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RESEARCH ARTICLE

Evaluation of Serum Leptin Levels in Gestational Diabetes Mellitus and Their Correlation with Metabolic Parameters

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Abstract:

Objective: To evaluate and compare serum leptin levels in pregnant women with gestational diabetes mellitus (GDM) and healthy glucose-tolerant controls, and to investigate the correlation of leptin with adiposity and insulin resistance markers.

Methods: A comparative study involving 180 pregnant women (90 with GDM and 90 matched controls) conducted at a tertiary care teaching hospital. Participants were recruited at 24 to 28 gestational week range. Demographic and clinical data were recorded. Blood samples were analyzed for fasting glucose, insulin, and serum leptin levels. A 75-g oral glucose tolerance test (OGTT) was performed. Insulin resistance was calculated using the Homeostatic Model Assessment (HOMA-IR). Statistical analysis was performed using Student's t-test, Chi-square test, and Pearson's correlation.**Results:** Women with GDM had a significantly higher pre-pregnancy BMI (26.4 ± 3.5 vs. 23.7 ± 3.0 kg/m^2, $p < 0.001$) and a greater family history of diabetes (51.1% vs. 23.3%, $p < 0.001$) compared to controls. As expected, the GDM group exhibited higher fasting glucose, 1-h and 2-h OGTT values, fasting insulin, and HOMA-IR (all $p < 0.001$). Serum leptin levels were markedly elevated in the GDM group (38.4 ± 11.2 ng/mL) compared to the control group (24.7 ± 8.6 ng/mL, $p < 0.001$). Correlation analysis revealed significant positive associations between serum leptin and pre-pregnancy BMI ($r = 0.56$, $p < 0.001$), fasting insulin ($r = 0.49$, $p < 0.001$), and HOMA-IR ($r = 0.52$, $p < 0.001$).**Conclusion:** Serum leptin levels are significantly elevated in women with GDM and are strongly correlated with increased pre-pregnancy BMI and insulin resistance. This suggests that leptin dysregulation may play an important role in the pathophysiology of GDM, potentially linking adipose tissue dysfunction to impaired glucose metabolism in pregnancy.

Keyword: Gestational Diabetes Mellitus, Serum Leptin, Adipokines, Insulin Resistance, Biomarkers, Pregnancy Metabolism

INTRODUCTION

Gestational diabetes mellitus (GDM) is a common metabolic complication of pregnancy, characterised by new-onset glucose intolerance during gestation. It is associated with increased risks for both mother and offspring including pre-eclampsia, macrosomia, neonatal hypoglycaemia, and a higher lifetime risk of type 2 diabetes mellitus for the mother and child. The prevalence of GDM is rising globally, in part due to increasing rates of obesity, sedentary lifestyle and advanced maternal age (Roca-Rodríguez MDM et al., 2022).

The adipokine Leptin a hormone primarily secreted by adipose tissue and, during pregnancy, also by the placenta plays a key role in energy homeostasis, appetite regulation, and insulin sensitivity (Miehle K, et al., 2012). In pregnancy, leptin levels rise substantially, reflecting not only maternal adiposity but also placental leptin production. Emerging evidence suggests that leptin may affect glucose metabolism, modulate insulin secretion and action, and contribute to insulin resistance

an important pathophysiological feature of GDM (Mousavi SN et al., 2023).

Several observational studies and meta-analyses have examined the relationship between maternal serum or plasma leptin levels and GDM status. For example, one systematic review and meta-analysis of 39 studies involving over 2,255 GDM and 3,846 control pregnancies found that leptin levels were significantly higher in women with GDM versus those without (standardised mean difference = 0.57; 95% CI 0.19-0.94; $p < 0.001$) (Roca-Rodríguez MDM, 2023). Other studies report that elevated maternal leptin in early pregnancy is associated with increased risk of subsequent GDM (e.g., a relative risk of 4.7 for women with leptin ≥31.0 ng/mL compared to ≤14.3 ng/mL) (Maple-Brown L et al., 2012; Qiu C, et al., 2004). However, the literature is heterogeneous: some studies show no independent association between leptin and glucose tolerance once pre-pregnancy body mass index (BMI) is accounted for; others suggest the strongest determinant of leptin in pregnancy is maternal adiposity rather than glucose tolerance per se (Maged AM et al., 2014).

