International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622
© Gynaecology Journal
www.gynaecologyjournal.com
2025;9(3): 161-165
Received: 12-04-2025
Accepted: 14-05-2025

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Association of maternal serum leptin concentration with gestational diabetes mellitus

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DOI: https://www.doi.org/10.33545/gynae.2025.v9.i3c.1649

Abstract
Background: Gestational diabetes mellitus (GDM) represents a significant public health concern due to its relationship with elevated risks of maternal and fetal mortality and morbidity. Leptin, a peptide hormone, affects glucose homeostasis as it alters insulin sensitivity. By causing insulin resistance, it inhibits the insulin-mediated glucose uptake in tissues resulting in hyperglycemia. The study aimed to assess the association of maternal serum leptin concentration with gestational diabetes mellitus.
Methods: This case-control study was conducted at the Department of Obstetrics and Gynecology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka Bangladesh from September 2022 to August 2023. A total of 82 singleton pregnant women between 18-40 years of age in their 24-28 weeks of gestation were enrolled purposively. All cases were divided into two groups. Among them 41 women with diagnosed GDM were considered as the case group and the rest of the 41 matched healthy pregnant women were selected as the control group. Data were analyzed using SPSS 27.0.
Results: The average leptin levels were significantly higher in the cases (20.62±9.18 ng/mL) compared to the controls (13.35±6.47 ng/mL) at 24-28 weeks of gestation (p<0.001). With a leptin cutoff of 18.9 ng/mL, the odds ratio calculation indicated that gestational diabetes mellitus (GDM) was 3.8 times more likely to develop in pregnant women with elevated serum leptin levels (≥18.9 ng/mL) compared to those with lower levels (<18.9 ng/mL) (OR=3.776, p=0.007). A significant positive correlation was observed between serum leptin levels and both fasting blood sugar levels (p=0.001) and 2-hour post-glucose levels (p=0.002).
Conclusion: Elevated levels of serum leptin are associated with gestational diabetes mellitus (GDM).

Keywords: Gestational diabetes mellitus, GDM, Gestational age, Maternal serum leptin, Multigravida, Primigravida

1. Introduction
Gestational diabetes mellitus (GDM) refers to diabetes diagnosed during the second or third trimester of pregnancy, which was not previously present before gestation [1]. The global incidence of diabetes is rising, and a significant number of women affected by gestational diabetes mellitus are likely to experience continued increases, given the global rise in obesity rates among girls and women of reproductive age [2]. According to estimates, approximately 16.7% (21.1 million) of live births worldwide in 2021 were affected by hyperglycemia during pregnancy, with the majority of cases (75% to 90%) attributed to gestational diabetes mellitus [3]. Normal pregnancy is associated with progressive insulin resistance, resulting in a 40% to 50% reduction in insulin sensitivity [4]. In early pregnancy, glucose tolerance remains normal or slightly improved, with normal peripheral insulin sensitivity and basal hepatic glucose production. However, as pregnancy progresses, maternal insulin sensitivity declines, creating an insulin-resistant state to support the energy needs of both the mother and the rapidly growing fetus [5]. Modifiable risk factors for gestational diabetes mellitus include pre-pregnancy obesity (BMI ≥30 kg/m²), consumption of high glycemic foods, a sedentary lifestyle, reduced physical activity, high maternal stress levels, and the presence of conditions related to insulin resistance. A previous history of recurrent abortions, stillbirths, perinatal and neonatal deaths, intrauterine deaths (IUD), and prior macrosomic infants (>3.5 kg) are significant obstetric factors that may contribute to the development of gestational diabetes mellitus (GDM) [6]. About 40-60% of women with GDM show no symptoms, emphasizing the importance of understanding the pathophysiology of GDM for early detection, management, and prevention to improve health

