. However, due to time and resource constraints, 50 cases were enrolled in each group during the study period.

Data were collected through direct interviews using a structured and validated questionnaire, designed specifically for this study to capture demographic data, obstetric history, family history of diabetes, and previous obstetric complications, such as macrosomia. This questionnaire was pilot-tested with 10 participants to ensure clarity before the main study.

Body mass index (BMI) was estimated according to the following equation: BMI = weight (kg)/height $(m)^2$. BMI was classified according to World Health Organization (WHO) criteria as follows: underweight (< 18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0—29.9 kg/m^2), and obese (≥ 30 kg/m^2).

All patients underwent a 75-gram OGTT in accordance with the recommendations of the American Diabetes Association (ADA) [8]. Following an overnight fast of 8 to 14 hours, a baseline venous blood sample was collected. Participants then consumed 75 grams of glucose dissolved in 300 mL of water. During the test, they were instructed to remain seated and refrain from eating or drinking. Additional venous blood samples were drawn at 1 and 2 hours after glucose ingestion. The test was administered uniformly, regardless of body weight, in accordance with the ADA 2010 guidelines, which do not require weight-based adjustments for the 75 g OGTT [9]. Hemoglobin A1c (HbA1c) levels were measured using high-performance liquid chromatography (HPLC), which is a standardized and highly accurate method for evaluating long-term glycemic control. The test was conducted with diagnostic kits specific for HPLC [not enzyme-linked immunosorbent assay (ELISA kits)], and results were interpreted according to ADA 2010 criteria: Normal: $< 5.7\%$, prediabetes: 5.7%-6.4%, and diabetes: $\geq 6.5\%$. Fasting lipid profile was assessed using enzymatic colorimetric assays, measuring total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides. Ideal reference values were based on standard clinical guidelines (total cholesterol < 200 mg/dL, LDL < 100 mg/dL, HDL ≥ 50 mg/dL, and triglycerides < 150 mg/dL). Serum ZAG levels were measured using a commercially available kit (MyBioSource, USA), following the manufacturer's instructions. The minimum detectable concentration was 1.80 ng/mL, with intra-assay and inter-assay coefficients of variation of 3.08% and 15.32%, respectively. The blood samples used to measure ZAG levels were collected separately after confirming the GDM diagnosis with the OGTT. Participants provided these samples while fasting at a follow-up visit. Therefore, when we compared ZAG levels with fasting and 2-hour blood sugar results, we used measurements from these later visits, not from the original glucose tolerance test.

Data were entered into Microsoft Excel 2016 and analyzed using Statistical Package for the Social Sciences (SPSS) Statistics version 26 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including mean, standard deviation, frequency, and percentage, were used to summarize the baseline characteristics. Categorical variables were expressed as frequencies and percentages and analyzed using the Chi-square test or Fisher's exact test, as appropriate. Continuous variables were analyzed using Student's t-test or the Mann–Whitney U test, depending on the data distribution. A P-value of < 0.05 was considered a statistically significant difference.

RESULTS

Age distribution between the GDM group (25.54 ± 3.69 years) shows a slight difference compared to the control group (26.26 ± 4.03 years); however, this difference isn't statistically significant (P-value > 0.05). BMI categories (normal, overweight, obese) were similarly distributed in both groups, though the GDM group had a slightly higher average BMI. Likewise, parity (number of previous pregnancies) showed no significant difference (P-value > 0.05), with both groups hav-

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Table 1. Distribution of patients' demographics according to the studied groups*.

Variables	GDM group (n=50)		Control group (n=50)		P-value	
Maternal age per year (Mean ± SD)	25.54 ± 3.69		26.26 ± 4.03		0.341	
Body mass index	Normal	23	46	23	46	0.634
	Overweight	19	38	22	44	
	Obese	8	16	5	10	
	Mean ± SD	26.11 ± 3.61		25.02 ± 3.63		0.150
Parity (Mean ± SD)	1.84 ± 1.43		1.94 ± 1.32			0.691
Gestational Age (weeks)	26.18 ± 1.45		26.12 ± 1.44			0.853
Family history of DM	Yes	17 (34)	6 (12)			0.009
	No	33 (66)	44 (88)			
History of macrosomia	Yes	28 (56)	10 (20)			0.0001
	No	22 (44)	40 (80)			

* GDM: Gestational diabetes mellitus, SD: Standard deviation, DM: Diabetes mellitus.

