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Research Article

Association of Maternal and Cord Blood IGF-I with Leptin Levels in Preeclampsia

Panjeta P, Ghalaut VS, Bala J, Nanda S* and Kharb S

Department of Biochemistry, Obstetrics and Gynecology, Rohtak, Haryana, India

***Corresponding author:** Simmi Kharb, Department of Biochemistry, Obstetrics and Gynecology, Rohtak, Haryana, India

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Abstract

Preeclampsia is a leading cause of maternal and neonatal mortality and morbidity and the cause of preeclampsia is still not clear. IGF is an important mediator of both placental and fetal development. Also, leptin has been measured in the fetal circulation and plasma leptin may also originate from a range of fetal and placental tissues. The present study was designed to analyze IGF-I and leptin levels in maternal and cord blood of women with preeclampsia and to assess its relationship with outcome of pregnancy. Twenty five normotensive pregnant women and 25 age- and gestation- matched preeclamptic women were selected. Routine investigations and serum IGF-I and leptin levels were analyzed in maternal and cord blood by Elisa kit based on the sandwich principle. Serum IGF-I levels of preeclamptics were significantly decreased as compared to normotensive mothers (p=0.000). Serum leptin levels were significantly higher in preeclamptics as compared to normotensive pregnant women. Maternal IGF-1 had a strong inverse correlation with leptin levels in preeclamptics (r=-0.528, p=0.007). Positive correlation was seen between normotensive mother IGF-1 and leptin levels (r=0.226, p=0.278). Findings of present study suggest that IGF-I and leptin are associated with rise in blood pressure in preeclampsia. Mechanisms responsible for this change and role played by IGF-I and leptin in the development of preeclampsia require further study.

Keywords: IGF-I; Leptin; Cord blood; Birth weight; Pregnancy; Preeclamptics

Introduction

Pre-eclampsia is a principal cause of maternal morbidity and mortality, and occurs in 3–10% of pregnancies worldwide [1]. In preeclamptic women, the amount of substrates available for fetal growth is limited by chronic uteroplacental ischemia. To compensate for the limited blood flow to placental and fetal tissues, the fetus signals placental release of antiangiogenic factors for increasing maternal blood pressure. The collective magnitude of angiogenic imbalances, gene–environment interaction and other factors would determine whether a patient with chronic trophoblast ischemia presents with pre-eclampsia, fetal growth restriction or both.

In late-onset pre-eclampsia, there is an increased fetal demand for substrate that surpasses the placental ability to sustain fetal growth may induce fetal signaling for placental overproduction of anti-angiogenic factors and subsequent 'compensatory' maternal hypertension [2]. It has been suggested that there are increased fetal demand for substrates in late-onset pre-eclampsia which surpass the placental ability to sustain fetal growth. The increased demands may in turn induce fetal signaling for placental overproduction of anti-angiogenic factors and subsequent 'compensatory' maternal hypertension [2].

The role of the fetus in the maternal manifestations of pregnancy complications deserves more attention. Recent evidence suggests that the fetus may play a central role in the clinical manifestations of pre-eclampsia which might have fetal survival value in the context of uteroplacental ischemia. Fetal growth and development are closely regulated by the paracrine and autocrine actions of various growth factors such as the Insulin-Like Growth Factors (IGF), fibroblast growth factors, epidermal growth factors, transforming growth factors and platelet derived growth factors. These growth-related factors do not cross the placental barrier, but may affect fetal growth through their effects on the placenta [3]. Placental Growth Hormone (GH) is secreted by the syncytiotrophoblast and is responsible for the gradual rise in serum IGF-I concentrations during the second half of pregnancy [4,5]. Circulating IGFBP-1 increases from early pregnancy onward and is produced by both liver and deciduas. Several studies have demonstrated lower circulating levels of IGF-I, but higher levels of IGFBP-1, during the third trimester of pregnancies complicated by IUGR, in particular pregnancies with a deficient uteroplacental supply line. Maternal serum IGFBP-1 was also found to correlate negatively with birth weight in normal and diabetic pregnancies [6].

