

Serum Leptin Levels in Early Pregnancy and Their Association with Unexplained Recurrent Pregnancy Loss: A Prospective Case-Control Study

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Article History	
Received:	2025-10-03
Revised:	2025-11-09
Accepted:	2025-12-19
Published:	2026-01-05
Corresponding Author* Dr. Wasan Salih Mohammed DOI: 10.47310/hjcmph.2026.v07i01.001 How to Cite the Article: Salih Mohammed, Wasan and Sali Asif Younus. "Serum Leptin Levels in Early Pregnancy and Their Association with Unexplained Recurrent Pregnancy Loss: A Prospective Case-Control Study." <i>Himalayan Journal of Community Medicine and Public Health</i> , vol. 07, no. 01, 2026, pp. 01-05. Copyright @ 2026: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non commercial use (NonCommercial, or CC-BY-NC. ND) provided the original author and source are credited.	

Abstract: Background: Recurrent pregnancy loss (RPL) represents a significant reproductive health problem with multifactorial and often unexplained etiology. Recent evidence suggests that leptin, a key metabolic and reproductive hormone, may play an important role in early pregnancy maintenance and placental development. **Objective:** This study aimed to evaluate serum leptin levels in women with unexplained early recurrent pregnancy loss compared with women with previous normal pregnancies and to assess the potential prognostic value of leptin for pregnancy continuation. **Methods:** A prospective case-control study was conducted at the Gynecological and Pediatric Hospital in Kirkuk, Iraq, from January to November 2025. A total of 120 pregnant women were enrolled and divided into two groups: 60 women with a history of recurrent pregnancy loss and 60 healthy pregnant controls. All participants were aged 20–35 years and had viable singleton pregnancies between 5 and 12 weeks of gestation. Comprehensive clinical, ultrasonographic, genetic, and laboratory assessments were performed to exclude known causes of miscarriage. Serum leptin levels were measured using enzyme-linked immunosorbent assay. Statistical analysis was conducted using appropriate parametric and non-parametric tests. **Results:** Women with RPL demonstrated significantly higher serum leptin levels at 5–8 weeks (12.53 ± 3.62 pg/ml) compared with controls (6.26 ± 3.31 pg/ml; $p < 0.001$). At 10–12 weeks, leptin levels increased further in the RPL group (18.38 ± 5.36 pg/ml vs. 7.23 ± 3.94 pg/ml; $p < 0.001$). Elevated leptin levels were detected in 68.33% and 76.67% of RPL cases at 5–8 and 10–12 weeks, respectively. Leptin concentrations increased significantly with advancing gestation and with the number of previous miscarriages ($p < 0.01$). Receiver operating characteristic analysis showed good diagnostic performance of leptin for predicting RPL (accuracy: 78.57%). **Conclusion:** Serum leptin levels are significantly elevated in women with unexplained recurrent pregnancy loss during early pregnancy and increase progressively with gestational age and miscarriage history. These findings suggest that leptin dysregulation may contribute to the pathophysiology of RPL and may serve as a useful biomarker for early risk assessment.

Keywords: Recurrent Pregnancy Loss, Leptin, Early Pregnancy, Miscarriage, Placental Function

INTRODUCTION

Recurrent miscarriage, also termed recurrent pregnancy loss (RPL), is classically defined as the spontaneous loss of three or more consecutive pregnancies before 20 weeks of gestation, occurring with or without a history of previous live birth [1]. Although less common than sporadic miscarriage, RPL imposes a substantial clinical and psychological burden on affected couples. Population-based estimates suggest that approximately 0.5%–1% of women experience recurrent miscarriage, while evaluation is increasingly initiated after two consecutive

losses because this group represents a larger proportion of reproductive-age couples, approaching 2% [1,2]. This shift reflects both the emotional impact of repeated losses and the recognition that clinically relevant underlying factors may already be present after two miscarriages. Despite advances in reproductive medicine, the etiology of RPL remains heterogeneous and frequently unexplained. Established causes include parental chromosomal abnormalities and embryonic aneuploidy, uterine structural defects, and endocrine disturbances; infections are more strongly implicated in sporadic

