Research Journal of Obstetrics & Gynecology

ISSN 1994-7925

science alert

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Research Journal of Obstetrics and Gynecology 5 (1): 4-7, 2012
ISSN 1994-7925 / DOI: 10.3923/rjog.2012.4.7

Cord Blood Atherogenic Index in Preeclampsia: Tracing Fetal Origins of Adult Disease

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ABSTRACT

Maternal physiology sets the fetal environment and maternal health status during pregnancy significantly affects the fetal growth in uterus. Since birth weight is summary measure of fetal growth and its determinants include genetic growth potential, length of gestation and maternal size and supply line to baby (in terms of metabolic and nutritional status of mother, uteroplacental perfusion and placental function). Hence, the present study was planned to study metabolic derangements in preeclampsia by analyzing cord blood Atherogenic Index (AI) in normal pregnant and preeclamptic women and to correlate them with birth weight and maternal AI. Study group consisted of 50 women with preeclampsia (group II) and 50 health normotensive pregnant women served as controls (group I). Serum Apo A-I and Apo B levels were analyzed by immunoturbidimetric immunoassay kits and atherogenic index was calculated. Apo B levels were significantly elevated in group II in both cord blood and maternal blood as compared to group I. Apo A-I. levels were lower in maternal blood and higher in cord blood of group II. a negative correlation was observed between cord blood AI and birth weight in group II. While, group I showed a positive correlation between cord blood AI and birth weight. These findings lend support to the hypothesis that roots for adult heart disease begins in utero programming.

Key words: Atherogenic index, cord blood, preeclampsia, birth weight

INTRODUCTION

Normal pregnancy is atherogenic especially in third trimester and this is a transient disturbance which reverts to normal after delivery and long term consequences of this are not unknown. In early pregnancy, maternal cholesterol contributes to fetal cholesterol and in late pregnancy, fetal biosynthesis satisfies the fetal requirement (Kharb *et al*., 2010; Barker *et al*., 1993).

Maternal physiology sets the fetal environment and maternal health status during pregnancy significantly affects the fetal growth in uterus. It still remains unclear whether placenta plays an important role in metabolism of lipoproteins in the pregnant women. Placental proteins have been hypothesized to contribute to lipoprotein metabolism of pregnant women since placental proteins are encoded by gene belonging to fetal genome (Kharb *et al.,* 2010; Barker *et al*., 1993).

According to Barker hypothesis' disturbed intrauterine growth has a negative influence on development of cardiovascular system and favors occurrence of hypertension, insulin resistance, hypercholesterolemia and hyperuricemia in later life and adult cardiovascular disease is programmed during rapid growth in fetal life (Barker, 1995). Since birth weight is summary

measure of fetal growth and its determinants include genetic growth potential, length of gestation and maternal size and supply line to baby (in terms of metabolic and nutritional status of mother, uteroplacental perfusion and placental function) (Dati, 1989). Hence the present study was planned to study metabolic derangements in preeclampsia by analyzing cord blood Atherogenic Index (AI) in normal pregnant and preeclamptic women and to correlate them with birth weight and maternal AI.

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, India. Study group consisted of 50 women with preeclampsia (group II). In inclusion criteria: subjects with singleton pregnancy with more than 20 weeks of gestation, blood pressure >140/90 mmHg with or without proteinuria. Exclusion criteria: subjects with chronic hypertension, renal disease, any metabolic disorder, diabetes mellitus and any supplements. Fifty health normotensive pregnant women served as controls (group I).

Ten milliliter cord blood was collected from placental end of umbilical cord and 5 mL of maternal venous sample was drawn from antecubital vein at the time of delivery. Serum was separated by centrifugation and Apo A-I and Apo B levels were analyzed by immunoturbidimetric immunoassay kits (Randox-make, Konelab-30). Atherogenic index was calculated as the ratio of Apo B to Apo A-I. Data so obtained was computed as Mean±SD and Student's t-test was applied, also regression analysis was carried out.

RESULTS

In the present study, both maternal and cord blood AI were significantly higher in group II as compared to group I ($p<0.001$). Cord blood AI was 61 and 58% of maternal levels in group II as compared to group I. Apo B levels were significantly elevated in group II in both cord blood and maternal blood as compared to group I. Apo A-I levels were lower in maternal blood and higher in cord blood of group II.

Mean birth weight of babies of mothers with preeclampsia was significantly less as compared to normotensive pregnant ($p<0.001$, Table 1). In maternal blood, AI was higher in group I as compared to group I ($p<0.001$, Table 1). Cord blood AI was higher in group I as compared to group I ($p<0.001$, Table 1).

Table 1: Birth weight and cord blood AI in both the groups (Mean±SD)

	Group I	Group II
Maternal		
Apo A-I	157.12±34.17	144.86±41.62^{ns}
Apo B	109.46±17.95	137.84±34.82***
AI	0.72±0.15	1.038±0.411***
Cord blood		
Apo A-I	56.92±11.86	62.98±18.9^{ns}
Apo B	25.24±8.08	35.36±11.04***
AI	0.44±0.096	0.601±0.32^{ns}
Birth weight	2.85±0.311	2.39±0.526***

ns: Not significant as compared to group I, ***Significant at $p<0.001$ as compared to group I

Female babies of preeclamptic mothers have higher atherogenic index (0.62±0.30 vs. 0.57±0.35) as compared to male counterparts. In normotensive pregnant, cord blood of females was more atherogenic (0.45±0.10 vs. 0.42±0.88).

Also, a negative correlation was observed between cord blood AI and birth weight in group II ($r = -0.281$, $p<0.05$). While, group I showed a positive correlation between cord blood AI and birth weight ($r = 0.316$, $p<0.05$).

DISCUSSION

Preeclampsia are associated with an enhanced hyperlipidemia which seems to have negative impact on fetal lipid apolipoprotein profile and atherogenic index as reflected by a higher cord blood Apo A-I, Apo B and AI in preeclamptic mothers (Kharb *et al*., 2009). It is not clear whether it is cause or consequence. Increased Apo B and decreased Apo A-I could cause acute atherosis in umbilical placental circulation similar to atherosclerosis in adult life or alteration of Apo B-A-I is consequence of placental insufficiency. No reports are available in literature where AI were compared in maternal and cord blood of preeclampsia or cord blood AI correlated with birth weight.

Demonstration of gender based changes in the present study supports the idea of active contribution of placenta to metabolism of maternal lipoproteins during pregnancy. Maternal dyslipidemia has been associated with preeclampsia (Kharb *et al*., 2009; Hubel *et al*., 1998; Rodie *et al*., 2004) and cardiovascular risks in these women are partly determined by genetic makeup and polymorphism of their children.

Sex differences are observed in animal models of fetal programming and sex hormones modulate regulatory systems for vascular function and blood pressure control.

There are sex differences in fetal programming and differences in growth play a role in development of adult hypertension (Grigore *et al*., 2008). Thus, findings of the present study suggest that atherogenic profile is different in males and females since in utero!.

Gender differences in lipoproteins are primarily due to genetic influences, subject to subsequent modification by environmental factors. Adverse in utero environment changes the structure, physiology and metabolism of fetus before birth and thereby determines the development of cardiovascular disease in later life (Wang and Trudinger, 2000).

These findings lend support to the hypothesis that roots for adult heart disease begins in utero programming.

Findings of the present generate a new hypothesis that future cardiovascular risk of women is partly determined by polymorphism of their children.

CONCLUSION

Preeclampsia are associated with an enhanced hyperlipidemia which seems to have negative impact on fetal lipid apolipoproteins profile and atherogenic index as reflected by a higher cord blood Apo A-I Apo B and AI in preeclamptic mothers. Demonstration of gender based changes in the present study supports the idea of active contribution of placenta to metabolism of maternal lipoproteins during pregnancy. These findings suggest that atherogenic profile is different in males and females since in utero. Adverse in utero environment changes the structure, physiology and metabolism of fetus before birth and thereby determines the development of cardiovascular disease in later life. The findings of present study lend support to the hypothesis that roots for adult heart disease begins in utero programming. Findings of the present generate a new hypothesis that future cardiovascular risk of women is partly determined by polymorphism of their children.

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