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# Inflammatory and Cellular Stress Signatures in Gestational Diabetes Mellitus: An Observational Study

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**ABSTRACT:**

**Background:** Gestational diabetes mellitus (GDM) is associated with metabolic dysfunction accompanied by alterations in inflammatory and cellular stress pathways. This study evaluated inflammatory and cellular stress markers in women with GDM.

**Methods:** This observational study included 60 pregnant women, comprising 30 healthy pregnant controls and 30 women with GDM. Glycaemic parameters were assessed using standard biochemical methods. IL-1, IL-4, ATF-4, and NF-κB were measured using sandwich ELISA. Appropriate statistical tests and Spearman's correlation were applied.

**Results:** Women with GDM had significantly higher fasting glucose, postprandial glucose, and HbA1c levels than healthy pregnant controls (all $p<0.001$). IL-1 was significantly higher in GDM ($8.59 \pm 0.40$ vs $5.21 \pm 0.45$; $p<0.001$), while IL-4 was significantly lower ($2.50 \pm 0.45$ vs $4.08 \pm 0.34$; $p<0.001$). ATF-4 ($2.76 \pm 0.20$ vs $1.47 \pm 0.18$; $p<0.001$) and NF-κB ($5.15 \pm 0.32$ vs $2.48 \pm 0.29$; $p<0.001$) were also markedly elevated in GDM. No significant correlations were observed between HbA1c and the inflammatory or cellular stress markers.

**Conclusion:** GDM is associated with a distinct inflammatory and cellular stress profile characterized by increased IL-1, ATF-4, and NF-κB and reduced IL-4, suggesting dysregulation of these pathways in GDM.

## INTRODUCTION

Gestational diabetes mellitus (GDM) is a common metabolic complication of pregnancy, characterized by hyperglycaemia first detected during gestation that does not meet the criteria for overt diabetes mellitus [1]. It develops when pancreatic β-cells fail to adequately compensate for the progressive physiological insulin resistance of pregnancy. Its pathogenesis involves complex interactions among metabolic adaptations, placental hormones, genetic susceptibility, inflammatory responses, and cellular stress pathways.[2] GDM is associated with maternal complications such as preeclampsia and operative delivery and adverse neonatal outcomes including macrosomia, hypoglycaemia, and respiratory complications.[3] Women with GDM also have an increased long-term risk of developing type 2 diabetes mellitus.[4]During normal pregnancy, insulin sensitivity progressively decreases, particularly during the second and third trimesters, and is compensated by increased pancreatic insulin secretion. In GDM, inadequate β-cell compensation results in persistent hyperglycaemia and metabolic dysfunction. Increasing evidence suggests that inflammatory activation and cellular stress responses contribute to impaired insulin signalling, β-cell dysfunction, placental abnormalities, and altered maternal metabolic adaptation [2,5]. Inflammation is an important component of GDM pathophysiology. Hyperglycaemia and metabolic disturbances can stimulate inflammatory signalling and alter cytokine

