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Study of Blood Glucose & Lipid Profile level in premature coronary artery disease

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ABSTRACT

Introduction: Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among the Asian population. According to World Health Organization, the numbers of deaths from CVDs in 2004 were 17.1 million and it is predicted that by 2030, about 23.6 million deaths will be reported due to CVDs, mainly from coronary artery disease (CAD) and stroke. The higher degree of prematurity and the severity of CAD in the Indian population as compared to any other ethnic group in the world can be explained on the basis of conventional lipid parameters. Lipid profile is one of the major risk factors of coronary artery disease and myocardial infarction. Dyslipidemia is a prominent and modifiable risk factor for cardiovascular diseases. Serum lipid levels are strongly correlated with coronary artery disease.

Materials and Methods: The total study group consists of 400 subjects, of which 200 were healthy individuals (controls) & amp; 200 premature coronary artery disease patients were taken as cases. Venous blood was used for analysis. Estimation of Plasma Glucose & lipid profile (Total cholesterol, Triglyceride & HDL) were analyzed on ERBA EM200 fully autoanalyzer. The data analysis was done by using mean, standard deviation & student t-test.

Results: Random plasma glucose (146.15 ± 8.12 vs. 90.66 ± 8.30), Plasma total cholesterol (TC) (198±12.26 vs.162.69±6.78), Triglyceride (TG) (160.41±13.12 vs. 118.10±7.0) & low density

Section: Research Paper
ISSN 2063-5346

lipoprotein cholesterol (LDL-c) (135.65±11.85 vs. 93.22±9.63) were higher, & HDL (30.25 ± 2.70 vs. 45.84 ± 4.38) was lower in CAD patients than control subjects. The level of plasma Glucose, total cholesterol, triglyceride & LDL cholesterol were significantly increased & the level of High-density lipoprotein cholesterol (HDL-c) was significantly decreased in Premature CAD as compared to healthy control group.

Conclusion: Our study shows highly significant elevation of glucose, cholesterol, and triglyceride LDL cholesterol in Study group (premature CAD patients) as compared to the Healthy control group. The increase low density lipoprotein (LDL) cause oxidation through free radicals; the oxidation results in the attachment of monocytes to the vessel wall and also damages to the endothelium, resulting in endothelial dysfunction. The decrease activity of HDL-C leading to accelerated atherogenesis and causes ischemic heart related complications

Key words: coronary artery disease; High-density lipoprotein; Low density lipoprotein; ischemic heart disease.

Introduction:

Ischemic heart disease (IHD) also referred to as coronary artery disease (CAD), this occurs because of an imbalance between myocardial oxygen consumption and myocardial supply. Most often, the imbalance is caused by inadequate blood supply to the heart. (1) The main risk factors for CAD are smoking, obesity, hypertension, diabetes mellitus, and elevated blood lipids.(2) Asians populations are mostly affected by cardiovascular disease (CVD) which is a leading cause of death and morbidity.(3) As per WHO statistics, 17.1 million deaths were caused by CVDs in 2004 and by 2030, 23.6 million deaths will be caused by CVDs, mainly coronary artery disease (CAD) and stroke.(4) Incidence of CAD is increasing at an alarming rate in developing countries.(5) India is in danger of experiencing a cardiovascular pandemic. According to predictions, by the year 2030, CAD will continue to be the biggest and most frequent threat to human existence. (6, 7)

Atherosclerosis is the primary cause of coronary artery disease. Impaired endothelial function is a significant factor for atherosclerosis. Reactive oxygen species & other risk factors defective the function of endothelial.[8]Thrombus formation& endothelial damageis important in the initiation of atherosclerosis and

Vascular effects of hypertension and other diseases**.** (9) Based on conventional lipid parameters, the Indian population has a higher level of prematurity and severity of CAD than any other ethnic group in the world. (10) As compared to the westerners, Indians generally tend to have a higher level of low density lipoprotein (LDL) and a lower level of high density lipoprotein (HDL). [11, 12, 13] Among the major risk factors for coronary artery disease and myocardial infarction is lipid profile. Cardiovascular diseases are commonly caused by dyslipidemia, a modifiable risk factor. [14] The levels of lipoproteins in the plasma are increased or decreased in dyslipidemia. [15] Strong correlation exists between coronary artery disease and serum lipid levels. [16] Lipid accumulations from lipoproteins lead to vascular injury, atheroma, and thrombus formation in ischemic heart disease.[17] The oxidative theory also suggests that the process starts with the oxidation of low density lipoprotein (LDL) by free radicals, which causes monocytes to adhere to the vessel wall and endothelium injury that leads to endothelial dysfunction. [18, 19] The cycle of events keeps repeating, the

plaque develops a stable structure as a result of the formation of a fatty centre that is surrounded by a fibrous matrix. [20]

Materials and Methods

Study Design:

The present study was hospital based cross sectional observational study, which had carried out in the Department of Biochemistry, SGT Medical College, Gurugram. The subjects for the study included from Medicine OPD SGT Medical College, Gurugram & Cardiology department of SGPGI, Lucknow. The written consents were taken from the patients prior to the study & the objectives of the study were fully explained. The written informed consent was taken from the subjects to be included in the study. The clearance was taken from institutional ethics committee of FMHS, SGT University.

Sample Size:

Sample Size Calculation for prevalence study:

$$n = \frac{Z^2\,P\,(1-P)}{d^2}$$

n = Sample Size

Z = Z Statistic for a level of confidence

P = Expected prevalence or proportion

d = Precision

Level of confidence = 95%

Disease prevalence in India = ~ 9%

Sample size calculated from the above given formulae = 197

Study groups

The study included a total 400 subjects; which was divided in to two groups. The first group has 200 cases & second group has 200 controls. Selection of cases was done on the basis of ECG graph & cardiac markers.

Group I (Controls): This group had 200 Age & gender matched healthy individuals

Group II (Cases): This group had 200 Patients of either gender suffering from premature CAD (Males <55 years & female<65years in females)

Premature coronary artery disease according to American Heart Association (AHA) defined as artherosclerotic narrowing of coronary arteries. (Males <55 years & in females <65years)

Exclusion criteria for cases

1. Any other Acute /Chronic inflammatory disorder
2. Smoking & Alcoholism
3. Recent use of lipid lowering drugs & corticosteroids
4. Pregnant or lactating women.

Section: Research Paper
ISSN 2063-5346

Exclusion criteria for Controls

1. Any other Acute /Chronic inflammatory disorder
2. Smoking & Alcoholism
3. Pregnant or lactating women.

Objectives:

- Estimate Random blood sugar (RBS) & Lipid profile in premature coronary artery disease patients & controls.
- Compare the result of RBS & Lipid profile in premature coronary artery disease patients & controls.

Sample collection:

Five ml blood will be collected from the patients as well as controls after taking appropriate aseptic precaution. The sample was collected in fluoride & plain vacutainer for the estimation of various parameters.

Methods:

- Estimation of Plasma Glucose by glucose oxidase and peroxidase (GOD-POD), commercially available kit from ERBA Diagnostics Mannheim, Germany. [21]
- Estimation of Serum Total Cholesterol by (CHOD-PAP), commercially available kit from ERBA Diagnostics Mannheim, Germany[22]
- Estimation of Serum Triglycerides by GPO Trinder method , commercially available kit from ERBA Diagnostics Mannheim, Germany[23]
- Estimation of Serum High-Density Lipoprotein (HDL) by PVS and PEGME method , commercially available kit from ERBA Diagnostics Mannheim, