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The Effect of Depression and Stress Hormones on the Development of Gestational Diabetes Mellitus

Gebelik Diyabetinin Gelişiminde Depresyon ve Stres Hormonlarının Etkisi

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ABSTRACT

Objective: This study aims to determine the effect of depression and stress hormones on the development of gestational diabetes mellitus (GDM) in pregnant women diagnosed with GDM using serum cortisol, plasma adrenaline (A), plasma noradrenaline (NA), and the Beck Depression Inventory (BDI).

Methods: 70 pregnant women diagnosed with GDM were included in the patient group, and 70 pregnant women without GDM were included in the control group. International Association of Diabetes and Pregnancy Study Group criteria were used for the diagnosis of GDM. Single-step 75 g oral glucose tolerance test was performed at 24-28 weeks of gestation. Serum cortisol, A, and NA levels were measured. BDI was used to investigate depressive symptoms.

Results: The patient and control groups were similar in terms of age, BMI gravidity, and parity. When compared with the control group, A and NA levels were significantly higher in the patient group (p=0.016, p=0.033, respectively). BDI results in the patient group were similar to those in the control group (p=0.151). The mean A levels of 33 pregnant women with minimal depression were 110.59±35.03 pg/mL, the mean A levels of 31 pregnant women with mild depression were 126.65±22.33 pg/mL, and the mean A levels of pregnant women with moderate depression were 95.09±30.86 pg/mL. This difference was statistically significant (p=0.005).

ÖZ

Amaç: Bu çalışmanın amacı, gestasyonel diyabet mellitus (GDM) tanısı almış gebelerde depresyon ve stres hormonlarının GDM gelişimi üzerindeki etkisini; serum kortizol, plazma adrenalin (A), plazma noradrenalin (NA) düzeyleri ve Beck Depresyon Ölçeği (BDI) kullanarak değerlendirmektir.

Yöntemler: Çalışmaya, GDM tanısı almış 70 gebe kadın hasta grubuna ve GDM tanısı olmayan 70 gebe kadın kontrol grubuna dahil edilmiştir. GDM tanısı için Uluslararası Diyabet ve Gebelik Çalışma Grubu Birliği kriterleri kullanılmış ve gebeliğin 24-28. haftaları arasında tek adımlı 75 g oral glukoz tolerans testi uygulanmıştır. Serum kortizol, A ve NA düzeyleri ölçülmüş; depresif semptomları değerlendirmek amacıyla BDI kullanılmıştır.

Bulgular: Hasta ve kontrol grupları yaş, vücut kitle indeksi, gravide ve parite açısından benzer bulunmuştur. Kontrol grubuna kıyasla, hasta grubunda adrenalin ve noradrenalin düzeyleri anlamlı derecede yüksek saptanmıştır (sırasıyla p=0,016 ve p=0,033). BDI sonuçları açısından ise gruplar arasında anlamlı bir fark bulunmamıştır (p=0.151). Minimal depresyonu olan 33 gebenin ortalama A düzeyi 110,59±35,03 pg/mL, hafif depresyonu olan 31 gebenin 126,65±22,33 pg/mL, orta derecede depresyonu olan gebelerin ise 95,09±30,86 pg/mL olarak ölçülmüştür. Bu fark istatistiksel olarak anlamlı bulunmuştur (p=0.005).

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ABSTRACT

Conclusion: This study suggests that the sympathoadrenal system may play a role in the etiopathogenesis of GDM in pregnant women rather than depression. However, larger prospective studies are needed to further elucidate the relationship between depression, stress hormones, and GDM.

Keywords: Gestational diabetes, depression, adrenaline, noradrenaline, cortisol, sympathoadrenal axis

ÖZ

Sonuç: Bu çalışma, gebelerde GDM'nin etiyopatogenezinde depresyondan ziyade sempatoadrenal sistemin rol oynayabileceğini göstermektedir. Ancak, depresyon, stres hormonları ve GDM arasındaki ilişkiyi daha iyi ortaya koymak için daha geniş örneklemli ve prospektif çalışmalara ihtiyaç vardır.