regulation. Interleukin-1 (IL-1) is a pro-inflammatory cytokine involved in immune activation and has been associated with β-cell dysfunction, impaired insulin secretion, and insulin resistance. In contrast, interleukin-4 (IL-4) has anti-inflammatory and immunoregulatory properties and contributes to immune homeostasis. Altered cytokine profiles have been reported in women with GDM, suggesting their potential involvement in disease-associated inflammatory responses [6,7]. Adipokines and other inflammatory mediators may further contribute to the interaction between maternal metabolic dysfunction and inflammatory pathways during pregnancy. Cellular stress represents another important component of GDM pathophysiology. Persistent hyperglycaemia can disturb intracellular metabolic and protein-processing processes, activating adaptive stress-response pathways. Although these pathways initially promote cellular adaptation and restoration of homeostasis, persistent activation may result in cellular dysfunction, impaired insulin signalling, and abnormal cell survival [5,8,9]. Activating transcription factor-4 (ATF-4) is an important regulator of the integrated cellular stress response. Activation of the PERK–eIF2α pathway promotes ATF-4 signalling, which regulates genes involved in cellular adaptation, protein homeostasis, metabolism, and apoptosis. Sustained activation may contribute to cellular dysfunction under prolonged metabolic stress [9,10]. Nuclear factor kappa B (NF-ϰB) is a key transcription factor linking cellular stress and inflammatory responses. It regulates genes involved in inflammatory cytokine production, immune activation, and cellular survival. Cellular stress-related signalling can interact with NF-ϰB pathways, thereby amplifying inflammatory activity and metabolic abnormalities in GDM. The CHOP–PPARα–NF-ϰB pathway provides further evidence of an interaction between cellular stress and inflammatory signalling in GDM [11,12]. Despite increasing evidence regarding inflammatory and cellular stress mechanisms in GDM, their combined characterization remains limited. Therefore, the present observational study aimed to compare inflammatory markers (IL-1 and IL-4) and cellular stress markers (ATF-4 and NF-ϰB) between women with GDM and healthy pregnant controls and evaluate their associations with relevant glycaemic and metabolic parameters.

## MATERIALS AND METHODS

This hospital-based observational study was conducted over a period of 24 months in the Department of Biochemistry, Faculty of Medicine and Health Sciences (FMHS), SGT University, Gurugram. The study was designed to compare inflammatory and cellular stress-related markers between women with gestational diabetes mellitus (GDM) and healthy pregnant controls.

### Study Population and Sample Size

The present analysis included 60 pregnant women aged 20–40 years, selected from the original study population of 120 participants. The participants were categorized into two groups. Group I comprised 30 healthy pregnant women without diabetes or anaemia, whereas Group II comprised 30 pregnant women diagnosed with GDM according to the applicable American Diabetes Association (ADA) diagnostic criteria.

### Eligibility Criteria

Pregnant women aged 20–40 years with either a normal pregnancy or confirmed GDM who provided written informed consent were eligible for inclusion. Women with acute or chronic inflammatory disorders, hepatic or renal disease, a history of drug or alcohol intoxication, or those receiving corticosteroids, hypolipidaemic agents, or hormone replacement therapy were excluded from the study.

### Sample Collection and Biochemical Assessment

After an overnight fast of 12–14 hours, approximately 5 mL of venous blood was collected from each participant under aseptic precautions. Serum was separated by centrifugation at 3500 rpm for 15 minutes and stored under appropriate conditions until analysis. Routine biochemical parameters, including fasting plasma glucose, haemoglobin, serum proteins, and lipid profile, were assessed. Glucose and lipid parameters were measured using an ERBA XL 360 automated biochemical analyser according to standard laboratory procedures.

### Assessment of Inflammatory Markers

The inflammatory cytokines interleukin-1 beta (IL-1β) and interleukin-4 (IL-4) were measured using commercially available sandwich enzyme-linked immunosorbent assay (ELISA) kits (ELK Biotechnology) in accordance with the manufacturer's instructions. Absorbance was measured at 450 nm using an ELISA microplate reader, and concentrations were determined from the corresponding standard calibration curves.

### Assessment of Cellular Stress Markers

Cellular stress-related proteins, including activating transcription factor-4 (ATF-4) and nuclear factor kappa B p65 (NF-κB p65), were quantified using commercially available sandwich ELISA kits according to the manufacturer's protocol. Optical density was measured at 450 nm, and the concentrations of the respective biomarkers were calculated using standard curves generated from known concentrations of the analytes.

### Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation for normally distributed data and as median with interquartile range for non-normally distributed data. The normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between the GDM and healthy pregnancy groups were performed using the independent-samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Spearman's rank correlation coefficient (ϱ) was used to evaluate associations between glycaemic parameters and inflammatory and cellular stress markers. All statistical tests were two-sided, and a p-value $<0.05$ was considered statistically significant.

### Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee of the Faculty of Medicine and Health Sciences, SGT University, Gurugram. Written informed consent was obtained from all participants before enrolment. Participant confidentiality and privacy were maintained throughout the study in accordance with institutional ethical requirements.

## RESULTS

The present study included 60 pregnant women, comprising 30 healthy pregnant women and 30 women diagnosed with gestational diabetes mellitus (GDM). The two groups were compared with respect to anthropometric characteristics, baseline biochemical parameters, glycaemic profile, lipid profile, inflammatory markers, and cellular stress markers. Correlation analysis was performed within the GDM group to evaluate associations between glycaemic status, inflammatory markers, and cellular stress markers. The mean body weight was significantly higher in women with GDM compared with healthy pregnant women (69.14 ± 6.49 kg vs. 59.21 ± 6.21 kg; $p<0.001$). However, mean height was comparable between the two groups (155.12 ± 4.67 cm vs. 155.08 ± 5.07 cm; $p=1.00$). Similarly, no statistically significant differences were observed in haemoglobin levels (11.25 ± 0.82 vs. 11.45 ± 0.65; $p=0.79$) or median total protein levels (6.80 vs. 6.80; $p=1.00$) (Table 1). Women with GDM demonstrated significantly higher glycaemic parameters compared with healthy pregnant women. Mean fasting blood glucose (117.65 ± 7.43 vs. 81.12 ± 8.15; $p<0.001$) and postprandial blood glucose (163.20 ± 11.03 vs. 108.72 ± 10.63; $p<0.001$) were significantly elevated in the GDM group. Furthermore, median HbA1c was significantly higher among women with GDM (6.88% vs. 4.81%; $p<0.001$), indicating significantly greater glycaemic levels in this group (Table 2). Significant alterations in lipid parameters were observed among women with GDM compared with healthy pregnant women. Median total cholesterol was significantly higher in the GDM group (249.12 vs. 213.01; $p<0.001$). Similarly, mean triglycerides (244.58 ± 17.95 vs. 176.65 ± 13.21), LDL-C (159.94 ± 13.24 vs. 119.07 ± 11.23), and VLDL-C (48.92 ± 3.59 vs. 35.32 ± 2.64) were significantly elevated in women with GDM (all $p<0.001$). Conversely, mean HDL-C (41.13 ± 4.80 vs. 55.18 ± 5.77) and HDL/LDL ratio (0.26 ± 0.04 vs. 0.47 ± 0.07) were significantly lower in the GDM group (both $p<0.001$). These findings demonstrated a significantly altered lipid profile among women with GDM (Table 3 and Figure 1).

Significant differences in inflammatory markers were observed between the two groups. Mean IL-1 levels were significantly higher among women with GDM compared with healthy pregnant women (8.59 ± 0.40 vs. 5.21 ± 0.45; $p<0.001$). In contrast, mean IL-4 levels were significantly lower in the GDM group (2.50 ± 0.45 vs. 4.08 ± 0.34; $p<0.001$). These findings indicated increased IL-1 and reduced IL-4 levels among women with GDM (Table 4). Cellular stress markers were significantly elevated among women with GDM compared with healthy pregnant women. Mean ATF-4 levels were significantly higher in the GDM group (2.76 ± 0.20 vs. 1.47 ± 0.18; $p<0.001$). Similarly, mean NF-κB levels were significantly increased among women with GDM (5.15 ± 0.32 vs. 2.48 ± 0.29; $p<0.001$). These findings demonstrated significantly increased expression of cellular stress-associated markers in women with GDM (Table 5 and Figure 2). Spearman's rank correlation analysis was performed among women with GDM (n=30) to assess associations between HbA1c, inflammatory markers, and cellular stress markers. No statistically significant correlations were observed between HbA1c and IL-1 ($\varrho=0.032$; $p=0.865$), IL-4 ($\varrho=0.071$; $p=0.711$),

ATF-4 (ϱ=0.163; p=0.389), or NF-ϰB (ϱ=−0.150; p=0.428).Similarly, IL-1 showed weak positive correlations with ATF-4 (ϱ=0.299; p=0.108) and NF-ϰB (ϱ=0.160; p=0.397), neither of which was statistically significant. IL-4 also demonstrated nonsignificant correlations with ATF-4 (ϱ=0.049; p=0.797) and NF-ϰB (ϱ=−0.190; p=0.316). Furthermore, the correlation between ATF-4 and NF-ϰB was weak and statistically nonsignificant (ϱ=0.147; p=0.437) (Table 6 and Figure 3).

