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Assessment of Clinical Outcomes of Maternal Leptin Level in Comparison among Three Groups: Women with GDM, Women with Preeclampsia, and Women in Normal Pregnancy

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Abstract: Background: Leptin, a pleiotropic adipokine tic metabolic and angiogenic hormone, is known to increase in the course of pregnancy. Its exact contribution and control in pathological pregnancies like gestational diabetes mellitus (GDM) and preeclampsia (PE) are yet to be completely clarified, especially when the confounding effect of maternal adiposity is factored in. Aim: The aim of the research was to compare maternal serum leptin levels in normal pregnancies and those complicated by GDM or preeclampsia, as well as to examine the relationship between hyperleptinemia and the pathological conditions, taking into account the impact of pre-pregnancy body mass index (BMI). Methodology: It was a cross-sectional study that was carried out on 70 pregnant women at their 3rd trimester (28-36 gestation). The sample was divided into three samples and groups: uncomplicated pregnancy (n=30), GDM (n=20), and PE (n=20). The level of leptin in the maternal serum was assessed, and the level of 25 ng/ml was considered to be high level of leptin. Demographic and clinical data was obtained, and statistical analyses were done in terms of group comparisons and Pearson correlation tests. Results Summary: The normal pregnancy group has a mean serum leptin level of 21.4 ± 7.2 ng/mL, that is lower than the GDM (28.9 ± 8.5 ng/mL) and PE (33.5 ± 10.1 ng/mL) groups. Hyperleptinemia was significantly higher overall in pathological pregnancies (52.5%) compared to normal controls (20.0%). Although pre-pregnancy BMI was a positive and significant correlate of leptin levels in all groups (r=0.76 in GDM, r=0.72 in PE), there was a higher proportion of high leptin levels in the pathological pregnancy groups in each of the BMI stratum. Also, leptin levels were strongly correlated with fasting insulin and HOMA-IR (r=0.71 and r=0.68, respectively) in the GDM group, and systolic BP, diastolic BP, and proteinuria (r=0.48, r=0.51, and r=0.59, respectively) in the PE group. Conclusion: We have shown that hyperleptinemia is strongly correlated with pathological pregnancies, namely, GDM and preeclampsia. Maternal BMI is one of the key factors leading to leptin levels, but the independent increase in these complications indicates that there is a likelihood of the pathophysiological effect of leptin, associated with insulin resistance in GDM and endothelial dysfunction in PE. Leptin has the potential to become a useful biomarker and should be considered as a potential therapeutic target.

Keywords: Leptin, Gestational Diabetes Mellitus, Preeclampsia, Pregnancy Complications, Adipokines, BMI, Insulin Resistance.

INTRODUCTION

Pregnancy is a complicated physiological condition which is associated with great metabolic, hormonal, and vascular changes which facilitate the growth and development of the fetuses. Of this variety of biochemical mediators, leptin, which is an adipocyte-derived hormone best described as an energy homeostatic hormone and an appetite regulator, has been found to be a significant participant in pregnancy biology. Leptin has not only an effect on maternal metabolism but also a role in placental metabolism in angiogenesis and in immune modulation, and hence affects maternal and fetal health [Miehle, K. *et al*., 2012; Masuzaki, H. *et al*., 1997; Hauguel-de Mouzon, S. *et al*., 2006].

Normal pregnancy is associated with a massive rise in leptin levels in the blood, peaking in the third trimester. This is due to both maternal adiposity and placental leptin. [Herse, F. *et al*., 2009; Laivuori, H. *et al*., 2006] It is postulated that the high leptin plays a role in maternal adaptation by maintaining energy balance as well as promoting angiogenesis to feed the placenta. Nevertheless, dysfunctional leptin-induced processes in pregnancy have been the cause of pregnancy complications, including gestational diabetes mellitus (GDM) and preeclampsia (PE), which have been the major cause of maternal and perinatal morbidity and mortality in the world [Hendler, I. *et al*., 2005; Masuyama, H. *et al*., 2010].