Anahtar Sözcükler: Gestasyonel diyabet, depresyon, adrenalin, noradrenalin, kortizol, sempatoadrenal aks

INTRODUCTION

Gestational diabetes mellitus (GDM) is characterized by high blood glucose levels that emerge in pregnancy and persist for the duration of the pregnancy (1). This metabolic disorder is associated with various complications, including miscarriage, fetal abnormalities, preeclampsia, stillbirth, macrosomia, and more. Moreover, individuals with GDM could face a greater risk of obesity, hypertension, and type 2 diabetes (2).

GDM is typically diagnosed through glucose tolerance testing, with the most commonly used method being the 75 g oral glucose tolerance test (OGTT). Alternatively, a two-step testing protocol, 50 g and 100 g OGTTs, can be used (3). Timely diagnosis and appropriate management play a crucial role in minimizing the potential health risks for both mother and baby.

Cortisol and plasma adrenaline (A), are released from the adrenal gland, while plasma noradrenaline (NA), comes from sympathetic nerves. During pregnancy, hormone levels, particularly cortisol, rise due to the effects of the placenta. Levels of NA and A remain stable at this time (4). Holzman et al. (5) discovered that elevated NA and A levels are linked to preterm births. Almon et al. (6) report that women with GDM show increased cortisol levels, which contribute to insulin resistance and promote GDM. Furthermore, high cortisol levels are associated with preterm births, miscarriages, and preeclampsia (6).

Social, genetic, and psychological factors are known to influence the development of GDM, placing both mothers and offspring at heightened risk for various physical and psychological complications (7,8). Mental health concerns, particularly among pregnant women at high risk for GDM, have attracted significant research interest globally. Studies conducted within this population suggest that anxiety and depression contribute substantially to the development of GDM alongside physiological factors (9). However, prior research exploring the relationship between depression and GDM has yielded conflicting results (10-14). This study aims to explore the influence of depression and stress hormones on emerging GDM, using serum cortisol, and Beck Depression Inventory (BDI) as key indicators.

MATERIALS AND METHODS

This study was approved by the Ankara Bilkent City Hospital No. 2 Clinical Clinical Research Ethics Committee (approval number: e2-23-3189, date: 18.01.2023) and complied with the principles outlined in the Helsinki Declaration. Pregnant women who attended an antenatal clinic between August 2022 and April 2023 and satisfied specific inclusion criteria were recruited. The criteria included the absence of risk factors or coexisting conditions such as hypertension, epilepsy, rheumatologic disorders, kidney disease, and thrombophilia. Furthermore, participants had no previous diagnosis of depression or history of antidepressant use, were between the 24th and 28th weeks of pregnancy, and underwent a 75 g OGTT. The study involved 70 pregnant women diagnosed with GDM as part of the patient group, while another 70 pregnant women without GDM formed the control group. Prior to participation, all individuals provided informed consent.

Demographic information (age, gender), gravidity, and parity were recorded for all participants. Height measurements were obtained without the use of headgear or footwear, using a standard height measurement scale. Weight was measured using a standard scale after removal of any accessories. Body mass index (BMI) was calculated.

After an overnight fast of 8-10 hours, venous blood samples were collected at 08:30 following a 30-minute rest period in a seated position. Serum cortisol, plasma A, and plasma NA levels were measured as stress markers at the beginning of OGTT. The samples were collected under standard conditions, for example in a quiet, temperature-controlled environment, following a rest period and without prior caffeine intake. Serum cortisol was measured using electrochemiluminescence, while plasma A and NA were assessed by radioimmunoassay. Plasma glucose levels were measured using a Roche automated biochemical analyzer.

