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SERUM LIPID PROFILE;

SIGNIFICANCE OF RISK FACTORS REGARDING OFFSPRINGS OF PREMATURE CORONARY HEART DISEASE PATIENTS

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ABSTRACT… Objectives: This study was conducted to determine serum lipid profile in children of premature coronary heart disease patients and compare results between children having parents with one, two or three risk factors. **Study Design:** A cross sectional comparative study. **Patients and Methods:** Ninety seven (97) subjects having parents with one risk factor, sixty three (63) subjects having parents with two risk factors and sixty five (65) subjects having parents with three risk factors were selected from Punjab Institute of Cardiology Lahore. Fifty (50) age and sex matched subjects of parents without a history of coronary heart disease were also selected. The serum total cholesterol, serum triglycerides, serum low density lipoprotein cholesterol and serum high density lipoprotein cholesterol was performed and results were compared. **Results:** Offsprings of parents having more number of risk factors had statistically significantly high total cholesterol and low density lipoprotein cholesterol as compared with offsprings of parents having one risk factor. Triglycerides and high density lipoprotein cholesterol of offsprings of parents having one, two, three or more than three risk factors were comparable. Offsprings of parents having premature coronary heart disease (CHD) had abnormal lipid levels as compared with control group. **Conclusions:** Coronary heart disease risk factors are significant regarding abnormal lipid levels. Offsprings of premature CHD patients are prone to develop CHD as compared to normal control group. Genetic predisposition seems to be important in development of CHD.

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INTRODUCTION

The development of the concept of risk factors and their relationship to the incidence of coronary heart disease (CHD) evolved from prospective epidemiological studies in the United States and Europe. The studies demonstrated consistent association among characteristics observed at one point in the time in apparently healthy individuals with the subsequent incidence of CHD[1]. The Literature has emphasized the importance of risk factors in determining outcome and heterogeneity of patients of CHD[2]. Being a complex multifactorial disease CHD results from an interaction between the individuals genetic background and many environmental factors[3]. Haslett et al[4] have described age, male gender and family history as non-modifiable risk factors and hypercholesterolemia, hypertension, diabetes mellitus, smoking, obesity and sedentary life style as modifiable risk factors. Age, male sex and hypercholesterolemia are established risk factors for CHD[5]. Family history of CHD is highly associated with disease incidence and prevalence[6]. Hypertension has frequent association with CHD[7]. Diabetes mellitus patients are at high risk for developing CHD[8].The smoking is an important risk factor for developing CHD[9]. There is inverse association between physical activity and risk of CHD[10]. Increase in serum cholesterol is associated with risk of CHD[11]. High

level of low density lipoprotein cholesterol (LDL-c) and decreased level of high density lipoprotein cholesterol (HDL-c) are important in development of CHD[12]. Hypertriglyceridemia is well known for development of CHD[13].

The study was performed to determine serum lipid profile in children of premature CHD patients and compare the results between children having parents with one, two or three (or more than three) risk factors.

PATIENTS AND METHODS

Ninety seven (97) subjects having parents with one risk factor (Group I), sixty three (63) subjects having parents with two risk factors (Group II) and sixty five (65) subjects having parents with three risk factors (Group III) were selected from Punjab Institute of Cardiology Lahore. Fifty (50) age and sex matched subjects of parents without a history of coronary heart disease (control group) were also selected from various areas of Lahore. The study was carried out in adolescent group. World health organization (WHO) puts adolesence from 11-12 years[14]. CHD was diagnosed on basis of clinical finding, electrocardiography and biochemical findings including creatine kinase (CK), creatine kinase MB (CK-MB) aspartate amino transferase (AST) and lactate dehydrogenase (LDH). The upper age limit for diagnosis of premature CHD was taken as 45 years for males and 50 years for females. Subjects suffering from diabetes mellitus, thyrotoxicosis, hypertension and hepatitis were excluded from study. 5 ml blood was drawn aseptically avoiding stasis, after overnight fast. Blood was clotted and centrifuged to obtain serum. The serum was analyzed in the laboratory on FP901 analyzer using commercially purchased kits. The serum total cholesterol (TC) was estimated by cholesterol oxidase (CHOD) method, serum triglycerides (TG) was estimated by glycerophosphate oxidase method, serum low density lipoprotein cholesterol (LDL-c) was estimated by CHOD method after heparin precipitation. While high density lipoprotein cholesterol (HDL-c) was estimated by CHOD method after precipitation with phosphotungstic acid and Mg chloride.

