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Research Article

Atherogenic lipid profile of intrauterine growth retarded newborns

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ABSTRACT

Background: Newborns with intrauterine growth restriction are under greater risk of coronary artery disease in future. The objective was to compare the concentration of serum lipids of umbilical cord blood of neonates of intrauterine growth retardation with normal term neonates.
Methods: 50 newborns with intrauterine growth retardation and 50 normal term newborns, with 38 to 41 gestational weeks, were studied. Total cholesterol, triglycerides, HDL, LDL and VLDL cholesterol were measured in umbilical cord blood samples. The period of study was from January 2011 to October 2015. Study design: Hospital based prospective study. Blood samples were collected from the study population in the neonatal unit and all serum lipid profile level was measured with all accuracy in a computerized automated biochemical analyzer in Biochemistry department. Statistical analysis were done by using student's unpaired 't' test by using graph pad prism software.
Results: TC concentration was significantly decreased in the IUGR compared AGA groups. LDL-C concentrations were decreased in the IUGR compared to the AGA group. TG concentration was significantly increased in IUGR as compared to the AGA group.
Conclusions: Intrauterine growth retarded newborns were with worse lipid profile in Indian IUGR neonates. Further research is needed into the determinants of fetal growth and to the ways preventing fetal under-nutrition.

Keywords: Lipid profile, Cord blood, Intrauterine growth retarded newborns, Atherogenesis

INTRODUCTION

Intrauterine environment is extremely labile to maternal influence, which could therefore play an important role in fetal growth.

The evidence for this comes from epidemiological studies demonstrating associations between various different types of maternal exposures on birth weight, with adverse consequences for metabolic disease. For coronary artery disease some factors not under the control of an individual include age, hereditary factors, and diabetes mellitus which cannot be modified. Other modifiable risk factors are influenced by lifestyle choices and include excessive alcohol consumption, high stress, smoking tobacco, physical inactivity, obesity, hypertension, and blood lipid profile.[1]

The earliest epidemiological study linking poor fetal growth and subsequent development of type 2 diabetes was the observation by Hales and colleagues**,** which found that among men in their sixties, those who had

lower birth weights and weights at 1 year were more likely to develop poor glucose tolerance and type 2 diabetes.[2]

Fetal programming

''Programming'' is a process by which developmental stress leads to disease. Though the concept of 'fetal origins' is now well established the mechanisms involved are not clear. It has been suggested that a growing fetus faced with adverse conditions, responds with endocrine, anatomic, metabolic and vascular or other structural adaptations that impart disease vulnerability.[3-5]

The commonest adversity for the fetus may be 'malnutrition' during intrauterine life. The first priority for a developing fetus is survival, and during the 'lean' periods this is achieved by reduced rate of growth (intrauterine growth retardation, IUGR). The time during intrauterine life when this occurs determines the systems affected. The "fetal origins" hypothesis proposes that the association reflects permanent resetting of blood pressure due to intrauterine undernutrition.[3]

Most common cause of fetal malnutrition may be poor maternal nutrition, though this has not been proved beyond doubt. Coronary heart disease and stroke, and the associated conditions hypertension and type II diabetes, originate through impaired growth and development during intrauterine fetal life and infancy. These diseases may be consequences of 'programming'.[5] Maternal 'nutrition' might play such a crucial role in determine the future risk of hypertension, type II diabetes and cardiovascular disease in the offspring. If this hypothesis proves to be correct may have profound implications for prevention of various disorders.

The most widely accepted phenomenon proposed to underlie the developmental origins hypothesis is that of programming. Other well recognised mechanisms includes

- Altered fetal nutrition
- The thrifty genotype hypothesis
- The thrifty phenotype hypothesis
- Predictive adaptive responses

Altered fetal nutrition

Fetal nutrition is an important key regulator of fetal growth, and thus an obvious candidate as a possible programming influence.[6] It has proved remarkably easy in experimental animals to permanently alter postnatal physiology in a way analogous to that seen in the human studies by manipulation of maternal diet during pregnancy.

In various experiments in rats, both global maternal under nutrition and protein diet restriction result in elevated blood pressure in the offspring of protein-deprived rats and low birth-weight.[7-9]

The thrifty genotype hypothesis

The thrifty genotype hypothesis was proposed by James Van Gundia Neel in 1962 as an explanation for the increasing prevalence of type II diabetes Neel JVG intended the paper to provoke further contemplation and research on the possible evolutionary and genetic causes of diabetes.[9] He suggested that the selection of "thrifty" genes that caused insulin resistance and slow fetal growth was an evolutionary adaptation and survival mechanism in undernourished conditions, but confers a disadvantage in settings of over nourishment.[10-12]