**Table 1.** Comparison of Anthropometric and Baseline Biochemical Characteristics Between Healthy Pregnant Women and Women With GDM

| Parameter | Healthy pregnancy (n=30) | GDM (n=30) | p-value | Statistical test |
|---|---|---|---|---|
| Height (cm) | 155.08 ± 5.07 | 155.12 ± 4.67 | 1.00 | Independent-samples t-test |
| Weight (kg) | 59.21 ± 6.21 | 69.14 ± 6.49 | <0.001 | Independent-samples t-test |
| Haemoglobin | 11.45 ± 0.65 | 11.25 ± 0.82 | 0.79 | Independent-samples t-test |
| Total protein | 6.80 (6.58–7.01) | 6.80 (6.62–7.00) | 1.00 | Mann–Whitney U test |

**Table 2.** Comparison of Glycaemic Parameters Between Healthy Pregnant Women and Women With GDM

| Parameter | Healthy pregnancy (n=30) | GDM (n=30) | p-value | Statistical test |
|---|---|---|---|---|
| Fasting blood glucose | 81.12 ± 8.15 | 117.65 ± 7.43 | <0.001 | Independent-samples t-test |
| Postprandial blood glucose | 108.72 ± 10.63 | 163.20 ± 11.03 | <0.001 | Independent-samples t-test |
| HbA1c (%) | 4.81 (4.61–4.99) | 6.88 (6.71–6.99) | <0.001 | Mann–Whitney U test |

**Table 3.** Comparison of Lipid Profile Between Healthy Pregnant Women and Women With GDM

| Parameter | Healthy pregnancy (n=30) | GDM (n=30) | p-value | Statistical test |
|---|---|---|---|---|
| Total cholesterol | 213.01 (210.51–218.35) | 249.12 (234.84–258.40) | <0.001 | Mann–Whitney U test |
| Triglycerides | 176.65 ± 13.21 | 244.58 ± 17.95 | <0.001 | Independent-samples t-test |
| HDL-C | 55.18 ± 5.77 | 41.13 ± 4.80 | <0.001 | Independent-samples t-test |
| LDL-C | 119.07 ± 11.23 | 159.94 ± 13.24 | <0.001 | Independent-samples t-test |
| VLDL-C | 35.32 ± 2.64 | 48.92 ± 3.59 | <0.001 | Independent-samples t-test |
| HDL/LDL ratio | 0.47 ± 0.07 | 0.26 ± 0.04 | <0.001 | Independent-samples t-test |

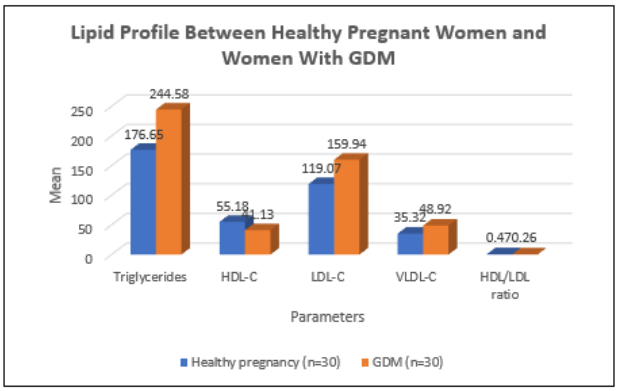


**Figure 1.** Comparison of Lipid Profile Between Healthy Pregnant Women and Women With GDM

**Table 4.** Inflammatory Markers

| Marker | Healthy pregnancy (n=30) | GDM (n=30) | p-value | Statistical test |
|---|---|---|---|---|
| IL-1 | 5.21 ± 0.45 | 8.59 ± 0.40 | <0.001 | Independent-samples t-test |
| IL-4 | 4.08 ± 0.34 | 2.50 ± 0.45 | <0.001 | |

