International Journal of Medical Science and Education

An official Publication of Association for Scientific and Medical Education (ASME)

Original Research Article

STUDY OF LIPID PROFILE IN CORD BLOOD OF FULL TERM NEONATES OF NORMAL LABOUR

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Received: 15/02/2017 ***Revised:02/07/2017*** ***Accepted: 12/07/2017***

ABSTRACT

Background: Neonates of mothers having a family history of hypertension & coronary heart disease have a high risk of developing atherosclerosis in later life as they have increased blood lipids & cholesterol since birth. Therefore efforts are being made for screening for hyperlipidemia in the cord blood to screen the newborn who is at risk of developing atherosclerosis in later life so that necessary steps can be taken for its prevention at an early age. **Material and methods:** This work was undertaken to study lipid profile of cord blood of full-term pregnant women in normal labor. The parameters evaluated were serum triglyceride (TG), cholesterol (CHOL), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein. The family history of coronary artery disease and hypertension is recorded in each case. **Results:** The difference of mean for TG, CHOL, HDL, LDL, VLDL of cases is highly significant compared to control by ANOVA test. Serum TG, CHOL and VLDL were highly significant in all the three groups when compared to each other by post hoc test. Mean values were high for all parameters in female neonates but it was significantly high in CHOL and LDL levels. **Conclusion:** The genetic factors plays an important role in the transmission of hyperlipidemia and cord blood lipid estimation may provide a useful means to detect hyperlipidemia in high risk babies of full term pregnant women having family history of hypertension and coronary artery disease so that preventive measures could be taken early to retard the process of atherosclerosis.

Keywords: hyperlipidemia, hyperlipidemia, atherosclerosis, of cardiovascular disease.

INTRODUCTION

There is convincing evidence that cardiovascular diseases have genetic component and there is robust gene-environment interaction in development of manifest atherosclerosis. **(1)**

Atherosclerosis, a major cause of cardiovascular disease, is a process that begins in early life and progresses silently for decades. Among the factors incriminated in the development of atherosclerosis, increased blood cholesterol especially low-density lipoprotein (LDL) and other lipids are important. **(2)**

Neonates of mothers having a family history of hypertension & coronary heart disease have a high risk of developing atherosclerosis in later

life as they have increased blood lipids and cholesterol since birth. **(3, 4)**

Therefore efforts are being made for screening for hyperlipidemia in the cord blood of full-term pregnant women in normal labor having a family history of hypertension and coronary heart disease as cholesterol levels in the subsequent periods are likely to be influenced by these factors.

MATERIAL AND METHOD

This work was undertaken to study lipid profile of cord blood of full-term pregnant women in normal labor. The parameters evaluated were serum triglyceride (TG), cholesterol (CHOL), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein. The family history of coronary artery disease and hypertension is recorded in each case, with a special reference, to assess the role of genetic factors in the transmission of hyperlipidemia.

It was a hospital-based analytical type of observational study carried out at the labor room of Mahila Chikitsalaya, Sanganeri Gate, S.M.S. Hospital, Jaipur. Samples were taken at the simple random system. Total 240 women and their neonates were taken as a study group. Out of 240 cases of full-term pregnant women studied, 64 were taken as cases and 176 were taken as controls. In 64 cases 33 women were having a family history of coronary artery disease (CAD), in first order family and 31 women were having a family history of hypertension (HT), in first order family.176 women were not having positive family history and served as controls.

The control neonate was full term neonate without an adverse fetomaternal factor. Five to ten ml of mixed blood collected from the placental end of the umbilical cord. After clotting at room temperature, serum was separated and stored at 4 degrees C. The analysis of lipids done by auto analyzer Randox Imola by a kit method in the central lab within 16 hours after obtaining the sample. The undressed weight of newborn infants was measured by Seca weight scale with a sensitivity of 10 mg.

Total cholesterol (TC) and triglycerides (TG) levels were assayed using enzymatic colorimetric tests with cholesterol esterase and cholesterol oxidase Enzymatic method (CHOD-PAP)] and glycerol phosphate oxidase (GPO-PAP method) respectively. **(5,6)** HDL-cholesterol was measured by the sedimentary method. **(7)** Serum LDL was estimated from the Freidwald and Fredrickson's (1972) formula, which is LDL=Total Cholesterol-[HDL+VLDL]. VLDL is estimated by formula VLDL (mg/dl) = TG/5.

