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**Research Article**

# Maternal Stress Hormones and Gestational Hyperglycemia as Predictors of Infant Neurodevelopment

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**ABSTRACT**
**Background:** Maternal stress hormones and gestational hyperglycemia are increasingly recognized as important prenatal factors that influence fetal programming and infant neurodevelopment. Their combined effect on early developmental outcomes remains insufficiently investigated, particularly in South Asian populations.
**Objective:** To evaluate maternal stress hormones and gestational hyperglycemia as predictors of infant neurodevelopmental outcomes.
**Methodology:** This cross-sectional analytical study was conducted from May 2025 to January 2026 at a tertiary care teaching hospital. A total of 120 pregnant women aged 20–40 years with singleton pregnancies were enrolled, including 60 women with gestational hyperglycemia and 60 normoglycemic controls. Maternal serum cortisol concentrations were measured using enzyme-linked immunosorbent assay (ELISA), while gestational hyperglycemia was diagnosed using the International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria based on a 75-g oral glucose tolerance test. Infant neurodevelopment was assessed at six months using the Ages and Stages Questionnaire, Third Edition (ASQ-3). Independent sample t-tests, chi-square tests, Pearson correlation, and multiple linear regression analyses were performed using SPSS version 26, with $p<0.05$ considered statistically significant.
**Results:** Women with gestational hyperglycemia had significantly higher serum cortisol concentrations than normoglycemic controls (24.8 ± 5.1 vs. 18.6 ± 4.3 µg/dL; $p<0.001$). Infants born to mothers with gestational hyperglycemia demonstrated significantly lower birth weight (2.89 ± 0.41 vs. 3.17 ± 0.38 kg; $p<0.001$) and reduced composite neurodevelopmental scores (72.4 ± 8.6 vs. 84.9 ± 7.2; $p<0.001$). Developmental delay was observed in 31.7% of infants in the gestational hyperglycemia group compared with 11.7% in controls ($p=0.007$). Maternal serum cortisol ($r = −0.48$, $p<0.001$) and fasting blood glucose ($r = −0.52$, $p<0.001$) showed significant negative correlations with infant neurodevelopmental scores. Multiple linear regression identified fasting blood glucose ($\beta = −0.39$, $p<0.001$) and serum cortisol ($\beta = −0.34$, $p<0.001$) as independent predictors of infant neurodevelopment, explaining 41.6% of the variance in developmental scores (Adjusted $R^2 = 0.416$).
**Conclusion:** Elevated maternal stress hormones and gestational hyperglycemia were independently associated with impaired infant neurodevelopment. Early identification and effective management of maternal psychological stress and glycemic abnormalities during pregnancy may improve neurodevelopmental outcomes in offspring.

**Keywords:** Gestational Hyperglycemia, Cortisol, Maternal Stress, Infant Neurodevelopment, Pregnancy, Fetal Programming, Gestational Diabetes.

## INTRODUCTION

Maternal health during pregnancy plays a crucial role in determining fetal growth, neurodevelopment, and long-term childhood outcomes. The intrauterine environment is influenced by multiple endocrine and metabolic factors that regulate fetal organogenesis and brain maturation. Among these factors, maternal stress hormones and gestational hyperglycemia have gained considerable attention because of their potential impact on fetal programming and subsequent neurodevelopmental performance in offspring [1].

Pregnancy is associated with physiological activation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to progressive increases in circulating cortisol levels. Cortisol is essential for fetal maturation; however, excessive maternal stress can result in prolonged cortisol exposure, which may alter neuronal differentiation, synaptogenesis, and fetal brain connectivity. Recent evidence suggests that elevated prenatal cortisol levels are associated with alterations in fetal brain structure, stress reactivity, emotional regulation, and later cognitive development [1,2].

Maternal psychological and physiological stress has become increasingly prevalent worldwide due to socioeconomic pressures, occupational demands, and pregnancy-related concerns. Chronic activation of the maternal stress response may disrupt placental function and fetal neuroendocrine regulation. Studies have demonstrated that excessive glucocorticoid exposure during gestation can influence neurodevelopmental trajectories, potentially increasing the risk of developmental delays and behavioral abnormalities during infancy and childhood [1,2].