In this trial, GDM was diagnosed using the criteria established by the International Association of Diabetes and Pregnancy Study Group (IADPSG) (15). A 75 g OGTT was performed between the 24th and 28th weeks of gestation, and GDM was defined when any of the threshold values were met or surpassed. The threshold values were defined as fasting plasma glucose (FPG) ≥92 mg/dL, 1-hour PG ≥180 mg/dL, or 2-hour PG ≥153 mg/dL. The test was administered after a fasting period of at least eight hours. Blood glucose levels were obtained before intake of the 75 g glucose solution, as well as at 60 and 120 minutes after consumption.

Following the diagnosis of GDM, depressive symptoms in participants were assessed using the BDI. Originally formulated by Aaron T. Beck in 1961 and subsequently revised in 1971, the BDI was translated into Turkish by Hisli (16) in 1988, and its validity and reliability confirmed in 1989. This inventory consists of 21 self-report items aimed at assessing depressive symptoms and associated attitudes. It employs a four-point Likert Scale, allowing respondents to choose the statement that best reflects their current state. All items are assigned a score from 0 to 3, resulting in a total score between 0 and 63. In the Turkish adaptation study, a cutoff score of 17 was

determined for identifying depression (16). Depression severity is classified into 4 categories: minimal (0-9), mild (10-16), moderate (17-29), and severe (30-63).

Statistical Analysis

Statistical analysis were performed using SPSS Statistics 20. Categorical variables were indicated as frequencies and percentages, whereas continuous variables were reported as means with standard deviations. The one-sample Kolmogorov-Smirnov test was conducted to determine the normality of the dataset. Since the data did not follow a normal distribution, the Mann-Whitney U test was used to evaluate comparisons between two groups, and the Kruskal-Wallis test was used to evaluate comparisons for multiple groups. Differences in categorical variables were assessed with the chi-square test. A p-value of less than 0.05 was accepted as statistically significant.

RESULTS

The patient and control groups demonstrated comparable characteristics regarding age and BMI (p=0.910, p=0.118, respectively). Similarly, no significant differences were found between the two groups in terms of gravidity and parity (p=0.158, p=0.464, respectively). Nevertheless, FPG, first-hour PG, and second-hour PG levels were markedly greater in the patient group than in the control group (p<0.001 for all). No statistically significant difference was found in serum cortisol levels between the groups (p=0.136). In contrast, A and NA levels were significantly higher than those of the control group (p=0.016, p=0.033; respectively). BDI scores were similar between groups (p=0.151). Additionally, no participants in the study were identified with severe depression (Table 1).

In the GDM group, OGTT values were not different among those with minimal levels and mild and moderate depression based on the BDI (p>0.05). The mean A levels of 33 pregnant women with minimal depression were 110.59±35.03 pg/ml; the mean A levels of 31 pregnant women with mild depression were 126.65±22.33 pg/mL and the mean A levels of 35 pregnant women with moderate depression were 95.09±30.86 pg/mL. This finding was statistically significant (p=0.005). Also, the difference in mean BMI between pregnant women with mild and moderate depression was statistically significant (p=0.018). The mean BMI of diabetic pregnant women with moderate depression was higher than that of others (Table 2).

DISCUSSION

In this study, the BDI was administered to pregnant women with GDM; BDI was similar between groups (p>0.05), which contrasts with previous findings in the literature. Arafa et al. (17) conducted the first study assessing the connection between depression and GDM. In their research, women with depression had a higher risk for GDM compared to those without a history of depression. Conversely, OuYang et al. (9) conducted a systematic review and concluded that additional research is needed to establish whether depression is a risk factor for GDM.