**Table 5.** Cellular Stress Markers

| Marker | Healthy pregnancy (n=30) | GDM (n=30) | p-value | Statistical test |
|---|---|---|---|---|
| ATF-4 | 1.47 ± 0.18 | 2.76 ± 0.20 | <0.001 | Independent-samples t-test |
| NF-κB | 2.48 ± 0.29 | 5.15 ± 0.32 | <0.001 | |

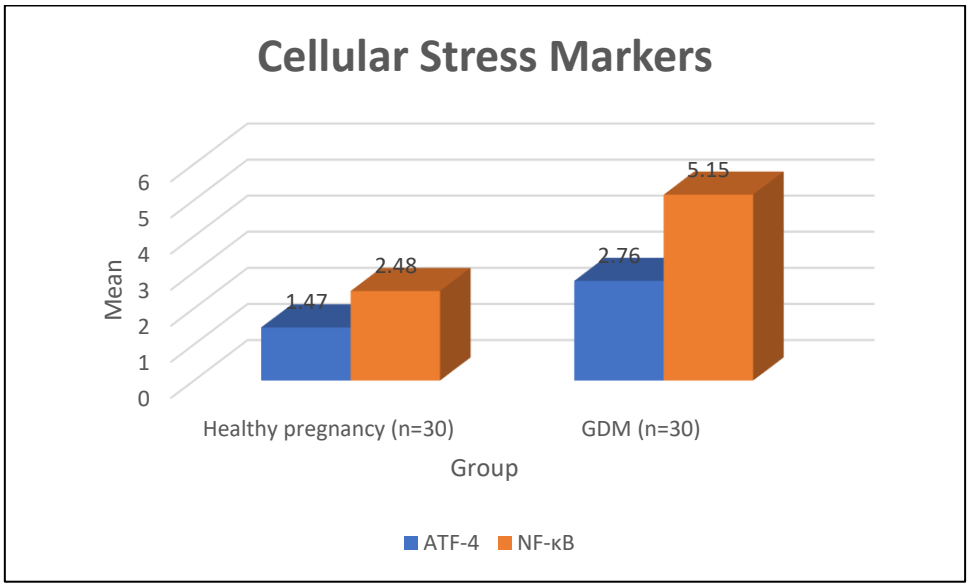


**Figure 2.** Cellular Stress Markers

**Table 6.** Correlation Analysis Within GDM Group

| Parameters correlated | Spearman's ϱ | p-value | Statistical test |
|---|---|---|---|
| HbA1c vs IL-1 | 0.032 | 0.865 | Spearman's rank correlation |
| HbA1c vs IL-4 | 0.071 | 0.711 | |
| HbA1c vs ATF-4 | 0.163 | 0.389 | |
| HbA1c vs NF-κB | −0.150 | 0.428 | |
| IL-1 vs ATF-4 | 0.299 | 0.108 | |
| IL-1 vs NF-κB | 0.160 | 0.397 | |
| IL-4 vs ATF-4 | 0.049 | 0.797 | |
| IL-4 vs NF-κB | −0.190 | 0.316 | |
| ATF-4 vs NF-κB | 0.147 | 0.437 | |