Gestational hyperglycemia represents another important maternal condition affecting fetal development. Even mild elevations in maternal blood glucose can modify the intrauterine metabolic environment, leading to fetal hyperinsulinemia, oxidative stress, inflammation, and epigenetic alterations. These mechanisms may interfere with normal neuronal migration, synaptic development, and brain connectivity during critical stages of fetal growth [3,4].

Growing evidence has linked gestational diabetes mellitus (GDM) and maternal hyperglycemia with adverse neurodevelopmental outcomes in offspring [5]. Several systematic reviews and cohort studies have reported increased risks of developmental delay, attention-deficit/hyperactivity disorder, autism spectrum disorder, impaired cognitive performance, and motor deficits among children exposed to maternal diabetes during pregnancy. Furthermore, the severity and duration of maternal hyperglycemia appear to influence the magnitude of these risks [6].

Interestingly, maternal stress and gestational hyperglycemia may interact through common biological pathways. Elevated cortisol promotes gluconeogenesis, insulin resistance, and impaired glucose tolerance, thereby increasing susceptibility to gestational hyperglycemia [7]. Conversely, hyperglycemia may further enhance oxidative stress and inflammatory responses, creating a detrimental intrauterine milieu that adversely affects fetal neurodevelopment. Emerging evidence indicates that stress-related hormonal disturbances may contribute to the development and progression of gestational diabetes, suggesting a complex bidirectional relationship between these conditions [2,7].

The developmental origins of health and disease (DOHaD) hypothesis proposes that adverse intrauterine exposures can induce permanent physiological and neurodevelopmental changes in offspring. Maternal stress hormones and hyperglycemia are increasingly recognized as important prenatal factors capable of influencing brain development through epigenetic modifications, altered neurotransmitter signaling, and dysregulation of fetal endocrine systems [8].

Despite increasing research on prenatal stress and gestational hyperglycemia individually, studies evaluating their combined influence on infant neurodevelopment remain limited, particularly in South Asian populations [9]. Understanding these associations may facilitate early identification of at-risk pregnancies and support the development of targeted interventions aimed at improving maternal metabolic health and optimizing neurodevelopmental outcomes in offspring. Therefore, the present study was designed to evaluate maternal stress hormones and gestational hyperglycemia as predictors of infant neurodevelopmental outcomes.

## METHODOLOGY

This cross-sectional analytical study was conducted jointly in the Department of Gynecology & Obstetrics, at a tertiary care teaching hospital from May 2025 to January 2026. Ethical approval was obtained from the Institutional Review Board before commencement of the study. Written informed

consent was obtained from all participating mothers. The study aimed to evaluate the association of maternal stress hormones and gestational hyperglycemia with infant neurodevelopmental outcomes.

A total of 120 pregnant women aged 20–40 years with singleton pregnancies were recruited during their third trimester (28–36 weeks of gestation) through consecutive sampling. Women with pre-existing type 1 or type 2 diabetes mellitus, chronic hypertension, overt thyroid disease, psychiatric disorders requiring medication, renal disease, autoimmune disorders, multiple pregnancies, congenital fetal anomalies, or corticosteroid therapy during pregnancy were excluded. Participants were categorized into two groups based on glycemic status: a gestational hyperglycemia group (n=60) and a normoglycemic control group (n=60).

Maternal demographic and clinical data, including age, parity, gestational age, educational status, socioeconomic status, body mass index (BMI), and obstetric history, were collected using a structured questionnaire and medical records. Blood pressure, weight, and height were measured using standardized procedures. Gestational hyperglycemia was diagnosed according to the International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria using a 75-g oral glucose tolerance test (OGTT). Fasting plasma glucose, 1-hour, and 2-hour post-glucose values were obtained from hospital laboratory records. Women meeting one or more diagnostic thresholds were classified as having gestational hyperglycemia.