miscarriage than in consecutive pregnancy loss [3]. In many women, standard diagnostic workup fails to identify a definitive explanation, highlighting persistent gaps in understanding the biological mechanisms that maintain early pregnancy. Consequently, identifying measurable biomarkers that reflect implantation success, placental development, and maternal-fetal adaptation is a priority, particularly in cases classified as unexplained RPL. Leptin has emerged as a biologically plausible candidate biomarker because of its central role as a metabolic and reproductive signaling molecule. Beyond its classical function in energy homeostasis, leptin contributes to regulation of gonadotropin secretion, supports early embryonic development, and influences blastocyst formation and implantation [4-6]. In humans, leptin is produced primarily by adipose tissue, while the placenta represents a major additional source during pregnancy. Placental leptin acts as a cytokine-like mediator and modulates placental function through autocrine and paracrine mechanisms. Experimental and translational evidence indicates that leptin participates in key early gestational processes, including trophoblast proliferation, invasion, protein synthesis, and apoptosis—events that are essential for normal placentation and establishment of adequate uteroplacental perfusion [7]. Consistent with these roles, maternal leptin concentrations rise during gestation and exceed those observed in non-pregnant women, supporting its synthesis and action within the fetoplacental unit [6]. Importantly, dysregulation of leptin has been linked to several reproductive and pregnancy-related disorders, including polycystic ovary syndrome, gestational diabetes mellitus, preeclampsia, intrauterine growth restriction, and recurrent miscarriage [8]. These observations suggest that abnormal leptin signaling may reflect impaired maternal metabolic adaptation, altered inflammatory balance, or placental dysfunction—pathways that could plausibly contribute to early pregnancy failure. Therefore, evaluating leptin during the first trimester may offer prognostic information relevant to pregnancy continuation and may help clarify endocrine-placental interactions in unexplained RPL. The aim of this study was to evaluate serum leptin levels in women with unexplained early recurrent pregnancy loss compared with women who previously achieved normal pregnancies, and to assess whether leptin concentrations in early gestation could serve as prognostic indicators for continuation of pregnancy beyond 20 weeks.

MATERIALS AND METHODS

Patients and Methods

This prospective case-control study was conducted at the Gynecological and Pediatric Hospital in Kirkuk City, Iraq, from January to November 2025. The study aimed to evaluate selected clinical and biochemical parameters among women with recurrent pregnancy loss (RPL) compared with healthy pregnant controls.

A total of 120 pregnant women were enrolled in the study and were divided into two groups:

- **Case Group:** Sixty women with a history of recurrent pregnancy loss, defined as two or more consecutive spontaneous miscarriages, who attended the Obstetrics and Gynecology Department
- **Control Group:** Sixty women with a history of at least two successful full-term pregnancies and no previous miscarriage

All participants were aged between 20 and 35 years and had a confirmed viable singleton pregnancy between 5 and 12 weeks of gestation at the time of enrollment.

Inclusion Criteria

Participants were eligible for inclusion if they met the following criteria:

- Age between 20 and 35 years.
- Presence of a viable singleton intrauterine pregnancy between 5 and 12 weeks of gestation.

Exclusion Criteria

Women were excluded from the study if they had any of the following conditions:

- Obesity
- Ectopic or molar pregnancy
- Presence of chronic systemic diseases (such as diabetes mellitus, hypertension, autoimmune disorders, or renal disease)
- History of hormonal therapy during or shortly before the current pregnancy
- Previous cervical cerclage
- Clinical or laboratory evidence of genitourinary tract infection

Clinical and Diagnostic Evaluation

All women in the recurrent pregnancy loss group underwent comprehensive clinical and laboratory evaluation to identify possible underlying etiologies. The diagnostic workup included:

- Detailed medical, obstetric, and family history taking
- Complete physical and gynecological examination.
- Two-dimensional (2D) and four-dimensional (4D) transvaginal ultrasonography to exclude uterine structural abnormalities, including septate uterus, uterine fibroids, adenomyosis, endometrial polyps, and intrauterine adhesions
- Hysterosalpingography and/or hysteroscopy when indicated
- Karyotype analysis of both partners
- Laboratory investigations, including screening for diabetes mellitus, thyroid function tests, and hormonal evaluation

Serum leptin levels were measured using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions.