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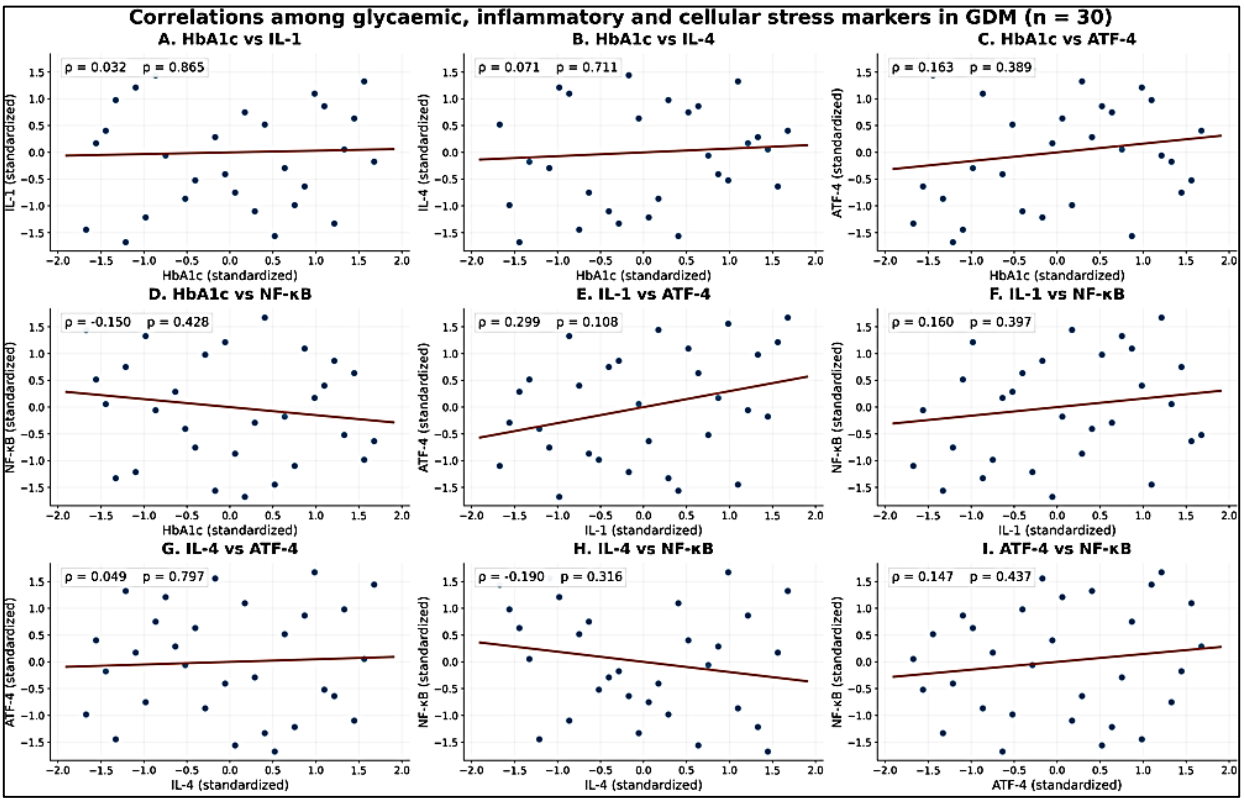