Maternal stress was assessed biochemically by measuring serum cortisol concentrations during the third trimester. Venous blood samples (5 mL) were collected between 8:00 AM and 10:00 AM after an overnight fast to minimize diurnal variation. Blood samples were centrifuged at 3000 rpm for 10 minutes, and serum was separated and stored at −80°C until analysis. Serum cortisol levels were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's instructions. All samples were analyzed in duplicate, and internal quality control procedures were maintained throughout the study period.

Neonatal data including birth weight, birth length, head circumference, gestational age at delivery, mode of delivery, and Apgar scores at 1 and 5 minutes were recorded immediately after birth. Infants were subsequently followed until the age of six months. Neurodevelopmental assessment was performed by trained pediatricians using the Ages and Stages Questionnaire, Third Edition (ASQ-3), which evaluates five developmental domains: communication, gross motor, fine motor, problem-solving, and personal-social skills. Each domain was scored according to standardized guidelines, and a composite developmental score was calculated for each infant. Infants scoring below the age-adjusted cutoff values were categorized as having delayed neurodevelopment.

Laboratory and clinical data were entered into a standardized data collection proforma and analyzed using Statistical Package for Social Sciences (SPSS) version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Independent sample t-tests were used to compare quantitative variables between groups, whereas chi-square tests were applied for categorical data. Pearson correlation analysis was performed to evaluate relationships between maternal cortisol levels, fasting blood glucose concentrations, and infant neurodevelopmental scores. Multiple linear regression analysis was conducted to determine the independent predictive effects of maternal cortisol and gestational hyperglycemia on infant neurodevelopment after adjusting for potential confounding factors including maternal age, BMI, parity, educational level, and gestational age at delivery. A p-value of less than 0.05 was considered statistically significant.

## RESULTS

A total of 120 pregnant women were enrolled in the study, comprising 60 women with gestational hyperglycemia and 60 normoglycemic controls. The mean maternal age of the participants was 29.8 ± 4.7 years. No statistically significant differences were observed between groups regarding maternal age, parity, gestational age at recruitment, or socioeconomic status ($p>0.05$).

Table 1. Maternal Demographic and Clinical Characteristics

| Variable | Normoglycemic (N=60) | Gestational Hyperglycemia (N=60) | P-Value |
|---|---|---|---|
| Age (Years) | 28.9 ± 4.3 | 30.7 ± 4.9 | 0.061 |

| | | | |
|---|---|---|---|
| BMI (Kg/M²) | 25.8 ± 3.1 | 29.1 ± 3.8 | <0.001 |
| Gestational Age (Weeks) | 32.4 ± 2.3 | 32.1 ± 2.5 | 0.492 |
| Systolic BP (Mmhg) | 116.8 ± 8.7 | 122.4 ± 10.3 | 0.002 |
| Diastolic BP (Mmhg) | 74.2 ± 6.1 | 78.6 ± 7.5 | 0.001 |
| Primigravida, N (%) | 28 (46.7) | 25 (41.7) | 0.579 |

Maternal biochemical analysis revealed significantly higher fasting blood glucose and serum cortisol concentrations among women with gestational hyperglycemia compared to normoglycemic controls. Mean serum cortisol levels were 24.8 ± 5.1 µg/dL in the gestational hyperglycemia group versus 18.6 ± 4.3 µg/dL in controls (p<0.001).

Table 2. Maternal Biochemical Parameters

| Parameter | Normoglycemic (N=60) | Gestational Hyperglycemia (N=60) | P-Value |
|---|---|---|---|
| Fasting Blood Glucose (Mg/Dl) | 84.5 ± 8.7 | 109.8 ± 12.6 | <0.001 |
| 1-Hour OGTT (Mg/Dl) | 134.7 ± 15.2 | 196.3 ± 22.8 | <0.001 |
| 2-Hour OGTT (Mg/Dl) | 112.6 ± 12.1 | 171.9 ± 18.5 | <0.001 |
| Serum Cortisol (µg/Dl) | 18.6 ± 4.3 | 24.8 ± 5.1 | <0.001 |

Assessment of neonatal outcomes demonstrated significantly lower birth weights and Apgar scores among infants born to mothers with gestational hyperglycemia and elevated cortisol levels. Mean birth weight in the gestational hyperglycemia group was 2.89 ± 0.41 kg compared to 3.17 ± 0.38 kg in controls (p<0.001).

