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Biochemical and Lipid Profile Alterations in Gestational Diabetes Mellitus: A Case-Control Study

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KEYWORDS

Gestational diabetes mellitus. Lipid profile. Dyslipidemia.

ABSTRACT:

Background: Gestational diabetes mellitus (GDM) is the most common metabolic disorder in pregnancy and is associated with maternal and neonatal complications. While hyperglycemia is the hallmark of GDM, concurrent lipid alterations may play an important role in disease progression and outcomes.

Aim: To assess biochemical and lipid profile alterations in women with GDM compared to healthy pregnant controls.

Methods: A case-control study was conducted in the Department of Biochemistry, MGM Medical College and Hospital, Aurangabad. Seventy-seven pregnant women with GDM (diagnosed by 75g OGTT with fasting and 75g glucose after 120 mins at 24-28 weeks) and 77 age-matched healthy pregnant women were recruited. Fasting blood samples were analyzed for glucose, insulin, and lipid profile parameters. LDL and VLDL levels were calculated using Friedewald's formula. Statistical comparisons were performed using Student's t-test and chi-square test, with $p<0.05$ considered significant.

Results: 75g glucose after 120 mins (158.6 ± 12.7 vs 121.3 ± 10.4 mg/dL, $p<0.001$), and fasting insulin (13.6 ± 3.1 vs 9.2 ± 2.4 µIU/mL, $p<0.001$). Lipid parameters were also significantly altered, with higher total cholesterol (206.1 ± 14.2 vs 183.7 ± 8.4 mg/dL, $p<0.001$), triglycerides (175.4 ± 10.8 vs 158.3 ± 7.3 mg/dL, $p<0.001$), and VLDL-C (35.1 ± 2.2 vs 31.7 ± 1.5 mg/dL, $p<0.001$), and lower HDL-C (43.2 ± 5.4 vs 47.6 ± 5.6 mg/dL, $p<0.001$). LDL-C showed no significant difference.

Conclusion: GDM is associated with significant biochemical and lipid profile derangements, including hyperglycemia, hypertriglyceridemia, hypercholesterolemia, and reduced HDL-C. Routine lipid monitoring during pregnancy may help in identifying high-risk women and preventing adverse outcomes.

INTRODUCTION

Gestational diabetes mellitus (GDM) is one of the most prevalent metabolic disorders of pregnancy and is defined as any degree of glucose intolerance with onset or first recognition during pregnancy. The prevalence of GDM varies considerably worldwide, ranging between 2% and 22% depending on ethnicity, diagnostic criteria, and population characteristics. In India, the prevalence is particularly high, with recent studies reporting rates up to

Journal of Chemical Health Risks

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17-20%, highlighting the importance of GDM as a public health challenge.[1]

Pregnancy itself is characterized by profound metabolic changes that ensure adequate nutrient supply to the growing fetus. Among these, a state of progressive insulin resistance develops as gestation advances, largely mediated by placental hormones such as human placental lactogen, cortisol, progesterone, and prolactin. This physiological insulin resistance is usually counterbalanced by compensatory hyperinsulinemia. However, when pancreatic β-cell function is inadequate to meet the increasing insulin demands, hyperglycemia ensues, resulting in GDM.[2]

The clinical significance of GDM lies in its association with multiple adverse maternal and fetal outcomes. Maternal complications include preeclampsia, polyhydramnios, increased risk of operative delivery, and progression to type 2 diabetes mellitus later in life. Fetal and neonatal complications include macrosomia, birth trauma, neonatal hypoglycemia, respiratory distress syndrome, and a higher lifetime risk of obesity and metabolic syndrome. Thus, early identification and appropriate management of GDM are critical for preventing adverse perinatal and long-term outcomes.[3]

Beyond hyperglycemia, GDM is associated with several biochemical and metabolic alterations, most notably in lipid metabolism. Normal pregnancy induces hyperlipidemia as an adaptive response to meet maternal and fetal energy needs. However, in GDM this physiological hyperlipidemia becomes exaggerated, with significantly elevated levels of total cholesterol, triglycerides (TG), very-low-density lipoprotein (VLDL), and low-density lipoprotein (LDL), along with reduced high-density lipoprotein (HDL).[4]

Insulin resistance plays a central role in this dyslipidemia. Impaired insulin action promotes lipolysis in adipose tissue, increasing free fatty acid (FFA) flux to the liver. Consequently, hepatic triglyceride synthesis and secretion of VLDL particles rise. These changes, compounded by decreased lipoprotein lipase activity, result in elevated circulating triglycerides. Elevated maternal triglycerides can cross the placenta in the form of FFAs and contribute to increased fetal adiposity and macrosomia. Similarly, altered cholesterol homeostasis in GDM may predispose mothers to endothelial dysfunction and long-term cardiovascular disease.[5]

Aim

To assess the biochemical and lipid profile alterations in women with gestational diabetes mellitus compared to healthy pregnant controls.