Arithmetic mean and Standard deviation were calculated. Data analysis was done by student's 'T' test. P value was read from specific tables.

RESULTS AND OBSERVATIONS

Serum lipid profile of ninety seven (97) children of parents having one risk factor (Group I), sixty three (63) children of parents having two risk factors (Group II) and sixty five (65) children of parents having three risk factors or more than three risk factors (Group III) was determined. Sex distribution is given in table 1 fig. 1. Age distribution is given in table 2 fig. 2. Age has been expressed as mean $\pm$ SD. Mean $\pm$ SD age in children of parents having one risk factor (Group I) was 14.9 $\pm$ 3.0, 15.46 $\pm$ 2.85 and 14.2 $\pm$ 2.8 for all subjects, males and females respectively. Mean $\pm$ SD age in children of parents having two risk factor (Group II) was 15.5 $\pm$ 2.9, 14.52 $\pm$ 3.15 and 16.8 $\pm$ 3.18 for all subjects, males and females respectively. In children of parents having three or more than three risk factors (Group III) mean $\pm$ SD ages for all subjects, males and females was 16.5 $\pm$ 2.9, 16.71 $\pm$ 3.09 and 16.2 $\pm$ 2.74 respectively.

On comparison between group I and group II the age was comparable between males and all subjects of both groups. However, mean $\pm$ SD age of females of group II was raised statistically highly significantly as compared with group I. Mean $\pm$ SD ages of males, females and all subjects of group III were raised significantly statistically as compared with respective subjects of group I. Comparison between group II and group III showed the mean $\pm$ SD age was comparable between female subjects of two groups while mean $\pm$ SD ages of males and all subjects of group III were raised significantly statistically with respective subjects of group II.

Comparison of serum total cholesterol (TC) between group I, group II and group III is given in table-III and fig-3.

Serum TC was when compared between all subjects of groups I and II, the difference was not significant statistically. When compared between all subjects of groups I and III the serum TC was

increased in group III and difference was significant statistically. Serum TC was increased in group III when compared with group II and the difference was significant statistically. The Serum TC was increased in male subject of group I when compared with male subjects of group II and difference was significant statistically.

TC was increased in-group I males when compared with group III males and difference was significant statistically.

TC comparison between male subjects of groups II and III showed non-significant difference statistically. Serum TC was compared between female subjects of groups I, II and III and in all three comparisons difference was not significant statistically.

Comparison of serum triglycerides (TG) between group I, group II and group III is given in table 4 and fig4. Serum TG when compared between all subjects of groups I and II, group I and group III and group II and group III, there was no-significant difference found in all of three comparisons statistically. Serum TG when compared between males of groups I and II was found increased in males of group II, difference was highly significant. TG when compared between male's subjects of group I and group III significant difference was found statistically. TG when compared between males subjects of group II and group III no significant difference was found statistically. When TG was compared between females subjects of group I, group II and group III, the difference was not significant statistically in any of three comparisons.

Comparison of serum low-density lipoprotein-cholesterol (LDL-c) is given in table 5 fig 5. Serum LDL-c was compared between male subjects of group I and group II, group I and group III, and group II and group III. In above all three comparisons the difference was not significant statistically. LDL-c was compared between female subjects of group I and group II, and difference was not significant statistically. LDL-c was increased in group III when compared with group I, and difference was significant statistically. LDL-c when compared between female subjects of group II and group III no significant difference was found. In all subjects comparison between group I and group II showed not significant difference statistically. LDL-c was increased significant statistically in group III when compared with group I. LDL-c was increased in group III when compared with group II and difference was significant statistically.

Comparison of serum high density lipoprotein-cholesterol (HDL-c) is given in table 6 fig 6. Serum HDL-c was decreased in male subjects of group I when compared with male subjects of group II, the difference was highly significant statistically. HDL-c when compared between male subjects of group I and group III, the difference was not significant statistically. HDL-c when compared between male subject of group II and group III, the difference was not significant statistically. HDL-c was decrease in female of group II when compared with female subjects of group I. the difference was highly significant statistically. HDL-c was decreased in female of group III, when compared with female of group I. the difference was highly significant. HDL-c was compared between females of group II and III and difference was not significant statistically. HDL-c was compared in all subjects between group I and group II, group I and group III and group II and group III. In all above three comparisons the difference was not significant statistically.