outcomes for both mothers and their offspring [7]. In the hypothalamus, the long-form isoform of the leptin receptor (LEPRb) plays a critical role in regulating appetite, body weight, and bone mass by controlling the secretion of neuropeptides and neurotransmitters. Additionally, the cleaved soluble leptin receptor acts as the primary binding protein for circulating leptin, influencing its bioavailability in the body [8]. In individual's sensitive to leptin, this hormone has opposing effects on insulin and glucose regulation. Leptin inhibits the production and release of insulin from pancreatic β-cells, while insulin stimulates leptin secretion from adipose tissue. This creates a hormonal regulatory feedback loop, known as the adipo-insular axis, which is essential for maintaining balance. Dysfunction in this axis plays a crucial role in the development of hyperinsulinemia and hyperglycemia [9]. In gestational diabetes mellitus (GDM), the placenta undergoes structural and functional changes, such as overgrowth, villous immaturity, and vascular hyperplasia of the chorionic villi [10]. GDM also creates an inflammatory environment that increases the production of pro-inflammatory cytokines like IL-6 and TNF-α, which can further elevate leptin levels [11]. Several studies have reported that higher maternal serum leptin levels are a risk factor for developing GDM and have a positive correlation with insulin resistance [12, 13], although some studies have failed to establish a significant correlation [14].

2. Methodology

This case-control study was conducted at the Department of Fetomaternal Medicine and Department of Obstetrics & Gynecology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh from September 2022 to August 2023. A total of 82 singleton pregnant women aged 18-40 years at 24-28 weeks of gestation were enrolled. The study included 41 women diagnosed with GDM (case group) and 41 women without GDM (control group). Inclusion criteria were singleton pregnancy, age between 18-40 years, gestational age between 24-28 weeks, and consent to participate. The exclusion criteria of this study included multiple pregnancies, pre-gestational or overt diabetes, thyroid disease, chronic hypertension, preeclampsia, excessive weight gain or BMI ≥30 kg/m², and use of medications affecting glucose metabolism (e.g., steroids, thiazide diuretics). After obtaining consent and confirming eligibility, data were collected through interviews, observations, clinical examination, and laboratory tests. Serum leptin levels were measured using the DRG Leptin ELISA kit at the Department of Microbiology and Immunology, BSMMU, Dhaka. The study was approved by the hospital's ethical committee, and data were analyzed using SPSS version 27.0.

3. Results

In this study, the distribution of respondents based on socio-demographic characteristics was comparable between the two groups, with no statistically significant difference observed (p-value >0.05). Similarly, the distribution of obstetrical characteristics did not differ significantly between the groups (p >0.05). Additionally, the variation in body mass index (BMI) and the mean (±SD) BMI between the groups was not statistically significant (p>0.05). However, the mean serum leptin level was significantly higher in the cases (20.62±9.18 ng/mL) compared to the controls (13.35±6.47 ng/mL), with a highly significant result (p<0.001). The study revealed a moderate positive correlation between maternal serum leptin levels and fasting blood glucose levels (r=0.356; p=0.001). The coefficient of determination (R^2=0.127) indicates that approximately 12.7% of the variability in fasting blood glucose levels can be attributed to changes in maternal serum leptin. A similar positive correlation was observed between maternal serum leptin and blood glucose levels two hours after a 75g glucose challenge (r=0.335, p=0.002). The R^2 value of 0.112 suggests that approximately 11.2% of the variance in post-glucose blood sugar levels can be explained by variations in maternal serum leptin. Furthermore, respondents with serum leptin levels ≥18.9 ng/mL had 3.8 times higher odds of developing gestational diabetes mellitus compared to those with leptin levels <18.9 ng/mL (OR=3.776, 95% CI=1.505-9.475; p=0.007).

Table 1: Distribution of the respondents socio-demographic characteristics.

Characteristics	Group				p-value
	Case		Control		
	n	%	n	%	
Age (Years)					
18 - 25 Yrs.	16	39.0%	21	51.2%	
26 - 34 Yrs.	18	43.9%	15	36.6%	[a]0.527[ns]
35 - 40 Yrs.	7	17.1%	5	12.2%	
Mean ± SD	27.73 ± 6.12		26.80 ± 6.07		[b]0.493[ns]
Education					
Up to primary	8	19.5%	5	12.2%	
Secondary/equivalent	23	56.1%	19	46.3%	[a]0.236[ns]
≥Higher secondary	10	24.4%	17	41.5%	
Occupation					
Housewife	34	82.9%	29	70.7%	
Student	1	2.4%	5	12.2%	[c]0.244[ns]
Service holder	6	14.6%	7	17.1%	

a: Chi-square test, b: Unpaired student's t-test c: Fisher's exact, NS=Not significant

Table 2: Distribution of the respondents according to their obstetrical characteristics.