Statistical analysis was performed by using SPSS 19.0 software (SPSS, USA). The mean and range were determined. Student's t-test was used for the comparison between lipid profile in same groups and ANOVA test was used for comparison between different groups. A p-value less than 0.05 was considered meaningful.

RESULTS

The mean ± S.D. of TG in control that is 34.93 ± 15.32 and the mean±S.D. of cases having family history of CAD & HT that is 69.58±31.49 and 50.93±24.86 respectively. The mean ± S.D. of CHOL in control that is 64.20 ± 18.45 and the mean±S.D. of cases having family history of CAD & HT that is 103.30±35.83 and 74.94±19.62 respectively.The mean ± S.D. of HDL in control that is 23.13 ± 7.92 and the mean±S.D. of cases having family history of CAD & HT that is 31.54 ± 9.10 and 26.54±10.47 respectively.The mean ± S.D. of LDL in control that is 33.90 ± 13.82 and the mean±S.D. of cases having family history of CAD & HT that is 58.78 ± 25.90 and 38.37±15.56 respectively. The mean ± S.D. of VLDL in control that is 7.01

± 3.08 and the mean±S.D. of cases having a family history of CAD & HT that is 13.89 ± 6.25 and 10.11±4.97 respectively. The difference of mean for TG, CHOL, HDL, LDL, VLDL of cases is highly significant compared to control by ANOVA test as shown in Table 1.

Serum TG, CHOL, and VLDL were highly significant in all the three groups when compared to each other by post hoc test (Tukey hsd test). The mean difference of serum HDL is nonsignificant when compared to cases having a family history of HT and control but significant in the comparison between cases of CAD & control and cases of CAD & HT. The mean difference of serum LDL is nonsignificant when compared to cases having family history of HT and control and cases of CAD & HT. but significant in the comparison between cases of CAD & control as shown in Table 2

Mean values were high for all parameters in female neonates but it was significantly high in CHOL and LDL levels. Male neonates of cases having a family history of CAD and HT have more levels of lipid profile but it was nonsignificant as shown in Table 3.

Cord blood show about 60 present of adult values of lipid profile. Cases having family history of CAD and HT have hypertriglyceridemia, hypercholesterolemia and hyperlipidemia and have risk of developing atherosclerosis in their later life.

DISCUSSION

Cardiovascular diseases (CVDs) are the largest single contributor to global mortality and will continue to dominate mortality trends in the future **(8)**. Nowadays, age-adjusted CVDs mortality is higher in major low and middle income countries than in developed countries **(9)**. Atherosclerosis is considered as a major cause of CVDs; it is a process that begins early in life and progresses silently for decades **(10)**.

In adults, increased low density lipoprotein cholesterol (LDL-C) and reduced high density lipoprotein cholesterol (HDL-C) levels are associated with atherosclerotic lesion, with its prodromal stages formed early in life **(11, 12, 13).** Children with high level LDL-C at birth might be more liable to high lipoprotein serum levels as they reach adulthood **(14, 15)**. In North America every child over the age of 3 years old has some degree of aortic fatty streaks **(16)**.

Extensive epidemiological evidence supports the relationship between both genetic and maternal factors such as hypercholesterolemia, diet, mode of delivery, length of pregnancy and pre-eclampsia with the cord blood lipids and lipoprotein profiles **(12, 17)**. Additionally, the fetal life is affected by maternal age. **(18)**

In the present study lipid profile were studied in the cord blood of full term pregnant women undergone normal labour. Cord blood of only full term pregnant women in normal labour were studied so as to exclude the effects of gestational age and perinatal factors on the fetal lipids.

The study aims at determining the lipid levels in the cord blood of full-term pregnant women in normal labour. An attempt was made to estimate the lipids in cord blood of pregnant women having family history of coronary artery disease and hypertension to establish the role of inheritance or genetic factors in transmission of hypercholesterolemia, hypertriglyceridemia and hyperlipidemia.