Table 2. Metabolic parameters of patients' investigations according to the studied groups*.

Variables	GDM Mean ± SD	Control Mean ± SD	P-value
FBS* (mg/dl)	104.8 ± 16.05	79.72 ± 7.65	0.0001
HbA1C (%)	5.51 ± 0.81	5 ± 0.66	0.001
1-hour post prandial (mg/dl)	163.52 ± 29.98	113.46 ± 7.32	0.0001
2-hours post prandial (mg/dl)	155.14 ± 22.11	103.78 ± 4.64	0.0001
Total cholesterol (mg/dl)	257.92 ± 19.33	201.24 ± 8.58	0.002
Triglycerides (mg/dl)	193 ± 13.44	149 ± 14.33	0.005
Low-Density Lipoprotein (mg/dl)	188.7 ± 21.3	134.6 ± 17.5	0.001
High-Density Lipoprotein (mg/dl)	41.6 ± 9.3	60.3 ± 10.3	0.020

* GDM: Gestational diabetes mellitus, SD: Standard deviation, FBS: Fasting blood sugar, HbA1C: Hemoglobin A1c.

ing comparable averages around 2 previous pregnancies. Finally, gestational age at assessment is almost identical across the two groups, averaging approximately 26 weeks (P-value > 0.05). Overall, none of these maternal characteristics showed statistically meaningful differences (P-value > 0.05). Cases of GDM were significantly (P-value < 0.05) associated with family history of diabetes, along with history of macrosomia in previous pregnancies, in comparison with controls (Table 1). Women with GDM exhibit significantly higher FBS levels, averaging approximately 105 mg/dL compared to about 81 mg/dL in the control group. Similarly, HbA1C was higher among GDM patients, averaging around 5.5% compared to 5% in the control group. Postprandial glucose measurements further emphasized these differences, with the GDM group showing markedly higher glucose levels both one and two hours after meals compared to the control group. Lipid profiles also differed significantly; GDM patients had higher levels of total cholesterol, triglycerides, and LDL ("bad" cholesterol), while their HDL ("good" cholesterol) levels were lower. Each of these observed differences was statistically significant (P-value < 0.05) (Table 3).

Interestingly, the mode for ZAG levels was 51 in the study group and 49.1 for the control group. The P-value for the difference in ZAG levels between the two groups was 0.0001, indicating a highly significant statistical difference (Table **??**).

In the GDM group, ZAG levels showed almost no noticeable correlation with FBS, indicated by a nearly horizontal trend line. In contrast, the control group exhibited a mild negative correlation, suggesting that as FBS levels increased, ZAG levels slightly decreased (Figure 1).

Table 3. Zinc α-2-glycoprotein (ZAG) between gestational diabetes mellitus (GDM) and control groups*.

ZAG*	GDM group	Control group
Mean (ng/ml)	40.49	55.7
Standard Deviation	7.63	10.96
Range	26	38.7
Percentile 25	34.2	48.1
Median	40.9	54.85
Percentile 75	47.6	63.1
Percentile 95	51	73.7
Mode	51	49.1

* P-value =0.0001

The GDM group exhibits a slight positive correlation, meaning that as BMI increases, ZAG levels also rise modestly. Conversely, in the control group, the correlation appears slightly negative, indicating that ZAG levels marginally decrease with increasing BMI (Figure 2). The ROC curve demonstrates that ZAG has a reasonably good ability to differentiate between women with and without GDM, as indicated by the curve rising above the diagonal reference line. The area under the curve (AUC), visibly larger than the diagonal baseline, suggests that ZAG can potentially serve as a meaningful indicator for detecting GDM (Figure 3).

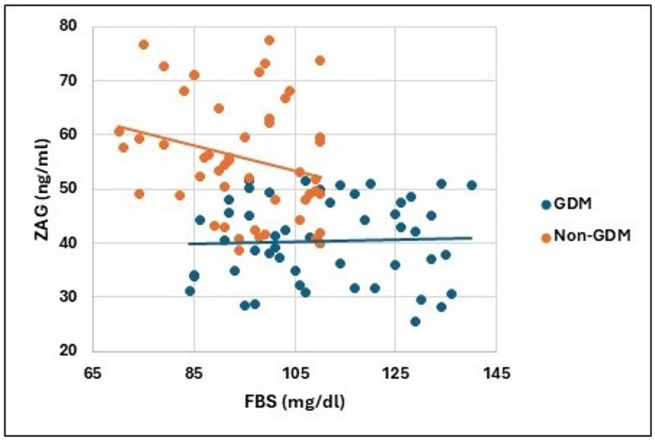

Figure 1. Correlation analysis of zinc α-2-glycoprotein (ZAG) and fasting blood sugar (FBS) between the gestational diabetes mellitus (GDM) group and controls.