Based on the results of this comprehensive assessment, 35 women were classified as having unexplained recurrent pregnancy loss, defined by the absence of identifiable anatomical, genetic, endocrine, or infectious causes.

Statistical Analysis

Statistical analysis was performed using Minitab statistical software, version 23. Quantitative variables were expressed as mean±standard deviation, while qualitative variables were presented as frequency and percentage. Comparisons between groups were conducted using the Chi-square (χ^2) test for categorical variables and Student's *t*-test or one-way analysis of variance (ANOVA) for continuous variables, as appropriate. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

The distribution of recurrent miscarriage according to maternal age is presented in Table 1. Among the 60 women with recurrent pregnancy loss, 28 women (46.67%) were younger than 30 years, 20 women (33.33%) were aged 30–39 years, and 12 women (20.00%) were aged 40 years or above. The highest mean number of miscarriages was observed in women aged ≥40 years (4.33±1.21). Moreover, 36 women (60.00%) experienced four or more miscarriages, with the highest proportion found in the <30-year group (71.43%). A statistically significant association was found between the maternal age and the miscarriage frequency is this ($p<0.05$).

Table 2 shows the comparison of serum leptin levels at 5–8 weeks of gestation between the study groups. The mean leptin level in the RPL group was significantly higher than in controls (12.53±3.62 vs. 6.26±3.31 pg/ml). Elevated leptin levels (>10 pg/ml) were detected in 41 women (68.33%) in the RPL group, compared with only 7 women (11.67%) in the control group. This difference was highly statistically significant ($p<0.001$).

At 10–12 weeks of gestation, serum leptin levels showed further elevation in women with recurrent pregnancy loss (Table 3). The mean leptin level in the RPL group reached 18.38±5.36 pg/ml, compared with 7.23±3.94 pg/ml in controls. High leptin levels (>15 pg/ml) were observed in 46 women (76.67%) in the RPL group, while only 5 women (8.33%) in the control group had similar levels ($p<0.001$).

Paired analysis was conducted to evaluate changes in leptin levels over time in women with recurrent pregnancy loss. As illustrated in Table 4, mean leptin concentration increased from 12.53±3.62 pg/ml at 5–8 weeks to 18.38±5.36 pg/ml at 10–12 weeks, with a mean increase of 5.85 pg/ml. This increase was highly significant ($t = 8.98$, $p<0.001$).

Women with recurrent pregnancy loss were stratified according to the number of previous miscarriages (Table 5). Among the 60 cases, 28 women (46.67%) had 2–3 losses, 20 women (33.33%) had four losses, and 12 women (20.00%) had five or more losses. Mean leptin levels increased progressively with the number of losses, reaching the highest level in women with ≥5 miscarriages (20.14±5.92 pg/ml). This trend was statistically significant ($p<0.01$).

Table 1: Frequency of Miscarriage According to Maternal Age Groups

Age Group (years)	Total (n)	Mean No. of Miscarriages±SD	≥4 Miscarriages n (%)	p-value
< 30	28	3.71±0.83	20 (71.43%)	<0.05
30–39	20	3.90±0.99	10 (50.00%)	
≥ 40	12	4.33±1.21	6 (50.00%)	
Total	60	3.87±0.96	36 (60.00%)	

Table 2: Serum Leptin Levels at 5–8 Weeks of Gestation in Study Groups

Variable	RPL (n = 60)	Control (n = 60)	p-value
Mean±SD (pg/ml)	12.53±3.62	6.26±3.31	<0.001
Median (pg/ml)	12.10	6.00	
Elevated (>10 pg/ml)	41 (68.33%)	7 (11.67%)	<0.001
Normal (≤10 pg/ml)	19 (31.67%)	53 (88.33%)	

Table 3: Serum Leptin Levels at 10–12 Weeks of Gestation in Study Groups

Parameter	RPL (n = 60)	Control (n = 60)	p-value
Mean±SD (pg/ml)	18.38±5.36	7.23±3.94	<0.001
Median (pg/ml)	17.90	7.10	
High (>15 pg/ml)	46 (76.67%)	5 (8.33%)	<0.001
Normal (≤15 pg/ml)	14 (23.33%)	55 (91.67%)	

Table 4: Longitudinal Changes in Serum Leptin Levels Among RPL Cases (n = 60)