Serum total cholesterol, triglycerides, low density lipoprotein cholesterol of children of parents having premature coronary heart disease patients was increased when compared with children of parents with CHD history. table 7 and fig 7.

DISCUSSION

It is generally acknowledged that coronary heart disease (CHD) is a multifactorial disease, a number of factors are involved in its development[15]. There is a clear correlation between the incidence of CHD and existing risk factors[16]. Male sex is established risk factor for CHD, females are sheltered from CHD until

Groups	Males	Females	Total
I (Children of parents having one risk factor)	54 (55.67%)	43 (44.33%)	97
II (Children of parents having two risk factors)	36 (57.14%)	27 (42.86%)	63
III (Children of parents having three or more risk factors)	35 (53.85%)	30 (46.15%)	65

Table-I. Gender distribution

Groups	Males	Females	All the subjects
I (Offsprings of parents with one risk factor)	15.46 ± 2.85 (n=54)	14.2 ± 2.8 (n=43)	14.9 ± 3.0 (n=97)
II (Offsprings of parents with two risk factors)	14.52 ± 3.15 (n=36)	16.8 ± 3.18 (n=27)	15.5 ± 2.9 (n=63)
III (Offsprings of parents with three or more risk factors)	16.71 ± 3.09 (n=35)	16.2 ± 2.74 (n=30)	16.5 ± 2.9 (n=65)
Statistical analysis			
I vs II	NS	HS	NS
I vs III	S	HS	HS
II vs III	HS	NS	S

Table-II. Comparison of age in groups I, II AND III
Number of subjects is given in parenthesis. (Age is expressed as mean ± SD)

Key:
HS =Highly Significant, NS = Non - Significant n = Number of subject, S = Significant

menopause because of beneficial effects of estrogen hormones[17]. There is a high degree of prevalence of premature CHD in offsprings and first degree relatives of premature CHD[18]. Abnormal lipid levels contribute significantly to development of CHD[19]. Hauser et al[20] observed that the hypertension along with diabetes mellitus, smoking, obesity and hypercholesterolemia is a major risk factor for development of CHD. Diabetics are at high risk for developing CHD and death[21]. The most effected system by smoking are respiratory system and cardiovascular system[22]. There is association between sedentary life style and development of CHD while exercise appear to have protective effect which may be related to its ability to increase HDL-c, lower blood pressure reduce blood clotting and promote collateral vessel development[4]. In general CHD chance increases with number of conventional risk factors however some individuals don't show any conventional risk factors, it suggests the contribution of genetic factors[23].

In present study serum total cholesterol of all subject having parents with three or more risk factors was increased as compared to offsprings having parents with one or two risk factors. These results are consistent with laur et al[23] who reported that family history effects lipids.

In present study triglycerides of male subjects having parents with two or three risk factors was increased as compared to offsprings having parents with one risk factor.

Groups	Males	Females	All the subjects
I (Offsprings of parents with one risk factor)	178 ± 25.5 (n=54)	178.9 ± 31.47 (n=43)	176.7 ± 27.92 (n=97)
II (Offsprings of parents with two risk factors)	161.6 ± 20.44 (n=36)	174.1 ± 26.4 (n=27)	175.8 ± 27.96 (n=63)
III (Offsprings of parents with three or more risk factors)	167.5 ± 27.38 (n=35)	184.2 ± 30.34 (n=30)	184.5 ± 27.98 (n=65)
Statistical analysis			
I vs II	HS (p<0.01)	NS (p>0.05)	NS (p>0.05)
I vs III	S (p<0.05)	NS (p>0.05)	S (p<0.05)
II vs III	NS (p>0.05)	NS (p>0.05)	S (p<0.05)

Table-III. Comparison of total cholesterol in offsprings of premature CHD patients on basis of risk factors in parents groups I, II AND III

Number of subjects is given in parenthesis. (Result are expressed as mean + SD)

Key: *HS = Highly Significant, NS = Non - Significant n = Number of subject, S = Significant*