Leptin is a protein hormone with a molecular weight of 16 kDa, circulating in the bloodstream as a monomer of tertiary structure with a closed intramolecular disulfide bond and consisting of 167 amino acids. Leptin is secreted primarily by adipocytes of adipose tissue, as well as by adipocytes of the bone marrow, fetal tissues of the heart, bone, and, during pregnancy, by the trophoblast of the placenta and amnion cells

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[Lepercq, J. *et al*., 2007; Laml, T. *et al*., 2001; Kauma, S. *et al*., 2002; Díaz, E. *et al*., 2002]. The level of leptin in the blood reflects the total energy reserve of adipose tissue [Bartha, J. L. *et al*., 2001; Salvatores, M. *et al*., 2006]. Leptin is essential for the development of pregnancy, since for trophoblast cells it is a trophic and mitogenic factor that reduces apoptotic activity and stimulates proliferation [McCarthy, J. F. *et al*., 1999; Adali, E. *et al*., 2009]. Leptin stimulates the synthesis of fetal fibronectin, matrix metalloproteinases 2 and -9, and integrin, thereby imparting an invasive phenotype to cytotrophoblast cells [Ibrahim, H. S. *et al*., 2013; Matarese, G. *et al*., 2005]. Leptin also promotes placental angiogenesis by synergistically interacting with several growth factors, including vascular endothelial growth factor, basic fibroblast growth factor, and platelet-derived growth factor B [Redman, C. W., & Sargent, I. L. 2010; Acromite, M. *et al*., 2004].

MATERIAL AND METHOD

The reasons why this cross-sectional observational study was carried out were to determine the maternal serum leptin in normal pregnant women, gestational diabetes mellitus (GDM), and preeclampsia (PE). The institutional review board allowed the study to have ethical approval, and all the participants were informed to participate in the study through a written informed consent before enrollment.

Study Population

There was a recruitment of seventy pregnant women (28 to 36 weeks of gestation) consecutively recruited at the antenatal clinic. The subjects were divided into three groups namely normal uncomplicated pregnancy (n=30), GDM (n=20) and PE (n=20) from different hospitals from Iraq with study period from 1-11-2024 to 2-10-2025 where The diagnosis of GDM and PE was based on clinical guidelines Women who had chronic hypertension, pre-existing diabetes or renal disease as well as those who had other systemic illness were excluded.

Clinical and Demographic Data Collection.

Medical records and interviews with the participants provided data regarding maternal age, gestational age at sampling, pre-pregnancy body mass index (BMI), and parity. The calculation of BMI before pregnancy was done by taking the weight per kilograms divided by the height in meters squared (kg/m2), and the result was then classified as normal weight (<25), overweight (2529.9), and obese.(30≤)

Blood Sampling and Leptin Assay.

Blood samples of the maternal vein were taken on one occasion during the third trimester following an fast overnight. Separating was done on serum and stocked at -80 °C. Mothers' serum leptin was detected by the enzyme-linked immunosorbent assay (ELISA) kit based on the protocol of the manufacturer. The cutoff of over 25 Ng/mL was considered the high level of leptin according to previous literature and calibration of the assay.

Metabolic and Clinical Parametric Evaluation.

Fasting glucose and insulin levels were assessed in the GDM group, and the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) was used to estimate the insulin resistance. To measure the severity of the disease, blood pressure (systolic and diastolic), and 24-hour proteinuria were taken in preeclamptic patients.

Statistical Analysis

Statistical software (SPSS version 22) was applied to analyze the data. Continuous variables are reported as mean SD (or median), as necessary. The frequencies and percentages report categorical variables. One-way ANOVA or Kruskal-Wallis test of non-parametric data were used to perform group comparisons of leptin levels and clinical parameters, and then the post hoc test. The relationship between leptin high levels and pathological pregnancy (combined GDM and PE) was analyzed through the chi-square test.

The correlation coefficient (r) of Pearson was applied to observe the relations of leptin and metabolic or clinical parameters in each of the pathological groups. The level of statistical significance was defined as $p < 0.05$.

RESULTS

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Table 1: Demographic and Clinical Characteristics of the Study Cohort (N=70)

Characteristic	Total Cohort (N=70)	Normal Pregnancy (n=30)	GDM (n=20)	Preeclampsia (n=20)
Maternal Age (years), Mean ± SD	29.5 ± 4.8	28.1 ± 3.9	30.9 ± 5.1	30.2 ± 5.0
Gestational Age at Sampling (wks), Mean ± SD	31.2 ± 2.1	31.5 ± 1.9	31.0 ± 2.0	31.0 ± 2.5
Pre-pregnancy BMI (kg/m²), Mean ± SD	26.8 ± 5.2	24.5 ± 3.8	28.1 ± 4.9	28.9 ± 6.1
Primigravida, n (%)	28 (40.0%)	14 (46.7%)	7 (35.0%)	7 (35.0%)

Table 2: Distribution of High Leptin Levels (>25 ng/mL) by Pregnancy Group

Group	Total n	n with High Leptin	% with High Leptin
Normal Pregnancy	30	6	20.0%
GDM	20	9	45.0%
Preeclampsia	20	12	60.0%
Total Cohort	70	27	38.6%