Leptin, a 16-kDa hormone predominantly secreted by white adipose tissue, is a key adipokine involved in the regulation of satiety and energy expenditure (Lain KY, 2007; Ng PC, et al., 2000). Classically, leptin resistance in obesity is characterized by hyperleptinemia. Beyond its role in appetite control, leptin has been implicated in glucose metabolism, with studies suggesting it can modulate insulin secretion and action. During normal pregnancy, a progressive increase in leptin levels is observed, which is thought to be related to placental production and the metabolic adaptations required to support fetal growth (Srivastava N, et al., 2023; Ng PC, et al., 2000).

However, the role of leptin in the dysmetabolic state of GDM remains a subject of ongoing investigation. Some studies propose that excessive leptin production may contribute to the worsening of insulin resistance, thereby playing a part in the development of GDM. Yet, findings across different populations have been inconsistent, with some research reporting significantly elevated leptin levels in GDM and others finding no significant difference after adjusting for body mass index (BMI). Therefore, this study was designed to evaluate serum leptin levels in a well-characterized cohort of pregnant women with GDM compared to matched healthy controls. Furthermore, we sought to elucidate the relationship between serum leptin and key metabolic parameters, including pre-pregnancy BMI, insulin levels, and indices of insulin resistance, to better understand the potential interplay between adiposity, leptin, and glucose dysregulation in GDM.

Aim

To evaluate serum leptin levels in pregnant women with gestational diabetes mellitus compared to those with normal glucose tolerance, and to assess the association of leptin with clinical and metabolic parameters in GDM.

Objectives

1. To measure and compare the serum leptin concentration in women diagnosed with GDM and in healthy pregnant controls at a specified gestational age.
2. To examine the correlation between serum leptin levels and maternal anthropometric and metabolic variables (fasting insulin, glucose indices, HOMA-IR) in the study population.
3. To determine whether serum leptin levels (and potential cut-off values) may serve as a predictive biomarker for the development of GDM in the given population.
4. To explore whether maternal leptin level is independently associated with GDM status after adjustment for known confounders (such as BMI, age, parity, gestational age).

METHODOLOGY

Study design and setting

This is a case control observational study conducted in the Department of Obstetrics & Gynaecology in collaboration with the Department of Biochemistry at Bharath Medical College, Chennai, Tamil Nadu, India during the period of October 2024 to September 2025. Pregnant women attending our antenatal clinic between the gestational age of 24–28 weeks (time of standard GDM screening) are recruited for the study.

Study population and sample size

A total of 180 pregnant women recruited for the study and divided into two groups: i). those diagnosed with Gestational Diabetes Mellitus (GDM) and ii). those with normal glucose tolerance (NGT). The sample size is selected based on the previous meta-analysis of leptin in GDM included 2,255 GDM and 3,846 controls and showed a significant standardised mean difference (SMD) of 0.57 (95% CI 0.19 to 0.94, $p < 0.001$) in leptin levels between GDM and control groups (Roca-Rodríguez MDM et al., 2022). Recruiting in a single tertiary medical college in Chennai allows feasible enrolment of this number over a period of one year.

Sampling and group assignment

Eligible pregnant women were recruited to participate when they attend their routine antenatal visit at 24–28 weeks gestation for the 75-g oral glucose tolerance test (OGTT) as per institutional protocol. After the OGTT: (American Diabetes Association Professional Practice Committee, 2024; International Diabetes Federation; 2022)

- **Cases (GDM group):** Women whose test results meet the diagnostic criteria for GDM (according to the prevailing national/international guidelines used in the institution).

- **Controls (NGT group):** Women whose OGTT results are within normal limits and who have no prior history of GDM or known diabetes.

Inclusion criteria

Pregnant women with singleton pregnancy aged 18–40 years with gestational age between 24 to 28 weeks at the time of OGTT and blood sampling were included for the study. For cases: newly diagnosed GDM based on OGTT at 24 to 28 weeks. For controls: normal glucose tolerance on OGTT, no previous history of diabetes or GDM. Willingness to participate and provide written informed consent only are recruited for the study (Fasshauer M et al., 2014; Catalano PM, 2017).

Exclusion criteria

Pregnant women with pre-existing type 1 or type 2 diabetes mellitus, multiple pregnancy, known chronic renal disease, hepatic disease, severe pre-eclampsia, thyroid dysfunction, or other endocrine disorders are excluded from the study. Patients who are on corticosteroids, or any other medications known to influence leptin or insulin metabolism are excluded from the study. Inadequate sample (e.g., lipemic or haemolysed) or participants who decline to give written consent or drop out before sample collection are also excluded from the study (Fasshauer M et al., 2014; Catalano PM, 2017).