Characteristics	Case	Control	p-value
	n (%)	n (%)	
Gestational age (Weeks)			
Mean ± SD	25.34 ±1.44	25.07 ±1.43	[b]0.402[ns]
Min - Max	24 - 28	24 - 28	
Gravida			
Primigravida	11 (26.8)	17 (41.5)	[c]0.162[ns]
Multigravida	30 (73.2)	24 (58.5)	

b: Unpaired student's t-test c: Fisher's exact, NS=Not significant

Table 3: Distribution of respondents according to the BMI.

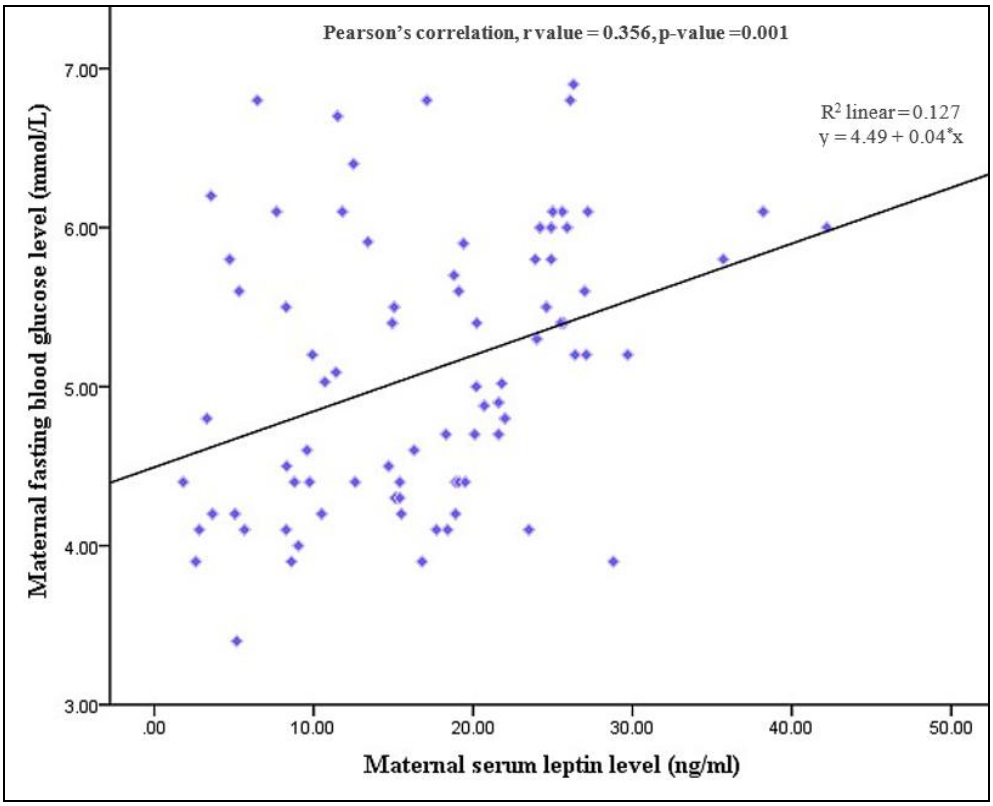
BMI (kg/m2)	Case		Control		p-value
	n	%	n	%	
Underweight (<18.5)	0	0.0%	1	2.4%	
Normal (18.5 - 24.9)	15	36.6%	21	51.2%	[c]0.183[ns]
Overweight (25.0 - 29.9)	26	63.4%	19	46.3%	
Mean ± SD	26.09 ± 3.75		24.64 ± 2.85		[b]0.052[ns]

b: Unpaired student's t-test c: Fisher's exact, NS=Not significant

Table 4: Comparison the mean (±SD) serum leptin level among the case and control groups respondents.