This hypothesis was further adapted to explain the association between smaller body size at birth and disease risk because insulin plays central role in both fetal growth and carbohydrate regulation**.**[13] However, a strictly genetic explanation dismissed any environmental influences which were inconsistent with findings from experimental animal models or from natural experiments, such as the Dutch Famine.[13]

The thrifty phenotype hypothesis

The Thrifty Phenotype Hypothesis an attempt to explain the associations between poor fetal and infant growth and increased risk of developing impaired glucose tolerance and the metabolic syndrome11 in adult life. Suboptimal maternal nutrition, the fetus learns how to be "thrifty" with its energy needs, resulting in intrauterine growth retardation.[14]

Although the learned response is a survival mechanism of the fetus to ensure energy needs are distributed to vital organs during intrauterine development, it comes at the cost of slower fetal growth and increase in peripheral resistance to insulin. However, critics of the thrifty phenotype hypothesis argued that extreme maternal poor nutrition is necessary to see impaired growth in the developing fetus, despite evidence of a graded risk of type II diabetes associated with birth weight.[12] The thrifty phenotype hypothesis says that reduced fetal growth is strongly associated with a number of chronic conditions include coronary heart disease, stroke, diabetes, and hypertension in later life. This increased susceptibility results from adaptations made by the fetus in an environment limited in its supply of nutrients.[14]

Predictive adaptive responses (PAR)

A main criticism of both the thrifty genotype and thrifty phenotype hypotheses is their uni-directionality, specifically that the risk of disease was only explained for those with fetal growth retardation and under conditions of maternal malnourishment. In addition, these hypotheses focused on fetal development, and did not include the periods of pre-conception, conception, or

infancy, which are also still susceptible to developmental plasticity. To address these concerns, Gluckman and Hansen proposed a broader explanation of the developmental origins of disease paradigm with the PAR. The PAR is the hypothesis that gene-environment interactions occurring early in development trigger a physiologic and physical phenotypic change during development, not necessarily for immediate survival, but to provide an advantage for a specific future environment.[15]

Using cues and information provided by the mother, the egg, embryo, fetus, and/or infant learns to adapt to a specific environment, which provides a basis for predicting the responses that will be needed to increase its survival and growth later in life. It is theorized that PARs lead to disease when there is a divergence from the environment that was expected to the environment that was actually realized.[15,16]

Study of umbilical cord blood lipid profile immediately after birth can shed new light on lipid metabolism in term newborns with intrauterine growth retardation compared to normal appropriate for gestational age (AGA) term newborns. This study will try to find out the influence of prematurity on cord lipid levels, cord blood lipid levels in term AGA and Intrauterine growth retarded newborns

METHODS

The present study was carried out in Government Medical College, Nagpur, Maharashtra, India. The period of study was from January 2011 to October 2015. Study design: Hospital based prospective study.

Present study consists of a total number of 100 newborns, which were further divided into 50 newborns with intrauterine growth retardation newborns (IUGR) and 50 term newborns with appropriate for gestational age(AGA). Mother gave their informed consent.

Protocol of study was approved by institutional ethics committee.

Inclusion criteria for mother

- Healthy mother only on iron, folic acid and calcium supplementation.

Exclusion criteria for mother

- History of alcoholism
- History of smoking
- History of hypertension,
- History of thyroid disorders
- History of diabetes Mellitus
- History of renal diseases
- History of hypercholesterolemia
- History of twins
- History of liver diseases
- History of tuberculosis and asthma
- Positive TORCH screening results
- Recent pregnancy
- History of celiac disease

Inclusion criteria for newborns

- Gestational age between 35-42 weeks,
- One-minute apgar score >7
- Absence of any congenital anomalies

Exclusion criteria for newborns

- Congenital malformations
- Neonates born to mother with maternal illness which is already excluded in exclusion criteria for mother
- Neonates with perinatal problems like hypoglycaemia, pathological jaundice
- Instrumental delivery, including extraction
- Neonates with hypoxic–ischemic encephalopathy (HIE), Sepsis
- Respiratory distress
- Multiple gestation

Any morbidity during pregnancy particularly anemia and pre-eclampsia were noted Hemoglobin less than 10 gm% was considered as an evidence of anemia. Proteinuria, edema and hypertension were considered as evidence of pre eclampsia. Weight, height and mid arm circumference of mother was recorded by standard methods. Weight less than 40 kg and height less than 145 cm were considered evidence of maternal malnutrition.[17]

Clinical data recording

All the mothers included in study were evaluated as per the pro forma given herewith. Each mother underwent detailed clinical history, physical examination and investigations. Neonates were selected on the basis of gestational age ranging from 35 – 42 weeks and birth weight ranging from 1200 – 3800 grams. Birth weight was taken within 24 hours of birth on an electronic weighing machine. Gestational age was confirmed by New Ballard et al scoring system done within 24 hrs of birth.[18]

Newborns were divided into two groups- term and near term. Babies between 35-36.6 weeks of gestation were taken as near term and those with 37 completed weeks of gestation upto 42 weeks were taken as term after conformation of gestational age by history, clinical examination and ultrasonography by Obstetrics and Gynaecology department of GMC Nagpur.