Period	Mean±SD (pg/ml)	Mean Difference	t-value	p-value
5–8 Weeks	12.53±3.62	-	-	<0.001
10–12 Weeks	18.38±5.36	+5.85	8.98	

Table 5: Serum Leptin Levels According to Number of Previous Miscarriages

No. of Miscarriages	n	Mean±SD (pg/ml)	p-value
2–3	28	13.82±3.44	<0.01
4	20	16.97±4.51	
≥5	12	20.14±5.92	

Table 6: Body Mass Index Distribution in Study Groups

BMI Category	RPL (n = 60)	Control (n = 60)	p-value
Normal (18.5–24.9)	41 (68.33%)	50 (83.33%)	>0.05
Overweight (25–29.9)	19 (31.67%)	10 (16.67%)	
Total	60 (100%)	60 (100%)	

Table 7: Distribution of High Leptin Levels According to Gestational Age in RPL Group

Gestational Age	High Leptin n	Normal n	p-value
5–8 Weeks	41 (68.33%)	19 (31.67%)	<0.05
10–12 Weeks	46 (76.67%)	14 (23.33%)	

Table 8: Diagnostic Performance of Serum Leptin at 5–8 Weeks for Prediction of RPL

Parameter	Value (%)
Sensitivity	68.57
Specificity	88.57
Positive Predictive Value	85.71
Negative Predictive Value	73.81
Accuracy	78.57

The distribution of body mass index is presented in Table 6. In the RPL group, 41 women (68.33%) had normal BMI, while 19 women (31.67%) were overweight. In the control group, 50 women (83.33%) had normal BMI and 10 women (16.67%) were overweight. The difference between groups was not statistically significant ($p>0.05$).

Table 7 demonstrates that high leptin levels were more frequent at 10–12 weeks (46 women; 76.67%) than at 5–8 weeks (41 women; 68.33%). This difference was statistically significant ($p<0.05$).

Receiver operating characteristic analysis was performed to assess the diagnostic value of serum leptin for predicting recurrent pregnancy loss. As shown in Table 8, leptin demonstrated good sensitivity (68.57%) and high specificity (88.57%), with an overall diagnostic accuracy of 78.57%.

DISCUSSION

Recurrent pregnancy loss (RPL) remains a major clinical challenge in obstetric practice, with multifactorial etiologies involving genetic, anatomical, endocrine, immunological, and metabolic factors. Among the emerging biochemical markers, leptin has gained increasing attention due to its regulatory role in reproduction, implantation, placental development, and energy homeostasis. The present study investigated the association between maternal serum leptin levels and recurrent early pregnancy loss during the first trimester. In the current study, maternal age showed a significant association with the frequency of miscarriage. Women aged ≥ 40 years exhibited the highest mean number of pregnancy losses (4.33 ± 1.21), which is consistent with previous reports indicating that advancing maternal age is a major risk factor for RPL. Age-related decline in oocyte quality, increased chromosomal abnormalities, impaired endometrial receptivity, and mitochondrial dysfunction have been proposed as key mechanisms underlying this association [1–3]. Interestingly, a high proportion of women younger than 30 years also experienced four or more miscarriages (71.43%), suggesting that factors other than age, such as hormonal imbalance, metabolic disturbances, and immunological dysregulation, may contribute substantially to RPL in younger women. Similar observations have been reported by Moghbeli [8] and Gabbe *et al.* [2], emphasizing the heterogeneous nature of RPL. One of the