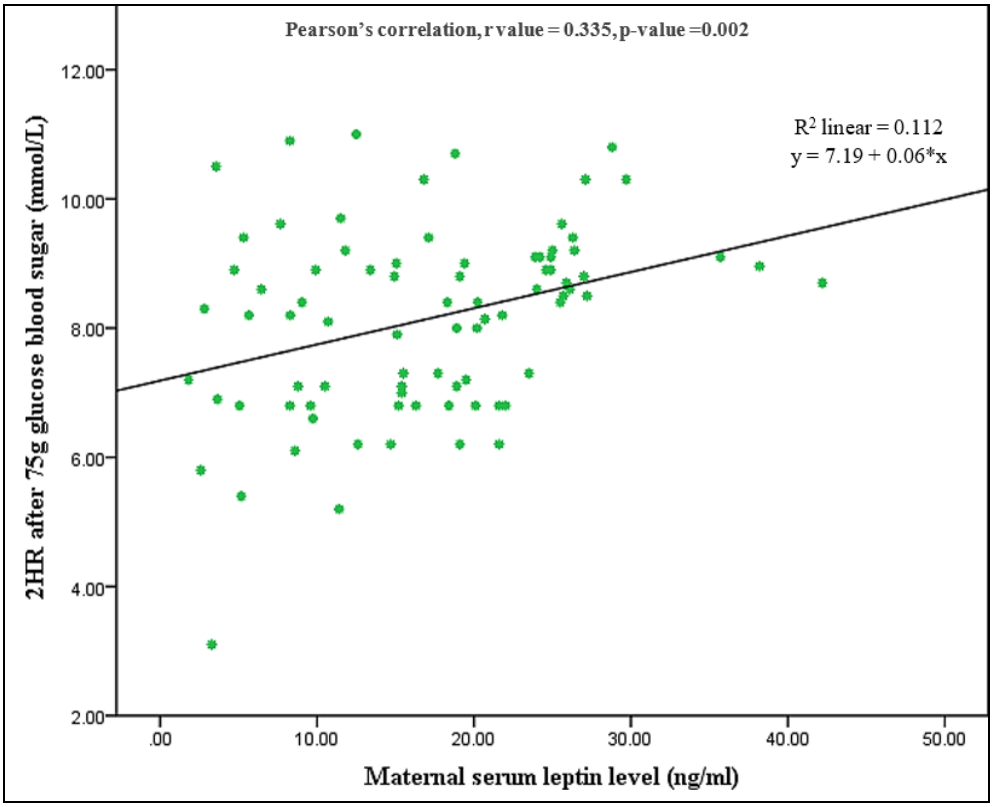
Variables	Case	Control	p-value
	(Mean ± SD)	(Mean ± SD)	
Serum leptin level (ng/mL)	20.62 ± 9.18	13.35 ± 6.47	[b]<0.001[s]

b: Unpaired student's t-test, s= significant test



R2 = percentage of variability of fasting blood glucose in GDM due to raised serum leptin level and vice versa,
r = Pearson's correlation coefficient

Fig 1: Correlation between maternal serum leptin and fasting blood glucose level.



R2 = percentage of variability of 2HA 75g glucose blood sugar in GDM due to raised serum leptin level and vice versa.
r = Pearson's correlation coefficient

Fig 2: Correlation between serum leptin level and 2hrs after 75gm glucose blood sugar.

Table 5: Odd's ratio (OR) and 95% confidence levels (CI) for GDM and Non GDM respondents according to serum leptin level in pregnancy.

Serum leptin level	Case	Control	p-value	OR	95% CI (Lower-upper)
	n (%)	n (%)			
≥ 18.9 ng/mL	25 (61.0)	12 (29.3)	[a]0.007[s]	3.776	1.505-9.475
< 18.9 ng/mL	16 (39.0)	29 (70.7)			