We measured serum levels of lipid profile comprising of total cholesterol (TC), triglyceride (TG), high-density lipoprotein-cholesterol (HDLC), low-density lipoprotein

cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C).

Specimen collection and analysis

After delivery of the placenta and immediately after cord clamping, 5 ml of umbilical venous blood was obtained from the placental end of the umbilical cord under aseptic precautions. It was then allowed to stand for few minutes. Serum was separated from the clotted blood after centrifuging at 3000 rpm for 30 minutes and analyzed immediately. From the serum, total cholesterol (TC), total triglyceride (Tg), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C) and very low density lipoprotein (VLDL-C) were measured with all accuracy in a computerized automated biochemical analyzer by enzymatic method.

Statistical analysis

Mean values and standard error of mean had been used to define data in each group. These values were compared between 50 normal term newborns and 50 newborns with intrauterine growth retardation group. Student unpaired't' test was used to test the significance between the data.

The p value less than 0.05 was considered as significant and the p value less 0.001 was considered as highly significant. GraphPad Prism version 6.00 software was used for analysis.

RESULTS

Baseline characteristics of the study groups are shown in (Table 1). There was no significant difference between intervention and control groups in terms of Gestational age at delivery & Mean weight in kg. The mean gestational age in near term AGA and near term IUGR was found to be 38.21±0.07 and 37.91±0.09 respectively. The mean weight in term AGA and preterm SGA was found to 2.69±0.02 and 2.05±0.01respectively.

TC concentration was significantly decreased in the IUGR compared AGA groups. LDL-C concentrations were decreased in the IUGR compared to the AGA group. TG concentration was significantly increased in IUGR as compared to the AGA group.

Table 1: Distribution of newborns according to gestational age and weight.

Variable	Appropriate for gestational age newborns (Mean±SEM) (N=50)	IUGR newborns (Mean±SEM) (N=50)
Gestational age at delivery	38.21±0.07	37.91±0.09
Mean weight in kg	2.69±0.02	2.05±0.01

Table 2: Lipid profile in relation to term at delivery in appropriate for gestational age newborns and IUGR newborns.

Variable	Appropriate for gestational age newborns (Mean±SEM) (N=50)	IUGR newborns (Mean±SEM) (N=50)	P value
Total cholesterol TC (mg/dl)	70.62±1.12	66.66±1.62****	<0.0001
HDL C(mg/dl)	13.66±0.31	11.72±0.36****	<0.0001
LDL-C (mg/dl)	56.58±1.16	53.09±1.34	NS
VLDL-C (mg/dl)	5.784±0.15	6.640±0.17****	<0.0001
Serum Triglycerides(mg/dl)	28.92±0.79	33.20±0.89****	<0.0001

* = (p<0.05); **= (p<0.01); ***= (p<0.001); ****(p<0.0001).

DISCUSSION

To the best of my knowledge, this study is first of its kind where sample size was robust thus differs. Several strengths of our study should be noted. None of the studies were conducted in our region where comparison of lipid profile between IUGR term neonates and AGA term neonates were done.

This study observed that lipid profile of normal healthy newborns in Central region of Maharashtra (Nagpur) showed lower values of total cholesterol, HDL fraction cholesterol and higher serum triglycerides and higher VLDL fraction of cholesterol as compared to foreign studies. It is known that higher values of serum triglycerides and low level of HDL fraction of cholesterol are associated with increased risk of coronary artery disease. Hence it can be said that newborns of this region are more likely to develop coronary artery disease in adulthood at an earlier age as compared to newborn of other countries.

Total serum cholesterol

In our study, we observed that in serum total cholesterol in IUGR group was lower than those in a term AGA

group, which was a statistically significant finding. However, our results demonstrated that the serum TC levels in the IUGR group were lower than those in the term AGA group, which is in agreement with Pecks U et al.[19]

For lower cholesterol IUGR, possible explanation are

- Alteration of cholesterol acceptor concentration or functionality and a disturbed interaction with reverse cholesterol transport (RCT) mechanisms at the placenta fetal interface. Reduction in cholesterol efflux acceptor capacity appears to diminish cholesterol availability and trans-placental cholesterol transport to IUGR fetuses. Moreover, disturbances of RCT are involved in the patho mechanisms of atherosclerosis by Pecks et al.[20]

- In other study by Pecks et al,The evaluation of the oxidative state of low-density lipoproteins in pregnancy complicated by intrauterine growth restriction and preeclampsia, they found lower maternal LDL-C and TC concentration in IUGR pregnancies thats why there is decreased cholesterol supply to the fetus in IUGR.[21]