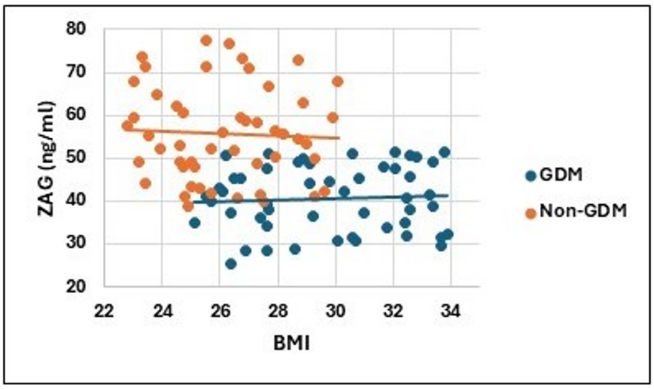

Figure 2. Correlation analysis of zinc α-2-glycoprotein (ZAG) and body mass index (BMI) between the gestational diabetes mellitus (GDM) group and controls.

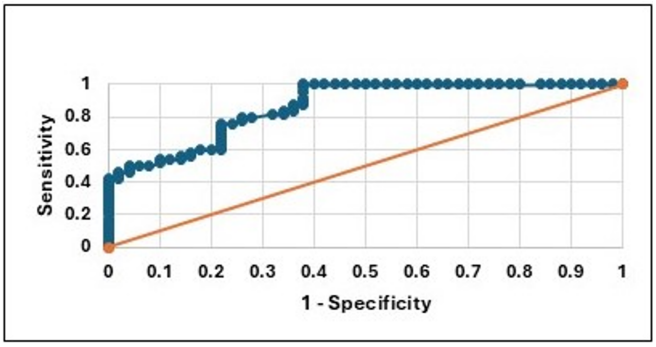

Figure 3. ROC curve analysis of zinc α-2-glycoprotein in identifying gestational diabetes mellitus.

DISCUSSION

The global prevalence of GDM continues to rise, driven mainly by epidemiological factors such as increasing rates of obesity among women of reproductive age and advancing maternal age [10]. The study included 100 participants, 50 participants in each case and control group. The maternal age was not different between the two groups; this was intentionally selected to eliminate selection bias. To note that advancing maternal age is associated with an increasing risk of gestational diabetes, as stated by previous studies [11, 12].

The family history of diabetes was significantly associated with GDM; this highlights the possibility of familial or genetic causes that could lead to the development of GDM. Farahvar et al. [13] in their systematic review found that the family history of T2DM increases the possibility of GDM in the offspring. Clinically, this highlights the need for early screening and targeted monitoring of pregnant women with a family history of diabetes to help prevent complications related to GDM.

A previous history of GDM is significantly associated with an increased risk of delivering macrosomic infants. Macrosomia is primarily attributed to elevated maternal glucose levels, which cross the placenta and stimulate fetal insulin-like growth factor secretion, promoting excessive fetal growth, as described by Alekseenkova et al. [14]. Additionally, Kouhkan et al. [15] Reported that a history of macrosomia is itself a significant risk factor for the subsequent development of GDM, suggesting a bidirectional relationship between GDM and macrosomia.

The FBG mean was significantly higher in cases of GDM than incontrol. Previous studies found that the FBS is used during the first trimester to predict the future development of GDM [16, 17]. While in the third trimester; elevated maternal FBS is associated with poor pregnancy outcome as found in previous studies by Zhao et al. [18] and Panyakat et al. [19]. The mean HbA1c was significantly higher in cases of GDM. The diagnosis of GDM remains an area of controversy; however, previous studies, including that by Ye et al., have suggested that HbA1c may serve as a useful predictor of adverse pregnancy outcomes in women with GDM [20]. While Lai,Yi et al. concluded that the HbA1c test offers limited value in diagnosing GDM [21].