c: Chi-square test s = Significant OR= Odd's ratio, CI= Confidence interval

4. Discussion

In this study, two-fifths (40.2%) of the total respondents were aged between 26-34 years, with a nearly identical mean (±SD) age for both the case and control groups: 27.73±6.12 and 26.80±6.07 years, respectively (p>0.05). A systematic review and meta-analysis by Li *et al*. [15] indicated that women aged 25 years and older had a significantly higher risk of developing gestational diabetes mellitus (GDM) in Asian populations. Similarly, Soheilykhah *et al*. [16] found no significant age differences among pregnant women in their study. The majority of participants in both groups were educated up to the SSC/equivalent level (cases: 56.1%, controls: 46.3%) and were housewives (cases: 82.9%, controls: 70.7%). Women from lower-middle-class social status made up 65.9% of the cases, compared to 43.9% of the controls. However, none of these socio-demographic factors showed statistical significance (p>0.05). Laila and Mallik [17] also reported similar findings in their study on risk factors for GDM in Bangladeshi women, where both cases and controls were predominantly housewives (74.2% vs. 88.6%, respectively, p=0.121), and around 60% of both groups were from upper-middle-class backgrounds (p=0.117). These observations align with Yang *et al*. (2002), who found no significant association between education and GDM in Chinese women, and Khan *et al*. (2013), who reported no significant differences in monthly income, female occupation, or education level between women with GDM and healthy pregnant women, further supporting the consistency of these non-significant associations with GDM. The mean gestational age was similar between the two groups, with cases at 25.34±1.44 weeks and controls at 25.07±1.43 weeks. Nearly three-fourths (73.2%) of the women with GDM and 58.5% of the controls were multigravida. No statistically significant difference was found between the case and control groups regarding obstetric characteristics (p>0.05). These findings are consistent with Alharti *et al*. [18], who identified multigravida and a history of GDM in previous pregnancies as major risk factors for GDM. Gandhewar *et al*. [19] also found that the incidence of GDM was higher among multigravida women (80.7%) compared to primigravida women (19.4%). Regarding body mass index (BMI), the study found no statistically significant difference between the case and control groups (p>0.05). However, the control group had a higher proportion of women with normal body weight (51.2%) compared to the cases (36.6%), while more women in the GDM group were overweight (63.4%) compared to the control group (46.3%). Despite this, no significant difference was observed in the mean BMI between the two groups (p>0.05). A large prospective study by Duman [20] in Pakistan also identified BMI as a significant risk factor for GDM. The mean (±SD) maternal serum leptin level was significantly higher in women with GDM (20.62 ± 9.18 ng/mL) compared to the healthy pregnant women (13.35 ± 6.47 ng/mL), with a highly significant difference (p<0.001). Women with serum leptin levels ≥18.9 ng/mL had 3.8 times higher odds of developing GDM compared to those with leptin levels <18.9 ng/mL (OR=3.776, 95% CI=1.505-9.475; p=0.007). Pearson's correlation analysis revealed a positive correlation between serum leptin levels and both fasting blood sugar (r=0.356, p=0.001) and two-hour post-glucose blood sugar levels (r=0.335, p=0.002), suggesting that increases in maternal serum leptin levels are associated with higher maternal blood glucose levels. These findings are consistent with several previous studies. Kautzky-Willer *et al*. [21] observed that plasma leptin levels were significantly higher in women with GDM (24.9±1.6 ng/mL) than in women with normal glucose tolerance (18.2 ± 1.5 ng/mL), with both groups exhibiting higher levels compared to non-pregnant women (8.2±1.3 ng/mL, p<0.0005). Vitoratos *et al*. [22] found that maternal serum leptin levels were significantly higher in women with GDM (16.52±5.07 ng/mL) compared to those with uncomplicated pregnancies (10.61±1.47 ng/mL), with a strong positive correlation between leptin concentrations and plasma glucose levels obtained one hour after 50g glucose administration (r=0.84, p<0.001). Liu *et al*. [23] similarly reported higher serum leptin levels in pregnant women with GDM (14.9±4.3 µg/L) compared to healthy controls (10.0±1.8 µg/L). Qiu *et al*. [24] found that elevated leptin concentrations were positively associated with GDM risk, with women having leptin levels ≥31.0 ng/mL experiencing a 4.7-fold increased risk of GDM. A study by Chen *et al*. [25] showed that serum leptin levels were significantly higher in women with GDM compared to controls before delivery, but decreased after delivery (p<0.001). These findings are further supported by Bawah *et al*. [26], who found elevated serum leptin to be significantly associated with GDM (OR=1.166; CI=1.104-1.233; p<0.0001).

Limitation of the study

This study had limitations, including a small, purposively selected sample from BSMMU, Dhaka, which may not represent the broader population. The study was conducted over a short period, limiting its scope, and the findings may not be generalizable.

5. Conclusion and Recommendation

The findings of this study indicate that pregnant women with gestational diabetes mellitus (GDM) tend to have higher serum leptin levels compared to healthy pregnant women. Additionally, a positive correlation was observed between serum leptin levels and maternal blood glucose levels, both fasting and two hours after a 75g glucose load. These results suggest that elevated serum leptin levels are associated with the development of GDM.

Funding

No funding sources.

Conflict of interest

None declared.

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How to Cite This Article
Ripa IJ, Sayeeda S, Majumder J, Yesmin F, Islam N, Parveen T, Akhtar N. Association of maternal serum leptin concentration with gestational diabetes mellitus. International Journal of Clinical Obstetrics and Gynaecology. 2025;9(3):161-165.

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