Groups	Males	Females	All the subjects
I (Offsprings of parents with one risk factor)	137.8 ± 39.35 (n=54)	170.16 ± 39.88 (n=43)	158.2 ± 33.79 (n=97)
II (Offsprings of parents with two risk factors)	161.16 ± 44.9 (n=36)	164.1 ± 49.88 (n=27)	160.9 ± 44.87 (n=63)
III (Offsprings of parents with three or more risk factors)	156.6 ± 48.12 (n=35)	164.8 ± 50.30 (n=30)	160.6 ± 42.89 (n=65)
Statistical analysis			
I vs II	HS (p<0.01)	NS (p>0.05)	NS (p>0.05)
I vs III	S (p<0.05)	NS (p>0.05)	NS (p>0.05)
II vs III	NS (p>0.05)	NS (p>0.05)	NS (p>0.05)

Table-IV. Comparison of triglycerides in offsprings of premature CHD patients on basis of risk factors

Number of subjects is given in parenthesis. (Result are expressed as mean ± SD)

Key: *HS =Highly Significant, NS = Non - Significant n = Number of subject, S = Significant*

Groups	Males	Females	All the subjects
I (Offsprings of parents with one risk factor)	108.9 ± 32.03 (n=54)	99.8 ± 27.84 (n=43)	102.5 ± 30.89 (n=97)
II (Offsprings of parents with two risk factors)	105.38 ± 20.8 (n=36)	99.25 ± 23.97 (n=27)	103.5 ± 22.42 (n=63)
III (Offsprings of parents with three or more risk factors)	111.7 ± 32.64 (n=35)	110.8 ± 27.32 (n=30)	111.2 ± 30 (n=65)
Statistical analysis			
I vs II	NS (p>0.05)	NS (p>0.05)	NS (p>0.05)
I vs III	NS (p>0.05)	S (p<0.05)	S (p<0.05)
II vs III	NS (p>0.05)	NS (p>0.05)	S (p<0.05)

Table-V. Comparison of low density lipoprotien cholesterol in offsprings of premature CHD patients on basis of risk factors in parents groups I, II AND III

Key: NS = Non - Significant n = Number of subject S = Significant

Groups	Males	Females	All the subjects
I (Offsprings of parents with one risk factor)	36.0 ± 8.87 (n=54)	45.27 ± 8.62 (n=43)	41.45 ± 8.7 (n=97)
II (Offsprings of parents with two risk factors)	40.47 ± 8.58 (n=36)	39.62 ± 6.42 (n=27)	40.07 ± 7.5 (n=63)
III (Offsprings of parents with three or more risk factors)	38.62 ± 6.75 (n=35)	40.96 ± 9.52 (n=30)	41.41 ± 7.97 (n=65)
Statistical analysis			
I vs II	HS (p<0.01)	HS (p<0.01)	NS (p>0.05)
I vs III	NS (p>0.05)	S (p<0.05)	NS (p>0.05)
II vs III	NS (p>0.05)	NS (p>0.05)	NS (p>0.05)

Table-VI. Comparison of high density lipoprotien cholesterol in off springs of premature CHD patients on basis of risk factors in parents of groups I,II AND III

Key:

HS = Highly Significant, NS = Non - Significant n = Number of subject, S = Significant

Groups	Total cholesterol	Triglycerides	Low-density lipoprotein cholesterol	High-density lipoprotein cholesterol
A (Control group)	136.66 ± 25.37 (n= 50)	108 ± 35.65 (n=50)	70.2 ± 25.67 (n=50)	34.1 ± 11.76 (n=50)
B (Children of premature CHD patients)	179.7 ± 29.85 (n=250)	162.84 ± 47.07 (n=250)	100.09 ± 28.5 (n=250)	37.95 ± 8.87 (n=250)
Statistical Analysis	HS (p<0.01)	HS (p<0.01)	HS (p<0.01)	S (p<0.05)

Table-VII. Comparison of serum Lipid Profile in Groups A and B The result are expressed as mean ± SD

Key:
HS = Highly Significant, n = Number of subject, S = Significant

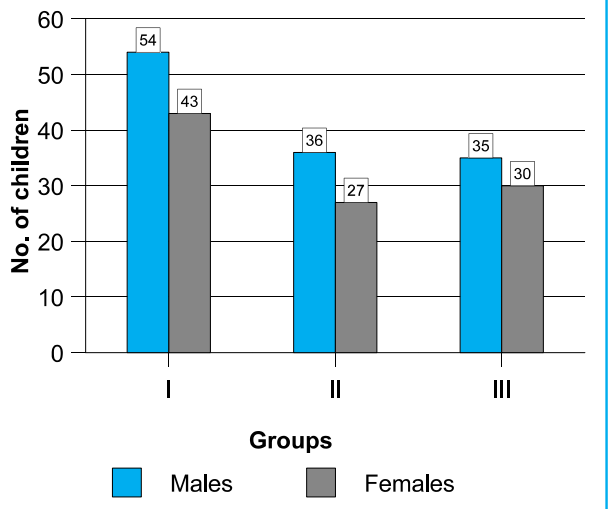

Fig-1. Gender distribution in children having parents with one, two or three risk factors