Lipid profile in cord blood of 33 cases were evaluated those having family history of CAD. Serum Triglyceride -In the cord blood of 33 full term women having family history of coronary artery disease mean serum triglyceride was 69.58±31.49 mg /dl with a range of 25.8-166.0

mg/dl. When compared to controls, the p-value was 0.000 which was highly significant. Hypertriglyceridemia was recorded (Table-1). In similar study done by B K Gupta et al the serum triglyceride value in cases of CAD was 45.4±22.2 which was lower than values in our study but they also showed higher values compared to controls. **(19)** In our study we also found higher values compared to controls, these results were consistent with our study. Serum cholesterol-The mean serum cholesterol was 103.30±35.83mg/dl with a range of 44.7-205.0mg/dl. When compared to controls, the p-value was 0.000 which was highly significant. The difference of mean was very high compared by ANOVA test and hypercholesterolemia was recorded.(Table-10).In similar study done by B K Gupta et al the mean serum cholesterol in cases of CAD was 111.2±31.2 which was in close approximation with our study.**(19)** They also showed in their study that the neonates of cases who were having family history of CAD have higher cholesterol levels at birth.

Boulten **(20)** et al also observed that the cord blood of these female with a history of early coronary heart disease through two consecutive generations had a higher mean total cholesterol than with no such history. Serum high density lipoprotein-In the cord blood of cases having family history of coronary artery disease mean serum HDL was 31.54±9.10mg/dl with a range of 12.9-51.8mg/dl. Serum low density lipoprotein-In the cord blood of cases having family history of coronary artery disease mean serum LDL was 58.78±25.90mg/dl with a range of 17.1-140.4 mg/dl. Serum very low density lipoprotein-In the cord blood of cases having family history of coronary artery disease mean serum VLDL was 13.89±6.25 mg/dl with a range of 5.2-33.0 mg/dl. When compared to controls, the p-value was 0.000 which was highly significant. The difference of mean was very high compared by ANOVA test and hyperlipidemia was recorded. (Table-1)

A study exactly similar to the present could not be found in literature. A few studies appear to be somewhat identical B K Gupta et al **(19)** Boulten et al **(20)** and Goldstein et al **(21).**

The present study shows that in families affected with ischaemic heart disease, a significant percentage of off-springs have raised levels of serum lipids. Goldstein et al studied plasma lipids and prevalent coronary heart disease among neonates with normal or high cord blood cholesterol levels. **(21)** It was observed that children whose lipids were in the higher quartile were more likely to have parents with high lipids or coronary disease.

Bellu et al **(22)** investigated levels of atherogenic lipid fractions in 1276 newborns. 400 cord blood samples were collected and it was observed that in neonates with a family history of ischemic cardiovascular disease had higher levels of total and LDL cholesterol while there was no difference in triglyceride levels. On day 4, 1200 samples were obtained and cholesterol and triglycerides measures and it was found that by this time this difference in total cholesterol was no longer detectable (LDL cholesterol not determined).

In the cord blood of cases having family history of hypertension mean serum TG, CHOL, HDL, LDL, VLDL were 50.63±24.86mg/dl, 74.94±19.62 mg/dl, 26.54±10.47mg/dl, 38.37±15.56mg/dl and 10.11±4.97 respectively. When compared to controls, mean serum TG, CHOL, HDL, LDL, VLDL 34.93±15.32mg/dl, 64.20±18.45mg/dl, 23.13±7.92mg/dl, 33.90±13.82 mg/dl, 7.01±3.08mg/dl respectively the p-value was 0.000 which was highly significant. The difference of mean was very highly significant compared by ANOVA test and hyperlipidemia was recorded (Table-1)

B K Gupta et al 2004 **(19)** In neonates with parental history of hypertension the cord-blood levels of cholesterol was 96.4±22.1 and triglycerides was 50.6±22.2 mg/dl. Hyperc-holesterolemia (cholesterol >85th percentile) was observed in 12.7% of control subjects. In neonates having family history of hypertension the incidence of hypercholesterolemia was 22.7% which was not significant. Hypertriglyceridemia (triglycerides >85th percentile) was recorded in 12.7% of control subjects, whereas the incidence in neonates having family history of hypertension was 33.3% (p= n.s.).Our findings match with Lakhtakia et al. **(23)**

In our study we found that serum TG, CHOL and VLDL is highly significant in all the three groups when compared to each other by post hoc test (tukey hsd test). Our study also showing that the mean difference of serum HDL is non significant when compared between cases having family history of HT and control but significant in comparison between cases of CAD & control and cases of CAD & HT. In case of LDL study showing that the mean difference of serum LDL is non significant when compared between cases having family history of HT and control and between cases of CAD & HT but significant in comparison between cases of CAD & control .(Table-2) A study exactly similar to the present could not be found in literature.

