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Original article

Study on Lipid Profile in Offspring of Dyslipidemic Parents

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ABSTRACT

As dyslipidemia is responsible for remarkable cause of cardiovascular disease which is considered as the number one cause of death globally, this study was undertaken to evaluate the lipid profile status of offspring of dyslipidemic parents in comparison with the offspring of normolipidemic parents. In this study, carried out on 89 subjects, the mean total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL-c) and low density lipoprotein (LDL-c) were 162.18 ± 20.97, 168.98 ± 33.51, 39.09 ± 3.62 and 90.41 ± 18.64 mg/dl respectively in adolescents with parental dyslipidemia and 158.38 ± 12.67, 157.22 ± 15.06, 40.51 ± 2.90 and 86.42 ± 12.39 mg/dl respectively in control adolescents. There were significant differences ($p < 0.05$) of TG and HDL-c between cases and controls where HDL-c choesterol was significantly lower in offspring of dyslipidemic parents then the offspring of normolipidemic parents. The findings of this study reflect the association of offspring dyslipidemia with parental dyslipidemia, probably due to some genetic predisposition. Offspring of dyslipidemic parents have higher levels of TC, TG, LDL-c and lower level of HDL-c compared to age and sex matched control subjects.

Key words: Offspring, Dyslipidemia, TC, TG, HDL-c, LDL-c. Here dyslipidaemia is considered as hyperlipidaemia

Introduction

Cardiovascular disease (CVD) is the number one cause of death globally and is projected to remain the leading cause of death. An estimated 17.5 millions people of died from CVD in 2005, representing 30% of all global deaths[1]. Dyslipidemias are disorders of lipoprotein metabolism which may result in hyperlipidaemia or hypolipidaemia. Among the dyslipidaemias hyperlipidaemia is the most common and world wide problem. Epidemiologic studies in children establish a strong statistical association between childhood overweight and hyperlipidaemia. Other risk factors of dyslipidaemias includes familial hypercholesterolemia, familial hypercholesterolemia, familial defective apoprotein-B, and familial hypertriglyceridemia. There may be dyslipidaemia of some secondary causes which includes nephritic syndrome, diabetes mellitus, and hypothyroidism[3].

Many studies have demonstrated that atherosclerosis has its silent beginning during childhood. Genetic component is believed to be the more predominant factor for coronary atherosclerosis early in life[4]. Patients with familial hyperlipidemia have a high incidence of premature coronary disease and many epidemiological studies have demonstrated a positive correlation between mean population plasma cholesterol concentration and morbidity and death from coronary disease[5]. More than half of patients with angiographically confirmed coronary artery disease (CAD) before age 60 years have a familial lipoprotein disorder[6].

Dyslipidemia were defined TC ≥ 240 mg/dl, HDL-c < 40 mg/dl, LDL-c ≥ 160 mg/dl and TG ≥ 200 mg/dl in adults, according to National Cholesterol Education Program

Adult Treatment Panel–III (NCEP ATP-III) 2002[7]. Adolescent is defined by WHO as a person between 10-19 years of age[8]. In children and adolescents, dyslipidemia were defined as TC ≥ 200 mg/dl, LDL-c ≥ 130 mg/dl according to National Cholesterol Education Program (NCEP) Expert Panel on Blood Cholesterol Levels in Children and Adolescents[9], TG levels > 150 mg/dl and HDL-c levels < 40 mg/dl[10]. For children in general, borderline levels of TC and LDL-c are defined as 170-199 and 110-129 mg/dl, respectively[11].

Dyslipidemia rarely leads to adverse health outcome in childhood, but its long term effects may be considerable. Large epidemiologic studies indicate that children's lipid levels correlate with those of adult family members. Children of parents with CHD have a higher prevalence of dyslipidemia in childhood, and identification of dyslipidemia in children can identify families at increased risk for CHD. Identifying children with dyslipidemia could lead to interventions or treatments that could prevent or delay adult dyslipidemia and CHD[2].

Subjects and Methods

The study was carried out in the the Department of Biochemistry and Department of Cardiology of Sylhet MAG Osmani Medical College, during the period of January 2007 to December 2007. 89

adolescents aged 10-19 years of selected hyperlipidaemic parents were taken as samples. 44 offspring of parents with known and biochemically proven dyslipidemia were considered as case (group-I) and 45 offspring of parents (both) without history and biochemically proven dyslipidemia were taken as control (group-II). Patients attending the out patients setup of cardiology department and admitted in the coronary care unit were considered as parents of cases and controls were taken from general population. Dyslipidemia of parents were defined as NCEP ATP-III Guidelines 2002[7].

Estimation of serum TC, TG, HDL-c, LDL-c, glucose and creatinine were done. The results were expressed as mean ± SD. All statistical analyses were done by SPSS software program, using Student's unpaired 't' test. A level of $p < 0.05$ was accepted as statistically significant.

Results

In group-I, the mean of TC, TG, HDL-c and LDL-c were 162.18 ± 20.97, 168.98 ± 33.51, 39.09 ± 3.62 and 90.41 ± 18.64 mg/ dl respectively. In group-II, the mean of TC, TG, HDL-c, and LDL-c levels were 158.38 ± 12.67, 157.22 ±15.06, 40.51 ± 2.90 and 86.42 ±12.39 mg/dl respectively. There was significant difference of HDL-c and TG between cases and controls ($p < 0.05$) (Table-I).