A study by Myers et al. (18) found that individuals with a variant form of the *oxytocin receptor (OXTR)* gene displayed more severe symptoms of both depression and anxiety compared to those without the variant. In a similar study, researchers examining the relationship between single nucleotide polymorphisms (SNPs) in the *OXTR* gene and psychological symptoms in Malaysian women with GDM found that certain SNPs were linked to increased stress symptoms, resulting in a 2.9-fold higher likelihood of experiencing stress (19). Another study, which aimed to evaluate the relationship between OXTR and *melatonin receptor 1B* gene SNPs and psychological symptoms in women diagnosed with GDM, found a significant difference in the frequency of gene polymorphisms in the AA and GG genotypes of OXTR rs53576 (p=0.04) (20). These findings suggest that SNPs in the *OXTR* gene could lead to depression and anxiety in pregnant women with GDM. However, there are several limitations to the studies: small sample sizes and the inclusion of

Table 1. Comparison of demographic characteristics, 75 grams oral glucose test, plasma stress hormone levels, and beck's depression inventory results between patients and control group

	Patients (n=70)	Controls (n=70)	p
Age (years)	27.51±5.64	27.23±4.72	0.910
BMI (kg/m^2)	26.48±7.83	26.73±4.46	0.118
Gravidity (n)	2.04±1.08	2.50±1.56	0.158
Parity (n)	0.87±0.96	0.74±0.87	0.464
Cortisol (µg/dL)	22.86 ± 5.19	24.74±6.74	0.136
A (pg/mL)	116±31.34	94.85±49.93	**0.016**
NA (pg/mL)	305.01±76.46	267.97±109.31	**0.033**
FPG (mg/dL)	92.86±17.11	87.37±5.28	<0.001
1-hour PG (mg/dL)	174.07±29.94	121.59±28.65	<0.001
2-hour PG (mg/dL)	152.90±28.32	102.50±21.39	<0.001
BDI			0.151
Minimal depression	33	34	
Mild depression	31	23	
Moderate depression	6	13	

BMI: Body mass index, A: Plasma adrenaline, NA: Noradrenaline, FPG: Fasting Plasma glucose, PG: plasma glucose, BDI: Beck's depression inventory

Table 2. Examination of the data between the classes formed according to the beck's depression inventory in the patient group

	Minimal depression (n=33)	Mild depression (n=31)	Moderate depression (n=6)	p
Age (years)	27.94±5.46	27.29±5.77	26.17±5.63	0.740
BMI (kg/m^2)	26.97±9.13	24.51±3.35	33.90±11.08	**0.018**
Gravidity (n)	1.94±1.07	2.16±1.08	2.00±1.15	0.655
Parity (n)	0.82±0.99	0.94±0.94	0.83±0.89	0.796
Cortisol (µg/dL)	23.09±5.50	22.61±4.17	22.90±7.58	0.947
A (pg/mL)	110.59±35.03	126.65±22.33	95.09±30.86	**0.005**
NA (pg/mL)	293.93±87.18	311.51±68.29	332.42±25.95	0.887
FPG (mg/dL)	90.79±14.44	91.35±14.92	112.00±16.82	0.172
1-hour PG (mg/dL)	170.82±32.86	121.59±28.65	180.00±39.22	0.907
2-hour PG (mg/dL)	149.91±25.81	176.39±23.52	155.00±44.76	0.648

BMI: Body mass index, A: Adrenaline, NA: Noradrenaline, FPG: Fasting plasma glucose, PG: Plasma glucose, BDI: Beck's depression inventory

only a few genetic markers due to resource constraints. In our study, genetic analyses could not be performed due to a lack of resources.

Feng et al. (21) conducted a study with 150 participants to investigate the relationship between stress hormones and GDM. This study found a slight increase in cortisol levels in the GDM group compared to the control group, although this difference was not statistically significant (p=0.09). However, the levels of A and NA were elevated in the GDM group (p=0.00, p=0.03, respectively) (21). Similarly, in our study with 140 pregnant women, serum cortisol levels did not significantly differ between the groups (p=0.136). However, A and NA were higher in the GDM group (p=0.016, p=0.033; respectively).

Interestingly, among women with GDM, those with moderate depression had the lowest mean adrenaline levels, despite having the highest BMI. One possible explanation is the physiological blunting of the adrenergic response in the context of chronic stress or depressive states. Moreover, higher BMI may influence hormonal feedback mechanisms. These complex interactions warrant further investigation.