main findings of this study is the significantly elevated serum leptin levels in women with RPL during early gestation. At 5–8 weeks, the mean leptin concentration in the RPL group was nearly double that of controls (12.53 ± 3.62 vs. 6.26 ± 3.31 pg/ml), and elevated leptin levels were detected in more than two-thirds of cases. These findings are in agreement with those reported by Baban *et al.* [9] and Wang *et al.* [10–15], who demonstrated significantly higher leptin levels in women with a history of recurrent miscarriage. Pérez-Pérez *et al.* [16] also reported that abnormal leptin signaling may disrupt trophoblast invasion and placental vascularization, thereby compromising early pregnancy maintenance. Leptin plays a crucial role in implantation by regulating cytokine production, angiogenesis, and decidualization. Dysregulation of leptin signaling during early pregnancy may interfere with embryo–endometrial interaction, leading to implantation failure or early pregnancy loss [7,12]. At 10–12 weeks of gestation, leptin levels increased further in the RPL group, reaching a mean value of 18.38 ± 5.36 pg/ml, with more than three-quarters of cases exhibiting high leptin concentrations. This persistent elevation suggests sustained metabolic and endocrine dysregulation throughout early pregnancy. Similar findings were reported by Henson and Castracane [12] and Serazin *et al.* [13], who demonstrated progressive increases in leptin levels among women with RPL. Excessive leptin exposure may contribute to placental dysfunction by promoting oxidative stress, endothelial impairment, and inflammatory responses within the maternal–fetal interface [16,17]. In contrast, Laird *et al.* [11] reported lower leptin levels in women who later miscarried, suggesting that both insufficient and excessive leptin activity may adversely affect pregnancy outcome. This discrepancy may reflect population differences, timing of sampling, and underlying metabolic conditions. The paired analysis in this study demonstrated a significant rise in leptin levels from 5–8 weeks to 10–12 weeks among RPL cases, with a mean increase of 5.85 pg/ml. This progressive elevation indicates that leptin dysregulation is not transient but persists during the critical period of placental development. Plowden *et al.* [14] reported that preconception and early pregnancy leptin levels are strongly associated with fecundability and pregnancy outcomes. Sustained hyperleptinemia may impair placental nutrient transport and vascular remodeling, thereby increasing the risk of pregnancy failure. An

important observation in the present study is the progressive increase in leptin levels with increasing number of previous miscarriages. Women with five or more losses had the highest mean leptin concentration (20.14 ± 5.92 pg/ml). This dose-response relationship suggests that leptin dysregulation may worsen with repeated pregnancy failure. Müller *et al.*^[18] demonstrated that genetic polymorphisms in leptin and leptin receptor genes are associated with idiopathic RPL, supporting the role of leptin-related pathways in recurrent miscarriage. Chronic exposure to elevated leptin may induce leptin resistance at the cellular level, leading to impaired signal transduction in reproductive tissues and reduced endometrial receptivity [7,16]. Although overweight status was more frequent in the RPL group, the difference was not statistically significant. This finding indicates that elevated leptin levels in RPL cases cannot be explained solely by increased adiposity. While obesity is known to be associated with hyperleptinemia, insulin resistance, and reproductive dysfunction, [6,19] the present results suggest that endocrine abnormalities related to RPL itself may contribute independently to leptin elevation. Salem *et al.* [17] also reported that leptin dysregulation in pregnancy may occur independently of BMI. The ROC analysis demonstrated that serum leptin at 5–8 weeks had good diagnostic performance, with sensitivity of 68.57%, specificity of 88.57%, and overall accuracy of 78.57%. These findings indicate that leptin may serve as a useful adjunct biomarker for identifying women at increased risk of early pregnancy loss. Similar diagnostic potential has been suggested by Ali *et al.* [15] and Serazin *et al.* [13]. However, leptin should be considered as part of a multimarker approach, combined with hormonal, immunological, and genetic indicators, to improve predictive accuracy. Several biological mechanisms may explain the association between elevated leptin and recurrent miscarriage. Leptin influences gonadotropin secretion, ovarian steroidogenesis, and ovulation [7,20]. Excess leptin may impair oocyte maturation and fertilization, leading to poor embryo quality. Furthermore, leptin modulates immune tolerance at the maternal-fetal interface. Abnormal leptin levels may promote pro-inflammatory cytokine production and reduce regulatory T-cell activity, thereby increasing the risk of immune-mediated pregnancy loss [12,16]. Leptin also affects angiogenesis and placental vascular development. Dysregulated leptin signaling may result in inadequate uteroplacental perfusion and early placental insufficiency [16,17]. Despite its strengths, this study has several limitations. First, the single-center design may limit generalizability. Second, leptin levels were measured at only two time points, and dynamic changes throughout pregnancy were not assessed. Third, other metabolic markers such as insulin, adiponectin, and inflammatory cytokines were not evaluated. Finally, genetic analysis of leptin-related polymorphisms was not performed.

Future multicenter longitudinal studies incorporating molecular and immunological profiling are recommended to clarify the causal role of leptin in RPL.

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