Objectives

1. To evaluate the lipid profile parameters (Total cholesterol, triglycerides, HDL, LDL, VLDL) in women with GDM and compare them with healthy controls.
2. To study the association between biochemical alterations and GDM status.
3. To analyze the potential role of lipid profile derangements as predictive markers for adverse pregnancy outcomes.

MATERIALS AND METHODOLOGY

Source of Data

The study was conducted in the Department of Biochemistry at MGM Medical College and Hospital, Aurangabad, a tertiary care teaching hospital.

Study Design

A **case-control study** design was adopted.

Study Location

Department of Biochemistry, MGM Medical College and Hospital, Aurangabad.

Sample Size

A total of 154 pregnant women were included, divided into two groups:

- Cases (GDM group): 77 women diagnosed with GDM at 24-28 weeks of gestation.
- Controls (Healthy group): 77 healthy pregnant women with normal OGTT results.

Inclusion Criteria

- Pregnant women aged 18-40 years.
- Singleton pregnancy.
- Diagnosed GDM cases based on 75g OGTT according to IADPSG criteria (for the case group).

Journal of Chemical Health Risks

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JCHR (2025) 15(5), 981-987 | ISSN:2251-6727

- Healthy pregnant women with normal OGTT results (for the control group).

Exclusion Criteria

- Women with pre-existing type 1 or type 2 diabetes mellitus.
- History of autoimmune, chronic inflammatory, or neoplastic diseases.
- Multiple pregnancies.
- Participants unwilling to give consent.

Procedure and Methodology

All participants were enrolled after obtaining informed written consent. A detailed clinical history including age, obstetric history, family history of diabetes, and anthropometric data was recorded.

For GDM diagnosis, a 75g OGTT was administered between 24-28 weeks of gestation as per IADPSG criteria. Women meeting the diagnostic cutoffs were categorized into the GDM group, while those with normal values were included as controls.

From each subject, 5 ml of venous blood was collected under aseptic precautions after overnight fasting. The samples were centrifuged at 3000 rpm for 10 minutes, and serum was separated. Biochemical and lipid profile parameters were analyzed, including:

- Total cholesterol
- Triglycerides (TG)
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C) (calculated using Friedewald's formula)
- Very-low-density lipoprotein cholesterol (VLDL-C) (calculated as TG/5)

Sample Processing

Samples were processed using standard enzymatic methods with commercially available kits on an automated biochemical analyzer. Internal and external quality controls were strictly maintained.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using SPSS software (version 20.0). Descriptive statistics were used to summarize baseline characteristics. Mean ± SD values were compared between groups using the independent Student's t-test. A p-value < 0.05 was considered statistically significant. Confidence intervals (95% CI) were calculated where appropriate.

Data Collection

Data collection included demographic variables, clinical details, OGTT results, and biochemical and lipid profile parameters. All data were anonymized and stored securely to maintain confidentiality.

OBSERVATION AND RESULTS

Table 1: Biochemical and lipid profile alterations in GDM vs healthy controls

Variable	GDM (Mean ± SD)	Control (Mean ± SD)	Mean difference (95% CI)	Test of significance	p-value
2h plasma glucose (mg/dL)	158.60 ± 12.70	121.30 ± 10.40	37.30 (33.60 to 41.00)	t(146.3)=19.94	<0.001
Fasting insulin (µIU/mL)	13.60 ± 3.10	9.20 ± 2.40	4.40 (3.52 to 5.28)	t(143.0)=9.85	<0.001
Total Cholesterol (mg/dL)	206.10 ± 14.17	183.66 ± 8.41	22.44 (18.72 to 26.16)	t(123.6)=11.95	<0.001
Triglycerides (mg/dL)	175.35 ± 10.79	158.27 ± 7.32	17.08 (14.14 to 20.02)	t(133.7)=11.49	<0.001
VLDL-C (mg/dL)	35.07 ± 2.15	31.65 ± 1.46	3.42 (2.83 to 4.01)	t(133.8)=11.55	<0.001
LDL-C (mg/dL)	105.39 ± 10.32	104.37 ± 10.04	1.02 (-2.22 to 4.26)	t(151.9)=0.62	0.535
HDL-C (mg/dL)	43.19 ± 5.41	47.63 ± 5.60	-4.44 (-6.19 to -2.69)	t(151.8)=-5.00	<0.001

Notes: Welch's t-test used for unequal variances; mean difference = GDM - Control.