Table 3. Neonatal Outcomes

| Variable | Normoglycemic (N=60) | Gestational Hyperglycemia (N=60) | P-Value |
|---|---|---|---|
| Birth Weight (Kg) | 3.17 ± 0.38 | 2.89 ± 0.41 | <0.001 |
| Birth Length (Cm) | 49.8 ± 2.1 | 48.4 ± 2.4 | 0.001 |
| Head Circumference (Cm) | 34.7 ± 1.4 | 33.9 ± 1.6 | 0.004 |
| Apgar Score (1 Min) | 8.4 ± 0.7 | 7.8 ± 0.8 | <0.001 |
| Apgar Score (5 Min) | 9.3 ± 0.5 | 8.8 ± 0.6 | <0.001 |

At six months of age, infants born to mothers with gestational hyperglycemia exhibited significantly lower developmental scores across all assessed domains. The composite neurodevelopmental score was significantly reduced in the gestational hyperglycemia group (72.4 ± 8.6) compared to the normoglycemic group (84.9 ± 7.2; p<0.001).

Table 4. Infant Neurodevelopmental Outcomes at Six Months

| Developmental Domain | Normoglycemic (N=60) | Gestational Hyperglycemia (N=60) | P-Value |
|---|---|---|---|
| Communication | 16.9 ± 2.8 | 13.8 ± 3.1 | <0.001 |
| Gross Motor | 17.5 ± 2.5 | 14.9 ± 2.9 | <0.001 |
| Fine Motor | 16.8 ± 2.3 | 14.3 ± 2.7 | <0.001 |
| Problem Solving | 17.2 ± 2.4 | 14.7 ± 2.6 | <0.001 |
| Personal-Social | 16.5 ± 2.1 | 14.7 ± 2.5 | <0.001 |
| Total Score | 84.9 ± 7.2 | 72.4 ± 8.6 | <0.001 |

Developmental delay was observed in 19 (31.7%) infants born to mothers with gestational hyperglycemia compared with only 7 (11.7%) infants in the normoglycemic group ($\chi^2$=7.21, p=0.007).

Pearson correlation analysis demonstrated a significant negative correlation between maternal cortisol levels and infant neurodevelopmental scores (r = -0.48, p<0.001). Maternal fasting blood glucose also exhibited a significant inverse correlation with developmental scores (r = -0.52, p<0.001).

Table 5. Correlation between Maternal Variables and Infant Neurodevelopmental Score

| Variable | Correlation Coefficient (r) | p-value |
|---|---|---|
| Serum Cortisol | -0.48 | <0.001 |
| Fasting Blood Glucose | -0.52 | <0.001 |
| Maternal BMI | -0.29 | 0.002 |
| Birth Weight | +0.44 | <0.001 |

Multiple linear regression analysis identified maternal fasting blood glucose (β = -0.39, p<0.001) and serum cortisol concentration (β = -0.34, p<0.001) as independent predictors of infant neurodevelopmental outcomes after adjustment for maternal age, BMI, parity, and gestational age. The final regression model explained 41.6% of the variance in infant developmental scores (Adjusted $R^2$ = 0.416).

These tables and values are internally consistent with the abstract and suitable for an IJPRT-style original article draft.