Table 3: Mean Leptin Levels by Pregnancy Group

Group	n	Mean Leptin (ng/mL) ± SD	Median Leptin (ng/mL)
Normal Pregnancy	30	21.4 ± 7.2	20.1
GDM	20	28.9 ± 8.5	27.3
Preeclampsia	20	33.5 ± 10.1	31.8
Total Cohort	70	26.8 ± 9.9	25.0

Table 4: Association Between High Leptin and Pathological Pregnancy (Composite of GDM & PE)

Leptin Category	Normal Pregnancy (n=30)	Pathological Pregnancy (GDM+PE, n=40)	Total
High Leptin (>25 ng/mL), n (%)	6 (20.0%)	21 (52.5%)	27
Normal Leptin (≤25 ng/mL), n (%)	24 (80.0%)	19 (47.5%)	43
Total	30	40	70

Table 5: Leptin Levels Stratified by Pre-pregnancy BMI Category (Whole Cohort)

BMI Category	n	Mean Leptin (ng/mL) ± SD	n with High Leptin	% with High Leptin
Normal Weight (BMI <25)	25	19.8 ± 6.1	3	12.0%
Overweight (BMI 25-29.9)	28	27.5 ± 8.3	11	39.3%
Obese (BMI ≥30)	17	36.2 ± 9.8	13	76.5%

Table 6: Interaction of Pregnancy Pathology and BMI Status on High Leptin

Group	Normal Weight (n=25)	Overweight (n=28)	Obese (n=17)
	n High Leptin / Total n (%)	n High Leptin / Total n (%)	n High Leptin / Total n (%)
Normal Pregnancy	1/10 (10.0%)	3/12 (25.0%)	2/8 (25.0%)
GDM	1/7 (14.3%)	4/9 (44.4%)	4/4 (100%)
Preeclampsia	1/8 (12.5%)	4/7 (57.1%)	7/7 (100%)

Table 7: Leptin Levels by Parity

Parity	Total n	Mean Leptin (ng/mL) ± SD	n with High Leptin	% with High Leptin
Primigravida	28	28.9 ± 10.5	13	46.4%
Multigravida	42	25.3 ± 9.2	14	33.3%

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Table 8: Correlation (Pearson's r) of Leptin with Metabolic Parameters in GDM Group (n=20)

Parameter	Correlation with Leptin (r value)	p-value
Fasting Glucose	0.52	<0.05
Fasting Insulin	0.71	<0.01
HOMA-IR	0.68	<0.01
BMI	0.76	<0.001

Table 9: Correlation (Pearson's r) of Leptin with Clinical Parameters in Preeclampsia Group (n=20)

Parameter	Correlation with Leptin (r value)	p-value
Systolic BP	0.48	<0.05
Diastolic BP	0.51	<0.05
Proteinuria (24h)	0.59	<0.01
BMI	0.72	<0.001

DISCUSSION

Table 1 shows the initial demographic data of the three study groups by showing that there are no statistically significant differences in terms of maternal age (28.130.9 years), gestational age at sampling (31.031.5 weeks), or primigravida status (3546.7%). Women with normal pregnancies had the lowest pre-pregnancy body mass index (BMI) (24.5kg/m 2) than those with gestational diabetes mellitus (GDM) (28.1kg/m 2) and preeclampsia (PE) (28.9kg/m 2), which indicates that high adiposity is a risk factor of complications despite similar ages and gestation timing. The even distribution of the covariates across all the cohorts eases the confounding by age or by gestational timing, thereby increasing the validity of the intergroup comparisons and exaggerating the contribution of BMI in the pathophysiology of adverse pregnancy result.

In normal pregnancies, high leptin levels (>25ng/mL) were found in 20%, with the percentage rising to 45% in GDM and 60% in PE. High concentrations of leptin gave an overall pathological pregnancy rate of 52.5%. This linear increase demonstrates that hyperleptinemia is more closely connected with obstetric complications, and PE has the highest prevalence despite the fact that both groups were equally represented (n = 20). The figures thus confirm the discriminatory ability of leptin in that almost three times the number of normal pregnancies were able to support levels 25ng/mL and less.

Normal pregnancies had a mean leptin of 21.4 -1 mL, which was increased to 28.9- -1 mL in GDM and 33.5 -1 mL in PE, with respective medians of 20.1, 27.3, and 31.8 -1 mL. These progressively larger values were coupled with the higher standard deviations in the pathological groups (7.2 vs 8.510.1 ng/mL), which can be seen as increased variability and dysregulation in GDM and PE. The results are consistent with the known third-trimester leptin peak that is increased by the placenta and adipose tissue in an adverse environment.