Cortisol, A, and NA are established biological markers of stress, and their levels typically increase in response to stress (23,24). In another study, Feng et al. (24) found significantly elevated levels of A, NA, and glucagon in women with GDM, suggesting that stress could contribute to the pathophysiology of GDM. Furthermore, stress hormones are recognized as factors that promote hyperglycemia, potentially worsening insulin resistance and elevating blood glucose levels (25,26). Prolonged oxidative stress can also contribute to increased insulin resistance and disrupt glucose metabolism. A previous study indicated that enhanced oxidative stress could impair stress habituation and elevate levels of A, NA, and cortisol (27,28). Cortisol, a key stress hormone, can increase the release of glucose from the liver, impair β-cell function, and reduce insulin secretion, all of which may facilitate hyperglycemia (29,30). In our study, cortisol levels did not significantly increase in the GDM group compared to the control group, which is similar to the findings of studies by Da Costa et al. (31) and Feng et al. (24). A study found that anxiety and depression can trigger chronic hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in excessive secretion of cortisol and insulin resistance, which in turn raises the risk of GDM (32). The absence of a significant elevation in cortisol in our study may be because no participants had severe depression, with most exhibiting minimal or mild depression. This likely prevented chronic HPA axis hyperactivity and a significant rise in cortisol levels.

Our research has several limitations, such as the small sample size and evaluating maternal serum cortisol, A, and NA levels, as well as the BDI, only at the time of initial diagnosis (measured once), without follow-up data on these parameters prior to pregnancy or during later gestational weeks.

The relationship between GDM and depression remains inconclusive, and there may be other mechanisms underlying hyperglycemia in women with GDM. Although our hypothesis is to explore the effect of depression and stress hormones on the development of GDM, our findings show that cortisol levels were similar between groups. The mean levels of A and NA were higher in the GDM group. (p=0.016, p=0.033; respectively). This suggests that activation of the sympathoadrenal system, rather than the HPA axis, may have a more significant role in the etiopathogenesis of GDM. Additionally, the comparable BDI scores in both groups may imply that the influence of depression on the development of GDM in pregnant women is minimal. These findings could contribute to the interpretation of GDM in clinical practice, and further research may explore whether stress hormone levels could serve as an additional or early test in GDM diagnosis, particularly when the 75 g OGTT is inconclusive.

Study Limitations

This study has several limitations. First, its cross-sectional design limits the ability to establish causality between stress hormones and the development of GDM. Second, the sample size, although adequate for initial analysis, may not fully capture the variability in hormonal and psychological responses among a broader population. Third, the assessment of depression was based solely on the BDI, which, while validated, is a self-reported measure and may be subject to reporting bias. Additionally, other potential confounding factors, such as sleep quality, socioeconomic status, or pre-existing mental health conditions, were not evaluated. Lastly, hormone levels were measured at a single time point, which may not reflect long-term or fluctuating levels during pregnancy. Larger studies with

more participants are needed before these findings can be applied routinely.

CONCLUSION

Our study highlights that the sympathoadrenal system might play a more important role than depression in pregnant women with GDM. However, additional large-scale prospective studies are necessary to better understand the relationship between depression, stress hormones, and GDM. Early detection and intervention for GDM could help prevent the risk of maternal and fetal complications associated with the condition.

Ethics

Ethics Committee Approval: This study was approved by the Clinical Research Ethics Committee (approval number: e2-23-3189, date: 18.01.2023) and complied with the principles outlined in the Helsinki Declaration.

Informed Consent: Consent form was filled out by all participants.

Footnotes

Authorship Contributions

Surgical and Medical Practices: A.Ö., Y.O., E.A., Concept: A.Ö., Y.O., H.Y., Design: Y.O., H.D., H.Y., Data Collection or Processing: A.Ö., Analysis or Interpretation: A.Ö., E.A., H.D., Literature Search: A.Ö., E.A., E.Ü., Writing: A.Ö., E.A., E.Ü.

Conflict of Interest: No conflict of interest was declared by the authors.

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