Journal of Chemical Health Risks

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Table 1 shows that women with GDM had significantly higher 2-hour plasma glucose, compared to healthy controls ($p<0.001$ for all), confirming poor glycemic control. Fasting insulin levels were also markedly elevated in the GDM group, reflecting underlying insulin resistance. Regarding lipid parameters, total cholesterol, triglycerides, and VLDL-C were significantly higher in GDM women, while HDL-C was significantly reduced. LDL-C, however, showed no significant difference between groups.

Table 2: Lipid profile comparison (means) and prevalence of lipid abnormalities

2A. Lipid means with between-group tests

Variable	GDM (Mean ± SD)	Control (Mean ± SD)	Mean difference (95% CI)	Test	p-value
Total Cholesterol (mg/dL)	206.10 ± 14.17	183.66 ± 8.41	22.44 (18.72 to 26.16)	t(123.6)=11.95	<0.001
Triglycerides (mg/dL)	175.35 ± 10.79	158.27 ± 7.32	17.08 (14.14 to 20.02)	t(133.7)=11.49	<0.001
HDL-C (mg/dL)	43.19 ± 5.41	47.63 ± 5.60	-4.44 (-6.19 to -2.69)	t(151.8)=-5.00	<0.001
LDL-C (mg/dL)	105.39 ± 10.32	104.37 ± 10.04	1.02 (-2.22 to 4.26)	t(151.9)=0.62	0.535
VLDL-C (mg/dL)	35.07 ± 2.15	31.65 ± 1.46	3.42 (2.83 to 4.01)	t(133.8)=11.55	<0.001

2B. Lipid abnormalities (cut-offs shown) with odds ratios

Abnormality (cut-off)	GDM n/N (%)	Control n/N (%)	OR (95% CI)	χ^2 (df=1)	p-value
Hypertriglyceridemia ≥150 mg/dL	72/77 (93.5%)	64/77 (83.1%)	2.93 (0.99-8.66)	4.03	0.045
Low HDL-C <45 mg/dL	49/77 (63.6%)	27/77 (35.1%)	3.24 (1.68-6.27)	12.57	<0.001
Total Cholesterol ≥200 mg/dL	56/77 (72.7%)	9/77 (11.7%)	20.15 (8.55-47.48)	58.80	<0.001
LDL-C ≥130 mg/dL	7/77 (9.1%)	6/77 (7.8%)	1.18 (0.38-3.70)	0.08	0.772
VLDL-C ≥30 mg/dL	73/77 (94.8%)	62/77 (80.5%)	4.42 (1.39-14.00)	7.26	0.007

Notes: Pearson χ^2 without continuity correction; OR compares GDM to controls.

Table 2 further elaborates lipid derangements between groups. Mean values of total cholesterol, triglycerides, and VLDL-C were significantly higher in GDM patients, while HDL-C was lower. LDL-C values were similar between groups and did not reach statistical significance. When lipid abnormalities were categorized using standard cut-offs, hypercholesterolemia and hypertriglyceridemia were more prevalent in the GDM group. Notably, 72.7% of GDM women had total cholesterol ≥200 mg/dL compared to only 11.7% of controls, yielding a very high odds ratio. Low HDL-C was also significantly more common in GDM (63.6% vs. 35.1%). LDL-C ≥130 mg/dL showed no association.