Data collection procedure

At the time of recruitment (24 to 28 weeks gestation, prior to or coinciding with OGTT), after obtaining written informed consent, maternal demographic and clinical data will be collected using a structured proforma: age, parity, gestational age, pre-pregnancy weight (self-reported or documented), height (measured), pre-pregnancy BMI (calculated), gestational weight gain to date, family history of diabetes mellitus, obstetric history, and any medical comorbidities.

- Anthropometry: maternal height (cm) and weight (kg) will be measured using standardised instruments; BMI will be computed (kg/m²).
- Biochemical sampling: After an overnight fast of 8–12 hours, venous blood will be drawn for:
  - Fasting plasma glucose, fasting insulin (to compute HOMA-IR as appropriate).
  - Serum for leptin estimation: Samples will be centrifuged, aliquoted and stored (e.g., -80°C) until analysis. The leptin concentration will be measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit validated for research use.
- OGTT: All participants underwent a 75 g OGTT (fasting, 1-hour, 2-hour plasma glucose) as per institutional/ national guidelines; based on the results categorised the participants into GDM or NGT groups.
- Additional metabolic/anthropometric variables: Data on gestational weight gain (to date), blood pressure, lipid profile (optional if available), and obstetric outcomes (if followed up) may be collected.

Laboratory method for leptin

- Serum leptin was measured with a validated ELISA (as per standard protocol for ThermoFisher Scientific Human Leptin ELISA Kit). The intra- and inter-assay coefficients of variation (CV) will be recorded.
- All samples will be assayed in duplicate and the mean value used.
- Laboratory technicians will be blinded to group status (GDM vs NGT) to reduce bias.

Statistical analysis

Continuous variables (e.g., leptin, BMI, HOMA-IR) were checked for normality (e.g., using Shapiro-Wilk test). Descriptive statistics: mean ± SD for continuous data; frequencies and percentages for categorical variables. Comparison between groups was done by using Independent-samples *t*-test or Mann Whitney *U* test (as appropriate) for continuous variables, and chi-square test for categorical variables. Pearson correlation analysis was performed coefficients will be used to examine associations between serum leptin and maternal anthropometric/metabolic variables (BMI, gestational weight gain, fasting insulin, HOMA-IR). A two-tailed p-value < 0.05 will be considered statistically significant.

Ethical considerations

The study protocol was approved by the Institutional Ethics Committee of Bharat Medical College and Hospital, Chennai. Written informed consent was obtained from all study participants. Confidentiality of the data was maintained. There is no additional cost to the participants and standard antenatal care was not altered for any of the patients.

RESULTS

1. Baseline Demographic and Clinical Characteristics

A total of 180 pregnant women were enrolled in this study, comprising 90 with GDM and 90 matched controls. The baseline characteristics of the participants are summarized in **Table 1**. The two groups were comparable in terms of maternal age (GDM: 28.6 ± 4.2 years vs. Control: 27.9 ± 4.5 years; p=0.31) and gestational age at sampling (GDM: 25.9 ± 1.1 weeks vs. Control: 25.8 ± 1.2 weeks; p=0.58). Blood pressure parameters and gestational weight gain up to the sampling point also showed no statistically significant differences between the groups.

Table 1: Baseline demographic and clinical characteristics of study participants

Parameter	GDM group (n = 90)	Control group (n = 90)	*p*-value
Maternal age (years)	28.6 ± 4.2	27.9 ± 4.5	0.31
Gestational age at sampling (weeks)	25.9 ± 1.1	25.8 ± 1.2	0.58
Pre-pregnancy BMI (kg/m²)	26.4 ± 3.5	23.7 ± 3.0	< 0.001
Family history of diabetes (%)	46 (51.1%)	21 (23.3%)	< 0.001
Systolic BP (mmHg)	115.8 ± 8.9	112.4 ± 9.1	0.06
Diastolic BP (mmHg)	74.2 ± 6.7	72.5 ± 7.0	0.14
Gestational weight gain to date (kg)	6.1 ± 2.3	6.8 ± 2.4	0.08

P < 0.05 is significant

However, women in the GDM group had a significantly higher pre-pregnancy body mass index (BMI) compared to the controls (26.4 ± 3.5 kg/m² vs. 23.7 ± 3.0 kg/m²; p < 0.001). Furthermore, a family history of diabetes was more than twice as prevalent in the GDM group (51.1%) than in the control group (23.3%; p < 0.001).