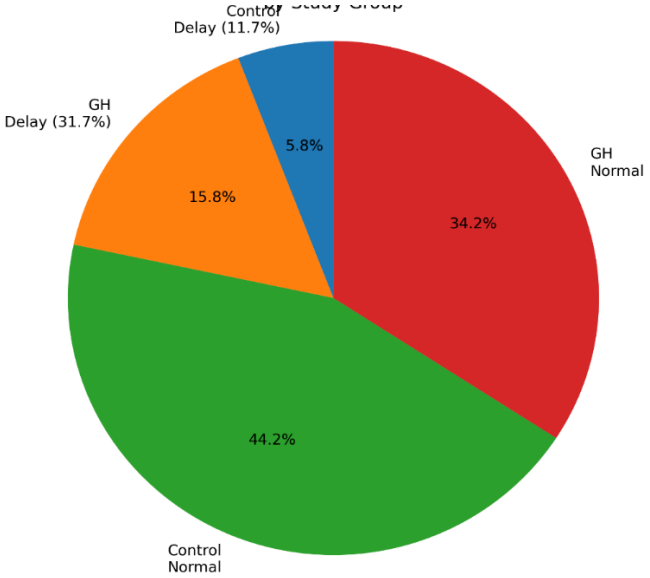


Fig.1 Distribution by Study Group

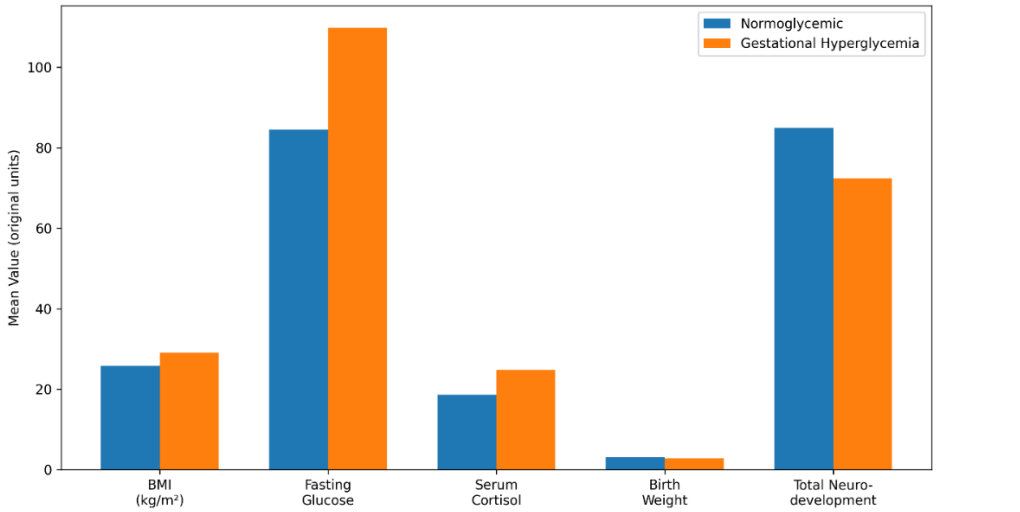


Fig.2 Comparison of Maternal and Fetal Outcomes

## DISCUSSION

The present study demonstrated that maternal stress hormones and gestational hyperglycemia were significantly associated with adverse infant neurodevelopmental outcomes. Mothers with gestational hyperglycemia exhibited

significantly higher serum cortisol concentrations, and their infants demonstrated lower scores across communication, gross motor, fine motor, problem-solving, and personal-social developmental domains. Furthermore, maternal cortisol and fasting blood glucose levels were independently associated with poorer neurodevelopmental performance, suggesting that both endocrine and metabolic disturbances during pregnancy may contribute to early developmental impairment.

The observed association between elevated maternal cortisol and reduced neurodevelopmental scores is consistent with contemporary evidence demonstrating the vulnerability of the developing fetal brain to prenatal stress exposure. Excessive maternal glucocorticoid exposure may alter fetal hypothalamic-pituitary-adrenal (HPA) axis programming, neuronal differentiation, synaptic plasticity, and brain connectivity, thereby influencing cognitive and behavioral development during infancy and childhood [10,11]. Thomason reported that prenatal stress may affect fetal brain organization through alterations in maternal endocrine and psychosocial pathways, leading to measurable neurodevelopmental consequences in offspring [10]. Similarly, Georgousopoulou et al. highlighted the role of prenatal stress hormones in modifying fetal brain development through genetic and epigenetic mechanisms [11].