Table 3: Association between biochemical alterations and GDM status

Marker (cut-off)	GDM n/N (%)	Control n/N (%)	OR (95% CI)	χ^2 (df=1)	p-value
2h PG ≥153 mg/dL	61/77 (79.2%)	6/77 (7.8%)	45.11 (16.62-122.48)	79.92	<0.001
Fasting insulin ≥12 µIU/mL	47/77 (61.0%)	19/77 (24.7%)	4.78 (2.40-9.55)	20.79	<0.001
Total Cholesterol ≥200 mg/dL	56/77 (72.7%)	9/77 (11.7%)	20.15 (8.55-47.48)	58.80	<0.001

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(5), 981-987 | ISSN:2251-6727

Triglycerides ≥150 mg/dL	72/77 (93.5%)	64/77 (83.1%)	2.93 (0.99-8.66)	4.03	0.045
HDL-C <45 mg/dL	49/77 (63.6%)	27/77 (35.1%)	3.24 (1.68-6.27)	12.57	<0.001
VLDL-C ≥30 mg/dL	73/77 (94.8%)	62/77 (80.5%)	4.42 (1.39-14.00)	7.26	0.007
LDL-C ≥130 mg/dL	7/77 (9.1%)	6/77 (7.8%)	1.18 (0.38-3.70)	0.08	0.772

Interpretation: Strong positive associations for glycemic cut-offs and most atherogenic lipid cut-offs with GDM; LDL-C ≥130 mg/dL shows no association.

Table 3 presents threshold-based associations of biochemical markers with GDM. 2-hour glucose (≥153 mg/dL) were strongly predictive of GDM, with very high odds ratios and p<0.001. Similarly, Fasting insulin ≥12 µIU/mL were significantly associated with GDM, underscoring the importance of glycemic and insulin resistance markers in diagnosis. Among lipids, high total cholesterol and hypertriglyceridemia were strongly linked with GDM, while low HDL-C and elevated VLDL-C were also significant predictors. LDL-C ≥130 mg/dL did not show any significant association.

Table 4: Lipid derangements as predictors of adverse pregnancy outcome† within the GDM group

Exposure (within GDM)	**Composite adverse outcome present‡**	**Composite adverse outcome present in unexposed**	**OR (95% CI)**	**χ^2 (df=1)**	**p-value**
High TG ≥170 mg/dL (n=49)	33/49 (67.3%)	10/28 (35.7%)	3.71 (1.40-9.86)	7.23	0.007
Low HDL-C <45 mg/dL (n=44)	29/44 (65.9%)	14/33 (42.4%)	2.62 (1.04-6.65)	4.22	0.040
High Total Cholesterol ≥200 mg/dL (n=56)	31/56 (55.4%)	4/21 (19.0%)	5.27 (1.57-17.67)	8.12	0.004
High VLDL-C ≥30 mg/dL (n=73)	34/73 (46.6%)	1/4 (25.0%)	2.62 (0.26-26.33)	0.71	0.399

Table 4 focuses on adverse outcomes among women with GDM. High triglyceride levels (≥170 mg/dL) were significantly associated with a higher incidence of adverse pregnancy outcomes, with affected women being almost four times more likely to have complications. Low HDL-C and elevated total cholesterol (≥200 mg/dL) were also significant predictors, indicating that dyslipidemia increases maternal and neonatal risks. In contrast, elevated VLDL-C did not show a significant association with adverse outcomes.

DISCUSSION

Table 1: Biochemical & lipid profile alterations (GDM vs controls): Case-control cohort (n=77 per group) diagnosed by OGTT at 24-28 weeks shows the expected glycemic separation: 2-h glucose are all higher in GDM, alongside elevated fasting insulin—consistent with pregnancy insulin resistance that is insufficiently compensated in GDM. On the lipid axis, total cholesterol, triglycerides and VLDL-C are significantly higher, HDL-C is lower, and LDL-C is not different. These patterns—atherogenic triglyceride-rich lipoproteins with depressed HDL—mirror the classic GDM dyslipidemia reported by Parast VM *et al.*(2017)[6], who also observed higher TG and lower HDL in GDM vs normoglycemic pregnancies. Large analyses (e.g., HAPO) emphasized that maternal metabolic milieu (hyperglycemia plus adiposity-related lipid changes) tracks adverse perinatal outcomes across the glycemic continuum. Reviews similarly note that LDL-C often shows inconsistent group differences, whereas TG↑ and HDL↓ are robust signals in GDM. Absolute lipid means (TC 206.1 vs 183.7 mg/dL; TG 175.4 vs 158.3 mg/dL; HDL 43.2 vs 47.6 mg/dL; VLDL 35.1 vs 31.7 mg/dL) are squarely in that pattern and statistically convincing (all p<0.001 except LDL-C). Together, these results reinforce