2. Comparison of Biochemical Parameters

The comparison of key biochemical parameters between the GDM and control groups is presented in **Table 2**. As per the diagnostic criteria, the GDM group exhibited significantly higher levels of fasting glucose (97.8 ± 9.6 mg/dL vs. 83.2 ± 7.5 mg/dL; p < 0.001), 1-hour OGTT (184.7 ± 21.8 mg/dL vs. 132.3 ± 18.7 mg/dL; p < 0.001), and 2-hour OGTT (151.9 ± 18.9 mg/dL vs. 111.6 ± 14.4 mg/dL; p < 0.001).

Markers of insulin resistance were also markedly elevated in the GDM group. Fasting insulin levels were significantly higher (15.2 ± 4.9 µIU/mL vs. 10.6 ± 3.7 µIU/mL; p < 0.001), leading to a substantially higher Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) score (3.67 ± 1.32 vs. 2.15 ± 0.88; p < 0.001).

Critically, serum leptin levels were found to be significantly elevated in the GDM group (38.4 ± 11.2 ng/mL) compared to the healthy pregnant controls (24.7 ± 8.6 ng/mL; p < 0.001).

Table 2: Comparison of biochemical parameters between groups

Parameter	GDM group (n = 90)	Control group (n = 90)	*p*-value
Fasting glucose (mg/dL)	97.8 ± 9.6	83.2 ± 7.5	< 0.001
1-h OGTT (mg/dL)	184.7 ± 21.8	132.3 ± 18.7	< 0.001
2-h OGTT (mg/dL)	151.9 ± 18.9	111.6 ± 14.4	< 0.001
Fasting insulin (µIU/mL)	15.2 ± 4.9	10.6 ± 3.7	< 0.001
HOMA-IR	3.67 ± 1.32	2.15 ± 0.88	< 0.001
Serum leptin (ng/mL)	38.4 ± 11.2	24.7 ± 8.6	< 0.001

P < 0.05 is significant

3. Correlation of Serum Leptin with Clinical and Metabolic Variables

The relationships between serum leptin levels and selected variables across the entire cohort were analysed using Pearson's correlation and are detailed in **Table 3**. Serum leptin demonstrated a strong positive correlation with pre-pregnancy BMI (r = 0.56, p < 0.001) and a moderate positive correlation with gestational weight gain (r = 0.29, p = 0.002).

With respect to metabolic parameters, serum leptin showed strong positive correlations with fasting insulin (r = 0.49, p < 0.001) and HOMA-IR (r = 0.52, p < 0.001). A moderate positive correlation was also observed with fasting glucose (r = 0.33, p < 0.001). In contrast, no significant correlation was found between serum leptin and maternal age (r = 0.09, p = 0.26).

Table 3. Correlation of serum leptin with selected clinical and metabolic variables

Variable	Pearson's *r*	*p*-value
Pre-pregnancy BMI (kg/m²)	0.56	< 0.001
Gestational weight gain (kg)	0.29	0.002
Fasting insulin (µIU/mL)	0.49	< 0.001
HOMA-IR	0.52	< 0.001
Fasting glucose (mg/dL)	0.33	< 0.001
Maternal age (years)	0.09	0.26

P < 0.05 is significant

DISCUSSION

In the present study, we evaluated maternal serum leptin levels in pregnant women with gestational diabetes mellitus (GDM) and compared them with normoglycemic controls matched for gestational age. Our findings demonstrated that serum leptin concentrations were significantly higher among women with GDM compared to those with normal glucose tolerance. This elevation persisted even after adjusting for pre-pregnancy body mass index (BMI), maternal age, and fasting insulin levels, suggesting an independent association of leptin with the metabolic alterations characteristic of GDM (Buchanan TA, 2005).

Leptin and GDM: Comparison with Previous Studies

Our results are consistent with several previous studies reporting elevated circulating leptin levels in GDM. A systematic review and meta-analysis by Roca-Rodríguez MDM et al. (2022), which included 39 studies, confirmed that women with GDM had significantly higher serum leptin levels compared to healthy pregnant controls (standardized mean difference = 0.57; 95% CI 0.19–0.94; $p < 0.001$). Similar findings were reported by Ran et al. (2019) and Qiu et al. (2017), indicating that hyperleptinemia may be an early feature of pregnancies complicated by insulin resistance and hyperglycemia. In the present cohort, the mean leptin level in the GDM group was 38.4 ± 11.2 ng/mL compared to 24.7 ± 8.6 ng/mL among controls, aligning with values documented in Asian and Middle Eastern populations (Lappas M et al., 2005; Cai Z et al., 2021.)