CONCLUSION

Lipid profile of the Indian population cannot be comparable with norms of the other country because there are many factors that can change profile value, like dietary habit, living habit and condition, environment and atmosphere also.

The present study favours the concept that genetic factors plays an important role in the transmission of hyperlipidemia and cord blood lipid estimation may provide a useful means to detect hyperlipidemia in high risk babies of full term pregnant women having family history of hypertension and coronary artery disease so that preventive measures could be taken early to retard the process of atherosclerosis.

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Table -1 Mean±S.D of lipid profile in case and control

		N	Mean	Std. Deviation	Std. Error	Minimum	Maximum	P-Value
TG	Control	176	34.930	15.3297	1.1555	13.2	168.0	.000
	CAD	33	69.585	31.4952	5.4826	25.8	166.0	
	HT	31	50.639	24.8670	4.4662	18.0	129.0	
	Total	240	41.724	23.1081	1.4916	13.2	168.0	
CHOL	Control	176	64.203	18.4522	1.3909	27.4	204.0	.000
	CAD	33	103.309	35.8381	6.2386	44.7	205.0	
	HT	31	74.942	19.6263	3.5250	38.0	113.2	
	Total	240	70.967	25.4911	1.6454	27.4	205.0	
HDL	Control	176	23.134	7.9297	.5977	9.1	50.0	.000
	CAD	33	31.548	9.1071	1.5854	12.9	51.8	
	HT	31	26.542	10.4732	1.8810	7.3	53.9	
	Total	240	24.731	8.9240	.5760	7.3	53.9	
LDL	Control	176	33.903	13.8248	1.0421	5.7	120.4	.000
	CAD	33	58.788	25.9038	4.5093	17.1	140.4	
	HT	31	38.374	15.5654	2.7956	8.4	69.6	
	Total	240	37.902	18.2274	1.1766	5.7	140.4	
VLDL	Control	176	7.016	3.0898	.2329	2.6	33.6	.000
	CAD	33	13.897	6.2586	1.0895	5.2	33.0	
	HT	31	10.113	4.9753	.8936	3.6	25.8	
	Total	240	8.363	4.6155	.2979	2.6	33.6	

Table-2 Tukey HSD Comparison of lipid profile between groups

Dependent Variable	(I) Name of Group	(J) Name of Group	Mean Difference (I-J)	Std. Error	Sig.
TG	Control	CAD	-34.6553*	3.7257	.000
		HT	-15.7092*	3.8255	.000
	CAD	Control	34.6553*	3.7257	.000
		HT	18.9461*	4.9124	.000
	HT	Control	15.7092*	3.8255	.000
		CAD	-18.9461*	4.9124	.000
CHOL	Control	CAD	-39.1063*	4.1282	.000
		HT	-10.7391*	4.2389	.032
	CAD	Control	39.1063*	4.1282	.000
		HT	28.3672*	5.4432	.000
	HT	Control	10.7391*	4.2389	.032
		CAD	-28.3672*	5.4432	.000
HDL	Control	CAD	-8.4150*	1.6042	.000
		HT	-3.4084	1.6472	.098
	CAD	Control	8.4150*	1.6042	.000
		HT	5.0065*	2.1152	.049
	HT	Control	3.4084	1.6472	.098
		CAD	-5.0065*	2.1152	.049
LDL	Control	CAD	-24.8850*	3.0728	.000
		HT	-4.4714	3.1552	.334
	CAD	Control	24.8850*	3.0728	.000
		HT	20.4137*	4.0516	.000
	HT	Control	4.4714	3.1552	.334
		CAD	-20.4137*	4.0516	.000
VLDL	Control	CAD	-6.8805*	.7461	.000
		HT	-3.0964*	.7661	.000
	CAD	Control	6.8805*	.7461	.000
		HT	3.7841*	.9838	.000
	HT	Control	3.0964*	.7661	.000
		CAD	-3.7841*	.9838	.000

*. The mean difference is significant at the 0.05 level.