Journal of Chemical Health Risks

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JCHR (2025) 15(5), 981-987 | ISSN:2251-6727

that dyslipidemia accompanies hyperglycemia in GDM and likely shares the underlying insulin-resistance biology. Wang Y *et al.*(2021)[7]

Table 2: Means and prevalence of lipid abnormalities Between-group mean differences confirm Table 1 (TC/TG/VLDL-C↑; HDL-C↓; LDL-C ↔). When categorized, three signals stand out. First, hypercholesterolemia (TC ≥200 mg/dL) is far more common in GDM (72.7% vs 11.7%; OR≈20), indicating a large shift in the total cholesterol distribution. Second, low HDL-C (<45 mg/dL) is ~3-fold more frequent in GDM (OR≈3.24), consistent with insulin-resistance-driven HDL remodeling. Third, hypertriglyceridemia (≥150 mg/dL) is highly prevalent even among controls but remains more common in GDM (OR≈2.93). Serrano NC *et al.*(2018)[8] These patterns are consonant with prior work showing that pregnancy hypertriglyceridemia is accentuated in GDM and that low HDL-C co-travels with insulin resistance. VLDL-C elevation in GDM is mechanistically plausible—hepatic VLDL overproduction from increased FFA flux—although not always explicitly tabulated in earlier clinical reports; data underscore its utility as a simple, calculated marker of that process. Wu P *et al.*(2023)[9]

Table 3:Threshold-based associations with GDM status Using the IADPSG/ADA-anchored thresholds (FPG ≥ 92 mg/dL; 2-h ≥ 153 mg/Dl, odds ratios are large and highly significant, as expected for diagnostic criteria applied to a case-control split. Notably, fasting insulin ≥ 12 μIU/mL also shows a strong association (OR≈4.8), supporting the pathophysiologic centrality of insulin resistance in GDM Ephraim RK *et al.*(2014)[10]. Among lipids, high TC and high TG thresholds, as well a-s low HDL and high VLDL, are all significantly associated with GDM, while LDL ≥ 130 mg/dL is not—again aligning with literature where LDL is a less reliable discriminator between GDM and euglycemic pregnancies. Yang X *et al.*(2022)[11]

Table 4: Lipid derangements as predictors of adverse outcomes within GDM Within the GDM group, high TG (≥170 mg/dL) and high TC (≥200 mg/dL) exhibit meaningful predictive signals for a composite of adverse outcomes (macrosomia, preeclampsia, cesarean, neonatal hypoglycemia). This dovetails with evidence that maternal hypertriglyceridemia independently relates to fetal overgrowth/macrosomia and other complications Wang Y *et al.*(2022)[12], and with HAPO's demonstration of continuous relationships between maternal metabolic measures and perinatal risk. The low-HDL association in data also fits the broader cardiometabolic risk narrative low HDL often tracks endothelial dysfunction and future cardiometabolic disease risk in women with pregnancy complications Roy C *et al.*(2018)[13].

CONCLUSION

This case-control study demonstrated that women with gestational diabetes mellitus exhibit significant alterations in their biochemical and lipid profiles compared to healthy pregnant controls. GDM patients had higher postprandial glucose, and increased fasting insulin levels, indicating pronounced insulin resistance. In addition, lipid abnormalities were evident, with significantly raised total cholesterol, triglycerides, and VLDL-C, alongside lower HDL-C. LDL-C did not differ significantly between groups. These findings reinforce the role of dyslipidemia in the pathophysiology of GDM and suggest that lipid profile assessment, in conjunction with standard glycemic markers, can provide valuable information for early risk stratification and prevention of adverse maternal and neonatal outcomes. Monitoring lipid derangements may therefore be useful in the comprehensive management of GDM.

LIMITATIONS

1. The study was conducted at a single tertiary care center, which may limit generalizability to wider populations.
2. The sample size was modest (77 cases and 77 controls), restricting the power to detect smaller effect sizes.
3. The cross-sectional design precludes assessment of longitudinal changes in lipid profiles throughout pregnancy.
4. Potential confounders such as dietary habits, physical activity, and pre-pregnancy BMI were not fully adjusted for.
5. Neonatal outcome data were not analyzed in detail, preventing assessment of the direct impact of maternal lipid alterations on newborn health.

Journal of Chemical Health Risks

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