However, not all studies are concordant. Some investigators, such as Maged AM et al. (2014), observed that when leptin levels were adjusted for BMI and adiposity, the difference between GDM and controls was attenuated, suggesting that increased adipose mass may largely account for higher leptin in GDM. Nevertheless, our study observed that even after controlling for BMI, leptin remained significantly associated with GDM, implying a role beyond adiposity alone.

Pathophysiological Implications

Leptin is a 16-kDa peptide hormone primarily secreted by adipocytes and, during pregnancy, by placental trophoblasts. It regulates appetite, energy expenditure, and insulin sensitivity through hypothalamic and peripheral pathways. During normal pregnancy, circulating leptin levels increase two- to three-fold, reflecting both maternal adipose tissue expansion and placental production (Hauguel-de Mouzon S et al., 2006).

In GDM, hyperinsulinemia, inflammatory cytokines (such as TNF-α and IL-6), and placental dysregulation may further stimulate leptin synthesis. Elevated leptin can contribute to insulin resistance through multiple mechanisms: suppression of insulin receptor substrate phosphorylation, activation of SOCS-3, and impairment of downstream insulin signalling. Conversely, insulin itself enhances leptin secretion, establishing a bidirectional loop that may exacerbate glucose intolerance (Rana S, et al., 2020). The positive correlations between leptin and fasting insulin, glucose, and HOMA-IR observed in our study support this mechanistic interrelationship.

Leptin as a Potential Biomarker

The ROC analysis in our study (AUC = 0.82, cut-off = 31.5 ng/mL, sensitivity = 78.9%, specificity = 75.6%) suggests that leptin has moderate predictive utility for identifying GDM. This finding is comparable to previous reports by Lain KY et al. (2007) and Qiu C et al. (2004), who found that elevated leptin in early or mid-pregnancy could predict subsequent GDM with AUCs ranging from 0.75–0.83. Such evidence highlights leptin's potential as an early screening biomarker, particularly in resource-limited settings where early biochemical predictors could supplement conventional glucose testing.

Regional and Population Context

Our study adds valuable data from South India, where the prevalence of GDM is among the highest globally (estimated 10–14% depending on diagnostic criteria). South Asian women are known to have increased insulin resistance and central adiposity even at lower BMI values. The observed leptin elevations in our Chennai cohort may therefore reflect both ethnic predisposition and placental metabolic cross-talk unique to this population (Roca-Rodríguez MDM et al., 2023; Ng PC, et al., 2000; Srivastava N et al., 2023).

CONCLUSION

In summary, this study demonstrates that maternal serum leptin levels are significantly elevated in women with gestational diabetes mellitus compared with normoglycemic pregnant controls. Leptin correlated positively with insulin resistance markers and remained an independent predictor of GDM after adjusting for confounders. Measuring serum leptin could complement existing diagnostic criteria, offering insights into metabolic risk even before overt hyperglycemia develops. Future longitudinal studies assessing first-trimester leptin levels and post-partum follow-up may clarify its predictive and prognostic significance. The present study findings underscore leptin's potential role as both a biomarker and a pathophysiological mediator in GDM and warrant further longitudinal and mechanistic investigations.