**Figure 3.** Correlation Analysis Within GDM Group

## DISCUSSION

In the present study, women with GDM had significantly higher body weight than healthy pregnant women (69.14 ± 6.49 vs 59.21 ± 6.21 kg; p<0.001), while height and haemoglobin were comparable. Glycaemic parameters were markedly higher in GDM, including fasting glucose (117.65 ± 7.43 vs 81.12 ± 8.15 mg/dL), postprandial glucose (163.20 ± 11.03 vs 108.72 ± 10.63 mg/dL), and HbA1c [6.88 (6.71–6.99) vs 4.81 (4.61–4.99)%], all with p<0.001.Brown et al. [13] reviewed 15 randomized trials involving 4501 women with GDM and reported that lifestyle interventions improved glycaemic control and reduced postprandial glucose, with a mean reduction of approximately 27.1 mg/dL in pooled analyses. Ryu et al. [14] also showed that oral hypoglycaemic agents were effective in many women with GDM; in reviewed studies, approximately 82–88% achieved glycaemic control with glyburide or insulin, while 10–46% of women receiving metformin required supplemental insulin. These findings highlight the clinical importance of the substantial glycaemic abnormalities observed in our GDM group. The major inflammatory finding of the present study was significantly higher IL-1 in GDM (8.59 ± 0.40 vs 5.21 ± 0.45; p<0.001) and significantly lower IL-4 (2.50 ± 0.45 vs 4.08 ± 0.34; p<0.001), indicating a shift toward a pro-inflammatory state. Ozler et al. [15] similarly demonstrated enhanced inflammatory activity in GDM. In their study of 58 women with GDM and 30 healthy pregnant controls, elevated TNF-α was independently associated with GDM (OR 11.98; 95% CI 2.44–58.75; p=0.002) and also predicted the need for insulin therapy (OR 18.62; p=0.006). Although they measured TNF-α rather than IL-1 or IL-4, their findings support the inflammatory imbalance demonstrated in our study. Lappas et al. [16] also reported altered inflammatory responsiveness in GDM placental tissue. They found that 13 of 16 cytokines increased significantly after stimulation in normal placenta, compared with only 3 of 16 cytokines in GDM placenta. This suggests dysregulated inflammatory signalling in GDM and is consistent with our finding of increased IL-1 accompanied by reduced IL-4. Cellular stress-related markers were markedly elevated in women with GDM. Mean ATF-4 was significantly higher in the GDM group (2.76 ± 0.20 vs 1.47 ± 0.18; p<0.001), while NF-ϰB was also substantially elevated (5.15 ± 0.32 vs 2.48 ± 0.29; p<0.001). Lappas et al. [16] demonstrated altered stress-responsive and inflammatory signalling in GDM placental tissue, supporting the presence of abnormal cellular adaptive mechanisms in diabetic pregnancy. Although their study did not directly measure ATF-4 or NF-ϰB, their findings support the

concept of disturbed cellular stress responses in GDM. The pronounced elevations of ATF-4 and NF-κB in our study extend these observations by demonstrating measurable systemic alterations in two important cellular stress-related regulatory pathways. Within the GDM group, HbA1c was not significantly correlated with IL-1 ($\varrho$=0.032, p=0.865), IL-4 ($\varrho$=0.071, p=0.711), ATF-4 ($\varrho$=0.163, p=0.389) or NF-κB ($\varrho$=−0.150, p=0.428). Similarly, IL-1 was not significantly correlated with ATF-4 or NF-κB. These findings suggest that inflammatory and cellular stress abnormalities in GDM may not directly parallel the degree of glycaemic elevation. This is supported by Ozler et al. [15], who demonstrated that inflammatory TNF-α independently predicted GDM and treatment requirement, suggesting that inflammatory markers may provide biological information beyond conventional glycaemic indices.

## CONCLUSION

The present study demonstrates that GDM is associated with a distinct inflammatory and cellular stress profile, characterized by significantly higher IL-1, ATF-4, and NF-κB levels and lower IL-4 levels compared with healthy pregnancy. These findings suggest that dysregulated inflammatory and cellular stress pathways may contribute to the pathophysiology of GDM. Such markers may have potential value in improving biological characterization of GDM beyond conventional glycaemic parameters.

## LIMITATIONS

The study was limited by its small sample size and single-centre observational design, which restricts generalizability and prevents assessment of causality. In addition, only selected inflammatory and cellular stress markers were evaluated, without longitudinal follow-up.

## DECLARATION

- **Consent for Publication**: Not applicable.
- **Availability of Data and Materials:** The datasets generated and analysed during the study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.
- **Competing Interests:** The authors declare no competing interests.•
- **Funding:** No external funding was received for this study.
- **Authors' Contributions:** S.S.: Conceptualization, methodology, investigation, sample collection, laboratory analysis, data analysis, and manuscript drafting. S.K.B.: Research supervision, study design, methodological guidance, interpretation of findings, critical manuscript revision, and final review. Both authors reviewed and approved the final manuscript and agreed to be accountable for their contributions.
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