The availability of these growth factors is controlled not only by gene expression, but also by proteolytic release. IGF-I and IGF-II circulate in association with specific binding proteins (IGFBPs), and their bioavailability depends on the proteolysis of the specific IGFBPs. IGF-I is believed to be the primary hormone influencing fetal growth in later gestation and is essential for placental and fetal development. The targeted gene deletion of the IGF-I gene in mice is shown to yield homozygote that have a birth weight about 60% that of normal [3]. During pregnancy, IGF-I concentrations parallel the increase in fetal size that occurs with advancing gestation [7]. There is also evidence to indicate an inverse relationship between increased IGFBP-1 concentrations at delivery and birth size [8].

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Table 1: Maternal IGF-1 and Leptin levels in both the groups (mean± SD).

	Group I (Control)	Group II (Study)
IGF-1 (ng/ml)	259±45.39	73.2±48.69*
Leptin (ng/ml)	21.77±6.30	57.48±18.67*

*p<0.001 as compared to group I.

Conflicting data are available regarding IGF-1 and leptin in preeclamptic mothers. Hence the present study was planned to assess IGF-1 and leptin levels in maternal blood of preeclamptics and to compare them with normotensive pregnant women.

Materials and Methods

The present study was conducted in the Department of Biochemistry in collaboration with Department of Obstetrics and Gynaecology, Pt. B.D. Sharma, PGIMS, Rohtak. Fifty pregnant women attending the Outpatient department of Obstetrics and Gynaecology were enrolled and divided into two groups: Group I (control, n=25) normotensive women with singleton pregnancy at the time of delivery; and Group II (study, n=25) age and gestation matched women with singleton pregnancy and systolic blood pressure ≥140mm Hg or diastolic blood pressure ≥90mm Hg with or without proteinuria at the time of delivery.

An informed consent was taken from all the patients. Women with history of chronic hypertension, any metabolic disorder before or during pregnancy or presence of high risk factors like heart disease, diabetes, renal disease were excluded.

Five ml blood was drawn aseptically and serum was separated by centrifugation. Routine investigations and serum leptin and IGF-1 levels were analyzed in maternal and cord blood of women with preeclampsia and normotensive pregnant women. DRG ELISA kit for IGF-1and leptin ELISA Kit for leptin estimation was based on is solid phase enzyme-linked immunosorbent assay (ELISA, based on the sandwich principle) [9].

Results

Serum IGF-I levels of preeclamptics were significantly decreased as compared to normotensive mothers (p=0.000, (Table 1)). Serum leptin levels of preeclamptic mothers were significantly increased as compared to normotensive mothers (p=0.000). Cord blood IGF-1 levels were significantly decreased in preeclampsia mothers as compared to normotensive mothers (72.2±28.65 *vs*. 33.2±22.21ng/ml, p=0.000), while cord blood leptin levels were significantly increased in preeclampsia mothers as compared to normotensive mothers (10.02±4.57 *vs*. 24.27±5.64 ng/ml, p=0.000).

Maternal IGF-1 had a strong inverse correlation with leptin levels in preeclamptics (r=-0.528, p=0.007). Positive correlation was seen between normotensive mother IGF-1 and leptin levels (r=0.226, p=0.278), however it was not statistically significant. Cord blood IGF-1 had a negative correlation with cord blood leptin levels in both the groups but, it was statistically not significant in neither of these groups (r=-0.145, p= 0.490; r=-0.246, p=0.236).

IGF-1 had a positive correlation with blood pressure in maternal blood in group I (r=0.108, p=0.607). In group II, IGF-1 had inverse correlation with maternal blood pressure (r=-0.345, p=0.092). None of these correlations were significant. IGF-1 was negatively correlated with blood sugar in both normotensive group (r=-0.123, p=0.558) and preeclamptics (r=-0.310, p=0.131), although none was statistically significant. Maternal IGF-1 has a strong negative correlation with urinary albumin levels amongst group II hypertensive mothers (r=-0.531, p=0.006) and was statistically significant.

Systolic blood pressure in preeclamptic group showed a significant positive correlation with serum leptin (r=0.642, p=0.001) in normotensive group it was also positively correlated with serum leptin levels (r=0.026, p=0.903).

IGF-I levels showed a positive correlation with lipid profile in both normotensive pregnant women and preeclamptics and it was inversed for HDL cholesterol in preeclamptics though it was not statistically significant in all the cases (for HDL in preeclamptics, r=-0.097, p=0.644 as compared with HDL in normotensive women, r=0.270; p=0.193).