Key:
I. Offsprings of parents having one risk factor
II. Offsprings of parents having two risk factors
III. Offsprings of parents having three risk factors

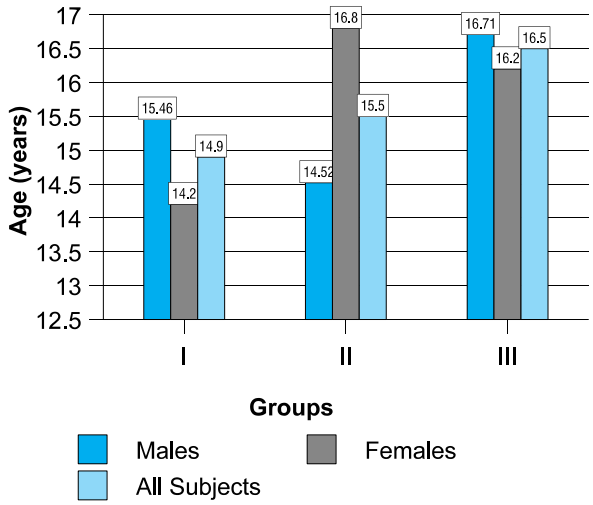

Fig-2. Age Distribution in Groups I, II and III

Key:
I. Offsprings of parents having one risk factor
II. Offsprings of parents having two risk factors
III. Offsprings of parents having three risk factors

In all subjects, serum triglycerides of subjects of subjects having parents with two or three risk factors although not significant statistically but is more then Subjects having parents with one risk factor. Finding of this study are consistent with Austin et al[24].

Genetic Factors of various life styles like smoking over consumption of saturated fats and sedentary life styles are responsible for hypertriglyceridemia[25].

Shah et al[26] reported that there was no significant difference between serum triglycerides of normal control and offsprings of premature CHD patients.

The serum low-density lipoproteins of this study were increased in all subjects having parents with three or more risk factors as compared to