The current research paper outlines major variances in maternal serum leptin levels in women who have normal pregnancy and gestational diabetes mellitus (GDM) and preeclampsia (PE). In general, the level of leptin was lowest during normal pregnancy and had an increasing tendency between the GDM and PE groups, which indicated a strong relationship between hyperleptinemia and these pathological conditions.

The physiological rise of leptin is entirely documented to be a vital adaptation measure aiding energy homeostasis and placental angiogenesis, which occurs in normal pregnancy [Dalamaga, M. *et al*., 2011; Kaaja, R. *et al*., 2004]. The observed elevated leptin levels in GDM and PE, even after controlling for the pre-pregnancy BMI, is evidence of the possible pathophysiological role of leptin other than adiposity. This elevation is also probably the result of an intricate interplay of insulin resistance and endothelial dysfunction, which are the hallmarks of GDM and PE, correspondingly.

In the BMI groups, the mean leptin in normal weight (12% high) of 19.8 ng/mL decreased to 27.5 ng/mL (overweight, 39.3% high) and 36.2 ng/mL (obese, 76.5% high) in the whole cohort .

Pathological groups had more prevalence with the normal weight (1014.3%), overweight (2557.1%), and obese (25100%). PE always dominated all strata (e.g., 100 percent in obese), then GDM, versus controls. This interaction suggests that pathology enhances this BMI effect of leptin, suggesting that disease-specific pathways - which could be inflammatory pathways - dominate the

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increase in leptin concentrations in proportion to adiposity.

The mean leptin levels between primigravidas (n 28) and multigravidas (25.3 ng/ml, 33.3% high) were higher in the former (28.9ng/ml) than in the latter. It is an unexpected trend and possibly indicates that metabolic requirements are higher or leptin resistance is less in first pregnancies, thus predisposing to complications. Therefore, in this manner, parity is revealed as a modulatory factor which should be taken into consideration in future analytic models.

In GDM, leptin was greatly associated with fasting insulin and homeostatic model assessment of insulin resistance (HOMA-IR), which appraises the idea that leptin could be a contributor or indicator of the mechanisms that prompt insulin resistance [Bodnar, L. M. *et al*., 2007]. The results agree with the previously known modulatory functions of leptin in glucose metabolism, adipocyte activity, and inflammatory pathways in the pathogenesis of GDM. The high level of leptin can therefore be the cause of insulin resistance or rather act as a biomarker of disturbed metabolic milieu in the affected pregnancies.

On the other hand, leptin was linked to blood pressure parameters and proteinuria, that are important clinical indicators of endothelial damage and vascular dysfunction in the PE cohort. This confirms the hypothesis that leptin can control or be a marker of placental vascular pathology and systemic endothelial dysfunction of preeclampsia. The angiogenic properties and implication in inflammatory cascades of leptin are likely to cause the vascular maladaptation that is seen [Bodnar, L. M. *et al*., 2005].

It is provided that the strong positive relationships between pre-pregnancy BMI and leptin in all groups support the classic effect of adiposity in increasing leptin levels. However, the upwardly skewed increased levels of leptin in pathological pregnancies in all BMI groups indicate that some other pregnancy-specific pathways are at play in GDM and PE that augment leptin production or diminish leptin clearance [Farley, D.M. *et al*., 2010; Haggerty, C. L. *et al*., 2013].

The differences related to parity, which are higher in primigravidas, could be associated with increased physiological need or a unique adaptive reaction to new pregnancies that, in turn, could contribute to the predisposition to metabolic and hypertensive complications.

These findings are clinically in support of leptin as an attractive biomarker in the early diagnosis and distinction of pregnancy complications

CONCLUSION

The study has shown that the maternal serum leptin levels are significantly high during pregnancies with gestational diabetes mellitus and preeclampsia, compared to normal pregnancies. This hyperleptinemia is highly correlated with pre-pregnancy body mass index, but it builds up independently of the disease states, thus creating emphasis on the multi-faceted nature of leptin, other than adiposity. The positive relationships between leptin and insulin resistance markers in gestational diabetes and blood pressure and proteinuria markers in preeclampsia indicate that it is involved in the underlying metabolic and vascular pathophysiology of these conditions. These results, therefore, support leptin as a potential biomarker in the detection and the mechanistic explanation of pregnancy-related complications and the potential in leptin as a therapeutic endpoint to enhance maternal and fetal health outcomes.

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