A significant positive correlation was observed between maternal leptin and serum cholesterol and LDL-C (r=0.479, p=0.015; r= 0.461. p=0.020 respectively) in normotensive pregnant women and it was inversed in preeclamptics (r=-0.230, p=0.269; r= -0.253, p=0.222 respectively).

Discussion

IGF system is an important mediator of both placental and fetal development and increased IGF-I and IGF-II levels are detectable in the preeclamptic fetoplacental unit [6-8]. The present study showed that serum IGF-1 levels were significantly decreased in preeclamptics as compared to the normotensive mothers (259±45.39 *vs*. 73.2±48.69 ng/ml).

Clinical studies have provided conflicting evidence regarding alterations of IGFs in preeclamptic pregnancies. Vatten et al observed an increase in IGF-1 from the first to second trimester associated with preeclampsia supposed to be the result of placental disease [10]. Contradicting these findings, others have reported decreased IGF-1 levels during pregnancy associated with the risk of developing preeclampsia [11,12]. Few workers have reported that IGF-1 levels are not changed in preeclampsia [13,14]. Wilson et al reported higher concentration of IGF-1 in the third trimester, probably reflecting placental contribution [15].

In the present study, cord blood IGF-1 levels were significantly decreased in preeclampsia mothers as compared to normotensive mothers (72.2±28.65 *vs*. 33.2±22.21 ng/ml, p=0.000). No reports are available in literature regarding cord blood IGF-1 levels in preeclampsia.

A strong inverse correlation was observed between maternal IGF-1 with leptin levels in preeclamptics (r=-0.528, p=0.007) and positive correlation was noted between normotensive mother IGF-1 and leptin levels (r=0.226, p=0.278), though it was statistically not significant. The present study showed that there was reversal of the correlation between IGF-I and leptin in preeclamptics. However, few reports have shown that no correlation exist between IGF-1 and leptin [16,17].

Maternal IGF-1 has a strong negative correlation with urinary albumin levels amongst group II hypertensive mothers (r=-0.531,

p=0.006). Haemodynamic factors and permselectivity properties of the glomerular filtration barrier govern the glomerular passage of albumin. IGF-1 receptors are present on all type of glomerular cells, and are responsible for in vivo effects of IGF-1 on glomerular function. IGF-1 infusion rapidly increases GFR, renal plasma flow and albuminuria in human subjects probably by causing glomerular vasodilation and a rise in the ultrafiltration coefficient. Evidence of a direct effect of IGF-1 on glomerular protein handling is not yet available, but IGF-1 could alter glomerular permselectivity properties by relaxing the mesangium. It is evident that systemic blood pressure can influence albuminuria. In patients with Insulin dependent diabetes mellitus, a rise in blood pressure and progression of albuminuria develop concomitantly. Macroalbuminuria has also been observed in essential hypertension, albeit at higher blood pressure levels [18]. Iftikhar et al also showed that leptin has a strong positive correlation with urinary albumin levels in both maternal and cord blood levels amongst hypertensive mothers [19].

In the present study, IGF-1 was negatively correlated with blood sugar in both normotensive group (r=-0.123, p=0.558) and preeclamptics (r=-0.310, p=0.131), although none was statistically significant. Mean birth weight in group I and group II was 2.59±0.46 kg and 2.48±0.53 kg respectively. Majority of the babies had birth weight between 2.1-3.5 kg in 92% of normotensive pregnant and 76% in mothers with preeclampsia. 20% babies of hypertensive mother had birth weight between 1.6-2.0 kg, whereas, no babies in the control group had birth weight <1.5 kg. Thus, birth weight was lesser in preeclamptics as compared to normotensive women though it was not statistically significant (p=0.404). IGF-1 showed a positive correlation with birth weight in both normotensive (r=0.134, p=0.523) as well as preeclamptic groups (r=0.287, p=0.164), though it was not statistically significant. Halhali et al showed that maternal and umbilical cord serum IGF-I correlated significantly ($p < 0.05$) with weight and length at birth [20]. Previous reports have shown that IGF-I and IGF-II correlated well with birth weight. In non-pregnant state, IGFBP-1 regulate the availability of IGF-I [21]. In pregnancy, relationship of IGF-I with fetal size suggests a similar role, as IGFBP-I showed a significant negative correlation with birth weight.