- Conformational changes of the ApoB lipoprotein during the process of oxidation might lead to an accumulation of oxLDL particles in placental tissue of IUGR and reduced fetal cholesterol bioavailability as evidenced by a decrease in fetal serum cholesterol levels Pecks U et al.[33]

Serum LDL-cholesterol

In our study, we observed that in serum LDL-Cholesterol in IUGR group was lower than those in a term AGA group, which was not statistically significant finding which is in agreement with Pecks U et al.[21] The origin of fetal LDL is still a matter of debate. LDL is usually produced at the liver. Hence, the low fetal LDL concentrations in IUGR may be secondary to a limited cholesterol pool of the fetus. However, in normal pregnancy fetal LDL levels decrease with gestational age, and studies have shown that in early stages of pregnancy (i.e. before the 6th month) fetal LDL correlates to LDL levels of the mothers, while in third trimester this correlation is virtually absent.[19] From above observation and from our study observation we can say that a basal LDL production rate is maintained by the fetus throughout gestation. Throughout pregnancy maternal LDL may contribute to the fetal LDL pool with decreasing amounts, until at term the LDL concentration reflects the basal production rate of the fetus.

Serum HDL-cholesterol

In our study, we observed that in serum HDL-Cholesterol in IUGR group was lower than those in a term AGA group, which was a statistically significant finding.This finding is similar with study conducted by Pecks U et al.[19]

Maternal cholesterol is actively transferred via the placenta. At the apical placental surface facing maternal blood, i.e., the syncytiotrophoblast, many lipoprotein receptors are involved in cholesterol uptake.[25] Stefulj J et al proposed sequential pathway mediated by ATP binding cassette transporters ABCA1 and ABCG1 by which Human term placenta endothelial cells participate in forming nasecnt HDL in the fetal blood, thus substantially contributing to the fetal HDL-Cholesterol.[26] The relevance of oxidized LDL particles in the pathogenesis of IUGR has been highlighted by Pavan et al who found that oxidized LDL inhibits trophoblast cell invasion in vivo and in vitro.[27] Pavan et al further provided evidence for the involvement of the liver X receptors (LXRs) in this process as a target for ox LDL associated oxysterols.[28] Downstream targets of LXRs are the ATP binding cassette transporters ABCA1 and ABCG1. Those transporters promote cholesterol efflux from cells including trophoblasts and placental endothelial cells, and hence are critically involved in the protection from cholesterol overload. And maintain lipid homeostasis.[26,29] OxLDL mediated LXR inhibition leads to a downregulation of ABCA1.[30] Since ATP-binding cassette (ABC) transporters are involved in the materno-fetal cholesterol transport, oxLDL mediated dysregulation of this system may have consequences for fetal cholesterol supply. This is consistent with the findings of Pecks U et al as the HDL cholesterol concentrations being diminished in cord blood of fetuses suffering from IUGR compared to control cases.[21]

Serum triglycerides

In our study, we observed that in serum triglycerides in IUGR group was higher than those in a term AGA group, which was a statistically significant finding. This finding of our study is in agreement with Koklu E et al, Hossain MA et al and Molina M et al.[22,31,32] One possible explanation of raised Triglycerides in IUGR group is Fetal deglycosylated apolipoprotein C-III (Apo C-III0) concentration is altered in intrauterine growth restriction, the de-glycosylated form of the apolipoprotein C-III (apoC-III0) increased in IUGR which might prevent catabolism of triglyceride by Pecks et al.[23]

Serum VLDL -cholesterol

It is observed that VLDL – C is higher in IUGR group as compared to term neonates. The difference was not statistically significant. After extensive search the overall pattern of change in VLDL – C is not clearly outlined.

The present study showed significantly higher triglcerides, VLDL and lower total cholesterol, HDL cholesterol in term IUGR as compared to appropriate for gestational age.

CONCLUSION

From the present study, it can be said that IUGR newborns are born with adverse lipid profile which can predispose them to early coronary atherosclerosis. This study showed that there is high incidence intrauterine growth restriction in malnourished mothers. Since adverse lipid profile has been documented in IUGR newborns, maternal malnutrition although indirectly is responsible for adverse lipid profiles.

It was clearly visible that cord blood lipids of IUGR in our study were changed towards an atherogenic phenotype when compared to appropriate gestational age controls. It is of note that the decreased concentration of HDL-C was associated with an increase in the degree of oxidized LDL particles. This observation makes sense, since HDL-C is known to protect LDL from oxidation. In conclusion, IUGR is associated with a lower fetal serum cholesterol concentration. The athero protective HDL-C is mostly affected. It may be interesting to see whether these susceptible IUGR neonates are at increased risk of developing cardiovascular diseases in future. The study also hints about the role of adverse maternal conditions in origin of early onset.

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