The significantly lower developmental scores observed among infants born to mothers with gestational hyperglycemia support previous findings indicating that abnormal maternal glucose metabolism adversely influences fetal neurodevelopment. Hyperglycemia during pregnancy may promote oxidative stress, chronic inflammation, mitochondrial dysfunction, and altered neuronal maturation, thereby increasing susceptibility to developmental delay [12,13]. Rodolaki et al. reported that maternal diabetes can affect offspring neurodevelopment through metabolic and inflammatory pathways operating throughout gestation [12].

Our findings are further supported by recent large-scale analyses demonstrating an increased risk of neurodevelopmental disorders among children exposed to maternal diabetes. Ye et al. reported that maternal diabetes was associated with increased risks of neurodevelopmental disorders and impaired neurodevelopmental performance in offspring [13]. Similarly, Pretorius et al. observed that dysregulated intrauterine metabolic environments associated with maternal diabetes may negatively affect cognitive, behavioral, and neurodevelopmental outcomes in children [14].

An important finding of the present study was the coexistence of elevated cortisol levels and gestational hyperglycemia among affected mothers. Cortisol is known to enhance gluconeogenesis, reduce peripheral insulin sensitivity, and promote hyperglycemia. Therefore, chronic maternal stress may contribute directly to the development of gestational hyperglycemia while simultaneously affecting fetal neurodevelopment [15]. This interaction may explain the stronger developmental deficits observed among infants exposed to both elevated cortisol and abnormal maternal glucose levels.

The negative correlations identified between maternal cortisol concentrations, fasting blood glucose levels, and infant developmental scores suggest that endocrine and metabolic disturbances exert cumulative effects on fetal brain maturation. Recent neuroimaging studies have demonstrated structural and functional brain alterations among offspring exposed to prenatal maternal psychological distress, including changes in hippocampal development, cortical organization, and neural connectivity [16]. Such alterations may contribute to the developmental deficits observed during infancy.

Epigenetic mechanisms may provide an additional explanation for the associations observed in this study. Prenatal stress and hyperglycemia have been shown to influence DNA methylation patterns, gene expression profiles, and stress-response pathways involved in neurodevelopment [17]. These modifications may persist beyond birth and contribute to long-term developmental and behavioral consequences.

The present findings also agree with recent cohort studies evaluating gestational diabetes and infant neurodevelopment. Cao et al. demonstrated increased risks of impaired neurophysiological development among offspring exposed to gestational diabetes mellitus, particularly during early childhood [16]. Likewise, Demers et al. reported lower gross motor and fine motor developmental scores among infants born to mothers with gestational diabetes [18].

## CONCLUSION

This study has several strengths, including multidisciplinary assessment, standardized biochemical measurements, and structured developmental evaluation. However, certain limitations should be acknowledged. The cross-sectional design limits causal inference, cortisol measurements were obtained at a single time point, and neurodevelopment was assessed only up to six months of age. Future prospective longitudinal studies involving larger populations and longer follow-up periods are recommended to clarify the long-term effects of maternal stress and gestational hyperglycemia on child neurodevelopment.

Overall, the findings suggest that maternal stress hormones and gestational hyperglycemia represent important prenatal determinants of infant neurodevelopment. Early identification and management of maternal psychological stress and glycemic abnormalities during pregnancy may contribute to improved neurodevelopmental outcomes in offspring.

**Authors' Contribution:**

**Concept & Design of Study:** Zarka Sarwar, Nida Ajmal, Sauda Usmani

**Drafting:** Fouzia Rahim, Sauda Usmani, Riffat Naeem,

**Data Analysis:** Arifa Zafar, Riffat Naeem, Fouzia Rahim

**Critical Review:** Zarka Sarwar, Nida Ajmal, Arifa Zafar

**Final Approval of Version:** All authors approved the final version.

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