Insulin Receptor (IR) and type 1 and type 2 Insulin-Like Growth Factor Receptors (IGF1R and IGF2R) regulate placental and fetal growth and content of specific N-glycans attached to them gets altered during pregnancy. Glucose transporter GLUT2 also undergoes alteration of N-glycosylation and its glucose transport activity changes. Altering N-glycosylation of the GLUT2 glucose transporter prevents its anchoring and retention at the cell surface; this impairs glucose uptake and insulin secretion. During first trimester, placental IR is predominantly expressed on apical membrane of syncytiotrophoblast and at term it is expressed on the basal membrane of syncytiotrophoblast. IR expression shifts from plasma membrane facing maternal circulation to that facing fetal circulation during pregnancy. Altered IGF signalling results in aberrant placental growth [21].

A small increase in blood glucose levels is detrimental to the developing embryo and fetal exposure to hyperglycemia has been associated with congenital abnormalities, such as Congenital Heart Disease (CHD). Good glycaemic control has been shown to reduce the rate of congenital abnormalities, preterm delivery and stillbirths. Congenital heart disease patients have been reported to have lower plasma cholesterol concentrations and higher serum glucose levels than non-congenital ones [22].

There was no significant correlation between IGF-1 and lipid profile. There are no reports in literature where IGF-1 and lipid profile have been correlated.

A significant positive correlation was observed between maternal leptin and serum cholesterol and LDL-C (r=0.479, p=0.015; r= 0.461. p=0.020 respectively) in normotensive pregnant women and it was inversed in preeclamptics (r=-0.230, p=0.269; r=-0.253, p=0.222 respectively). Leptin production has been reported to be dysregulated in pathologic states of pregnancy and overproduction of leptin is associated with alteration of fetal growth [6,9]. Combination of a small or compromised placenta and poor fetal growth is accompanied by lower levels of leptin in maternal blood.

Reduced placental perfusion pressure results in intrauterine growth restriction in offspring born of preeclamptic pregnancies [23]. Clinical studies have reported a deficit in circulating and cord blood IGF-1 levels in intrauterine growth restricted newborns and a correlation between IGF-1 levels and birth weight [24,25]. High maternal levels of IGF-1 regulate fetal weight and divert nutrients from mother to fetus, while maternal IGF-2 regulates placental development and maternal hemodynamic adaptation to pregnancy by increasing the maternal plasma volume [25,26]. Wang et al showed that mean serum IGF-1 levels in the Small-for-Gestational-Age (SGA) group were significantly higher than those in Average-for-Gestational-Age (AGA) neonates [27]. However, their study showed that maternal serum IGF-1 had no association with birth weight and there was no significant difference between the SGA and AGA groups. Similarly Bankowski et al also showed that preeclampsia is associated with decrease of IGF-1 content in the umbilical cord artery [28]. Our study also showed that maternal IGF-1 has no significant correlation with birth weight in preeclampsia. On the other side, Halhali et al showed that maternal IGF-I correlated significantly ($p < 0.05$) with weight and length at birth [29]. Reports of positive correlation of birth weight and leptin amongst normotensive pregnancies are available in literature [30].

The present study showed that IGF-1 had inverse correlation with blood pressure in preeclamptics. Janssen at al also showed that IGF-1 has a negative correlation with systolic blood pressure [31]. IGF-1 is an important regulator of cardiovascular function in the non-pregnant state and studies have shown that low IGF-1 levels are associated with endothelial dysfunction in hypertensive patients [32,33]. Thum and colleagues showed that IGF-1, and no GH, is directly involved in cardiovascular function, such as blood pressure [34].

The findings of present study indicate the role of leptin in pathogenesis of preeclampsia and also it can serve as a marker of fetal adiposity. Thus, deregulation of the tuned balance among IGF system components plays a crucial role in the pathogenesis of preeclampsia. Quantification of maternal IGF and leptin levels may serve as a predictive screening test for risk of developing preeclampsia alone or in combination with other known independent indicators of preeclampsia risk.

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