The mean level of ZAG was significantly lower in cases of GDM in comparison to control. Similarly, Xu et al. [6] that serum ZAG tend to be lower in cases of GDM than in controls . The possible mechanism underlying this relationship may be attributed to the role of ZAG in insulin resistance. As suggested by Balaz et al., ZAG influences adipose tissue function by reducing the expression of adiponectin, insulin receptor substrate-1 (IRS-1), and glucose transporter-4 (GLUT4) genes in primary human adipocytes. This downregulation may impair insulin signaling and contribute to the development of insulin resistance [22]. In contrast, a study by Näf et al. [23], which measured maternal ZAG levels at the end of pregnancy along with ZAG concentrations in cord blood, suggested that ZAG is not directly associated with glucose metabolism in late pregnancy. Instead, the study found that ZAG is more closely linked to adipose tissue function, fat metabolism, and is strongly correlated with gestational age. Notably, our research and the investigation performed by Xu et al. [6] assessed the role of ZAG in the diagnosis of GDM during the 2nd trimester, rather than at the time of parturition. This consistency in timing may account for the observed alterations in ZAG levels. Additionally, in both studies, ZAG assessments were performed within a narrow and controlled gestational age range, with no significant differences between the two groups (GDM group and control group). This approach helps to reduce the potential confounding factor of gestational age on the diagnostic accuracy of ZAG. In con-

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trast, Näf et al. [23] performed their ZAG assessments at the end of pregnancy, which may account for the discrepancies in results.

The present study reported no statistically significant association between serum ZAG levels and variables such as BMI and FBS. In contrast, Xu et al. [6] reported a significant negative correlation between ZAG levels and FBS, suggesting that ZAG decreases as FBS increases. A possible explanation for this discrepancy is that, in the present study, the analysis within the GDM group showed limited variability in FBS values (with a standard deviation of 16.05 mg/dL), resulting in a narrow range of data around the mean. This restricted variability may have limited the ability to detect a meaningful correlation.

In the present study, serum ZAG levels showed a significant positive correlation with 2-hour postprandial glucose levels in pregnant women without gestational diabetes (control group), suggesting a possible role for ZAG in glucose utilization. While previous studies have examined serum ZAG levels in the context of GDM, they did not establish an optimal cutoff point for its diagnostic use. In this study, receiver operating characteristic (ROC) curve analysis demonstrated that ZAG has good diagnostic potential, with an area under the curve (AUC) of 0.866 and an optimal cutoff value of $\leq$ 50.5 ng/mL for identifying GDM.

This study has several limitations that should be taken into consideration. First, a relatively small sample size, may reduce statistical power and limit the generalizability of the findings. Second, the use of a single-center population may not fully represent broader demographic or clinical variations. Additionally, variability in serum ZAG assay methods could influence the accuracy and consistency of measurements across studies. The cross-sectional design and absence of longitudinal follow-up data further limit the ability to assess causal relationships or changes in ZAG levels throughout pregnancy. Future multicenter studies with larger cohorts and standardized measurement techniques are recommended to validate and expand upon these findings.

CONCLUSION

The findings of this study suggest that ZAG may play a meaningful role in metabolic regulation during pregnancy, with lower serum levels observed in women with GDM. While the association between ZAG and specific metabolic parameters did not reach statistical significance, the strong diagnostic potential reflected by the ROC analysis highlights ZAG as a promising biomarker for early identification of GDM risk. Given its potential diagnostic value and role in glucose metabolism, ZAG could be further explored as a supplementary biomarker for GDM screening, especially in resource-limited settings. Future large-scale, longitudinal, and multicenter studies are recommended to confirm these findings, clarify the mechanisms linking ZAG to insulin resistance, and evaluate its predictive value in clinical practice.

ETHICAL DECLARATIONS

Acknowledgments

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Ethics Approval and Consent to Participate

This study was approved by the Ethical Committee of the Iraqi Board Scientific Council for Medical Specialization in Obstetrics and Gynecology (Reference Number: 143, dated 30-09-2022). Informed consent was obtained from all participants prior to inclusion in the study.

Consent for Publication

Not applicable (no individual personal data included).

Availability of Data and Material

Data collected during the study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that there is no conflict of interest.

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Authors' Contributions

Both authors have equal participation in the design, collection of the data, analysis of the results, and writing of the manuscript. Both authors read and approved the final version of the manuscript.

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