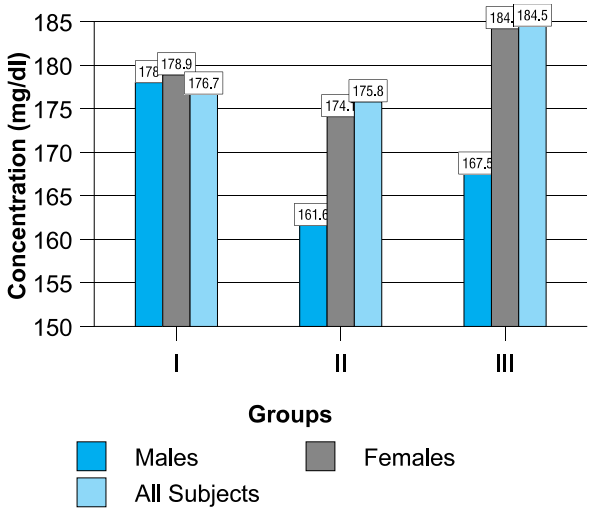

Fig-3. Comparison of Serum TC in groups I, II and III

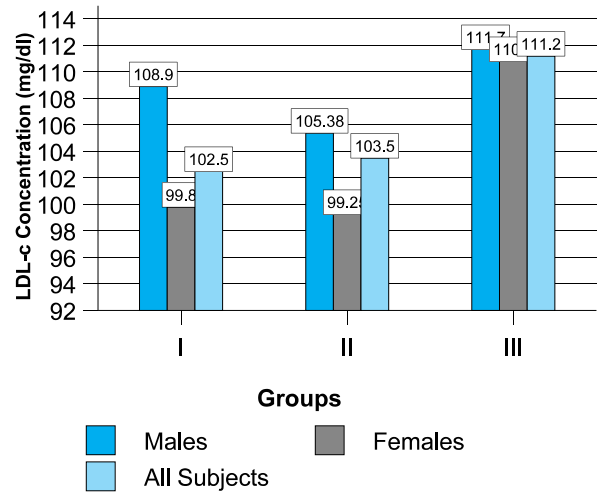

Fig-5. Comparison of LDL-c in groups I, II and III

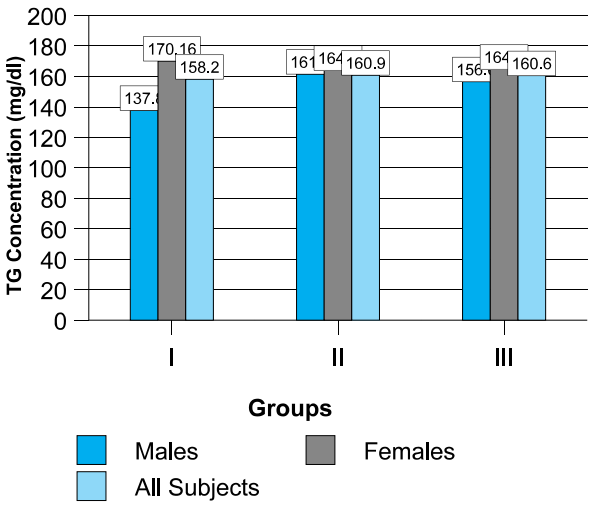

Fig-4. Comparison of Serum TG in groups I, II and III

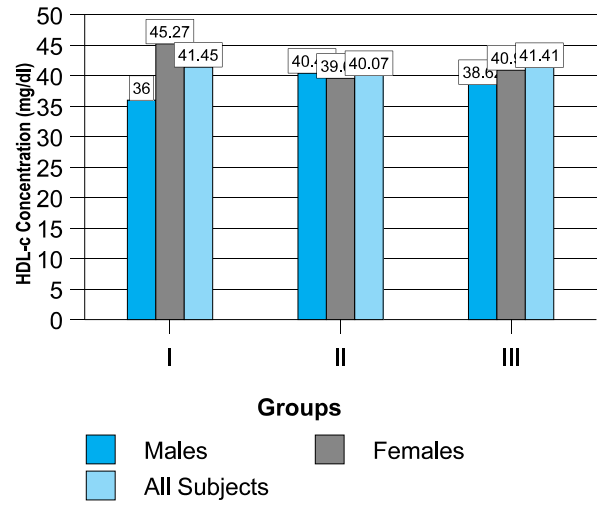

Fig-6. Comparison of HDL-c in groups I, II and III

affsprings having parents with one risk factor. Genest et al[18] have reported aggregation of elevated LDL-c in families.

Mean high density lipoprotein cholesterol (HDL-c) in offspring having parents with one, two ,three or more then three risk factors was compared. The comparison among there groups was non-significant. This was in agreement with shah et al[26] who also reported no significant difference.

The serum lipid profile results of this study highlight importance of risk factors in development of CHD although sometimes genetic predisposition predominates.

CONCLUSIONS

In the light of findings of this study it is concluded that risk factors have an important role regarding development of abnormal lipid levels in offsprings of premature coronary heart disease patients.

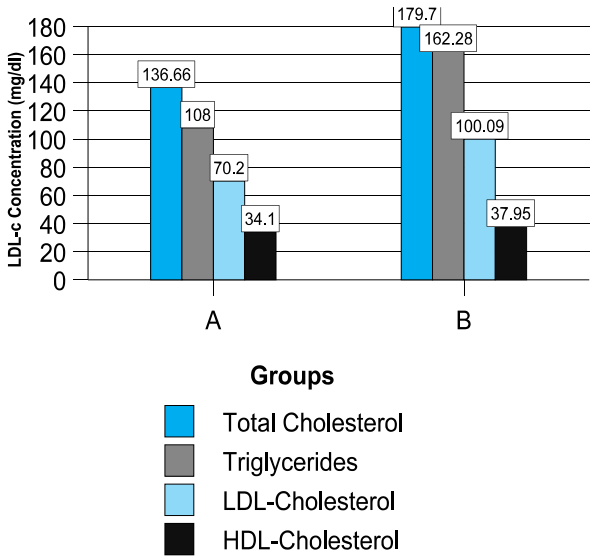

Fig-7. Comparison of serum lipid profile in offsprings without parental history of CHD (Group A) & offsprings of premature CHD patients (Group B)

In some cases where risk factors are not associated with development of lipid abnormalities there genetic predisposition seems to play a role.

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PREVIOUS RELATED STUDY

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"The starting point of all achievement is desire."

Napoleon Hill