Table-I: Comparison of lipid profile between .group-I and group-II.

Parameter (mg/dl)	Group-I (n = 44) Mean ± SD	Group-II (n = 45) Mean ± SD	p
TC	162.18 ± 20.97	158.38 ± 12.67	> 0.05
TG	168.98 ± 35.51	157.22 ± 15.06	< 0.05
HDL-c	39.09 ± 3.62	40.51 ± 2.90	< 0.05
LDL-c	90.41 ± 18.64	86.42 ± 12.39	> 0.05

Means compared using Student's unpaired 't' test.

Table-II shows the lipid profile status in dyslipidemic parents and normolipidemic parents. In normolipidemic parents, TC, TG, HDL-c and LDL-c levels are 177.42 ± 18.25, 178.06 ± 13.48, 41.94 ± 2.28 and 98.89 ± 18.43 mg/dl respectively. In dyslipidemic parents, TC, TG, HDL-c and LDL-c were 216.76 ± 31.63, 274.04 ± 65.37, 37.28 ± 8.78 and 126.16 ± 26.82 mg/ dl respectively. There was a highly significant mean difference ($p < 0.001$) of TC, TG, LDL-c and significant mean difference ($p < 0.05$) of HDL-c between dyslipidemic parents and normolipidemic parents.

Table-II: Comparison of lipid profile between dyslipidemic parents and normolipidemic parents.

Parameter mg/dl	Dyslipidemic parents (n=25) mean ± SD	Normolipidemic parents (n=36) mean ± SD	p
TC	216.76 ± 31.63	177.42 ± 18.25	< 0.001
TG	274.04 ± 65.37	178.06 ± 13.48	< 0.001
HDL-c	37.28 ± 8.78	41.94 ± 2.28	< 0.05
LDL-c	126.16 ± 26.82	98.89 ± 18.43	< 0.001

Means compared using Student's unpaired 't' test.

Discussion

Dyslipidemia is a conventional risk factor for CVD and also for cerebrovascular diseases in most cases[12]. Family history of parental dyslipidemia is one of the major factors to be considered in the decision of assessing the lipid profile in children and adolescents[7]. The aim of this study was to test the hypothesis that dyslipidemia in offspring is associated with dyslipidemia in parents in our population. Fasting serum lipid profile status were estimated in adolescents with documented parental dyslipidemia and compared these values with lipid profile of adolescents with no history and biochemically proven parental dyslipidemia to assess whether there is any association between lipid levels of parents with their offspring.

Analyzed data showed that there were significant differences ($p < 0.05$) of TG and HDL-c between cases and controls in this study. The mean of TG in cases and controls were 168.98 and 157.22 mg/dl respectively which were much higher than the study done by Rahman (2000)[13] (105.81mg/dl) and considerably very much higher than the United States data (72 mg/dl) and the study was done by Mendes et al. (2006)[14] (86.2 and 76.0 mg/dl in cases and control respectively). The mean levels of HDL-c in cases and controls were 39.09 and 40.51 mg/dl respectively in present study which is consistent with the study done by Gulati and Suxena (2003)[4] (mean 38.8 and by Rahman (2000)[13] in Bangladeshi children (36.12 mg/dl) and lower than the data from similar study by Mendes et al. (2006)[14] (44.5 and 51.2 mg/dl in cases and control respectively) and much lower than the United States data (52.6 mg/dl) and Schulpis and Karikas (1998)[13] (59-64 mg/dl). Recent studies showed that high TG and low HDL-c

may be an atherosclerotic determinant[15]. The mean plasma TC levels were similar to study done by Schulpis and Karikas (1998)[16] (157 mg/dl in boys and 172 mg / dl in girls) and slightly lower than United States data (170 mg/dl)[17]. No significant difference was observed in TC level between cases and control in this study.

In present study, the mean levels of LDL-c in cases and controls were 90.41 and 86.42 mg/dl respectively which were somewhat similar to the study done by Gulati and Saxena (2003)[4] and was considerably low than the similar study done by Mendes et al. (2006)[14] (144.2 and 100.4 mg/dl in cases and control respectively). There was no significant difference in LDL-c levels in this study between the cases and controls.

Dyslipidemia is a well established risk factor for cardiovascular disease. Mendes et al. (2006)[14] found significantly higher levels of TC, LDL-c and lower level of HDL-c in a study, but no significant difference was found in level of TG between cases and controls. In this study we found significantly higher levels of TG and lower levels of HDL-c in cases compared to controls but no significant difference were observed in TC and LDL-c. The comparatively lower levels of TC, HDL-c and higher levels of TG in the current study are consistent with the observations of generally lower values in less developed countries[15].

Conclusion

The findings of this study reflect the association of offspring dyslipidemia with parental dyslipidemia, probably due to some genetic predisposition. Offspring of dyslipidemic parents have higher levels of lipid profile status compared to age and sex matched controls. If parents with dyslipidemia become more conscious about their status and lead life more carefully, the rate of dislipidemic offspring will be lower.

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