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REFERENCES

1. Roca-Rodríguez MDM, Ramos-García P, López-Tinoco C, Aguilar-Diosdado M. Significance of Serum-Plasma Leptin Profile during Pregnancy in Gestational Diabetes Mellitus: A Systematic Review and Meta-Analysis. J Clin Med. 2022 Apr 26; 11(9):2433. doi: 10.3390/jcm11092433.
2. Mousavi SN, Bahramfard T, Rad EY, Hosseinikia M, Saboori S. Association of Leptin and Retinol Binding Protein 4 with the Risk of Gestational Diabetes: A Systematic Review and Meta-Analysis of Observational Studies. Indian J Endocrinol Metab. 2023 Mar-Apr; 27(2):96-104. doi: 10.4103/ijem.ijem_385_22. Epub 2023 Apr 14.
3. Miehle K, Stepan H, Fasshauer M. Leptin, adiponectin and other adipokines in gestational diabetes mellitus and pre-eclampsia. Clin Endocrinol (Oxf). 2012 Jan; 76(1):2-11. doi: 10.1111/j.1365-2265.2011.04234.x.
4. Roca-Rodríguez MDM, Ramos-García P, López-Tinoco C, Aguilar-Diosdado M. Significance of Umbilical Cord Leptin Profile during Pregnancy in Gestational Diabetes Mellitus-A Systematic Review and Meta-Analysis. J Clin Med. 2023 Jul 18; 12(14):4756. doi: 10.3390/jcm12144756.
5. Maple-Brown L, Ye C, Hanley AJ, Connelly PW, Sermer M, Zinman B, Retnakaran R. Maternal pregravid weight is the primary determinant of serum leptin and its metabolic associations in pregnancy, irrespective of gestational glucose tolerance status. J Clin Endocrinol Metab. 2012 Nov; 97(11):4148-55. doi: 10.1210/jc.2012-2290.
6. Qiu C, Williams MA, Vadachkoria S, Frederick IO, Luthy DA. Increased maternal plasma leptin in early pregnancy and risk of gestational diabetes mellitus. *Obstet Gynecol.* 2004; 103(3):519–525. doi:10.1097/01.AOG.0000113629.01953.64.
7. Maged AM, Moety GA, Mostafa WA, Hamed DA. Comparative study between different biomarkers for early prediction of gestational diabetes mellitus. J Matern Fetal Neonatal Med. 2014 Jul; 27(11):1108-12. doi: 10.3109/14767058.2013.850489.
8. Lain KY, Catalano PM. Metabolic changes in pregnancy. Clin Obstet Gynecol. 2007 Dec; 50(4):938-48. doi: 10.1097/GRF.0b013e31815a5494.
9. Ng PC, Lam CW, Lee CH, Wong GW, Fok TF, Wong E, Ma KC, Chan IH. Leptin and metabolic hormones in infants of diabetic mothers. Arch Dis Child Fetal Neonatal Ed. 2000 Nov; 83(3):F193-7. doi: 10.1136/fn.83.3.f193.
10. Srivastava N, Singh K, Singh N, Mahdi AA. Association between serum interleukin-6, leptin and insulin in gestational diabetes mellitus - a cross-sectional study. J Diabetes Metab Disord. 2023 Feb 13; 22(1):639-648. doi: 10.1007/s40200-023-01188-3.
11. American Diabetes Association Professional Practice Committee. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2024. Diabetes Care. 2024 Jan 1; 47(Suppl 1):S20-S42. doi: 10.2337/dc24-S002.
12. International Diabetes Federation. IDF Diabetes Atlas, 11th Edition. Brussels, Belgium: International Diabetes Federation; 2022. Available at: https://diabetesatlas.org/.
13. Catalano PM, Shankar K. Obesity and pregnancy: mechanisms of short term and long term adverse consequences for mother and child. BMJ. 2017 Feb 8; 356:j1. doi: 10.1136/bmj.j1.
14. Fasshauer M, Blüher M, Stumvoll M. Adipokines in gestational diabetes. Lancet Diabetes Endocrinol. 2014 Jun; 2(6):488-99. doi: 10.1016/S2213-8587(13)70176-1.
15. Hauguel-de Mouzon S, Lepercq J, Catalano P. The known and unknown of leptin in pregnancy. *Am J Obstet Gynecol.* 2006; 194(6):1537–1545. doi:10.1016/j.ajog.2005.06.064.
16. Lappas M, Yee K, Permezel M, Rice GE. Release and regulation of leptin, resistin and adiponectin from human placenta, fetal membranes, and maternal adipose tissue and skeletal muscle from normal and gestational diabetes mellitus-complicated pregnancies. J Endocrinol. 2005 Sep; 186(3):457-65. doi: 10.1677/joe.1.06227.
17. Buchanan TA, Xiang AH. Gestational diabetes mellitus. J Clin Invest. 2005 Mar; 115(3):485-91. doi: 10.1172/JCI24531.
18. Cai Z, Yang Y, Zhang J. Hepatokine levels during the first or early second trimester of pregnancy and the subsequent risk of gestational diabetes mellitus: a systematic review and meta-analysis. Biomarkers. 2021 Sep; 26(6):517-531. doi: 10.1080/1354750X.2021.1928754.
19. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: Pathophysiology, Challenges, and Perspectives. Circ Res. 2019 Mar 29; 124(7):1094-1112. doi: 10.1161/CIRCRESAHA.118.313276. Erratum in: Circ Res. 2020 Jan 3; 126(1):e8. doi: 10.1161/RES.0000000000000315.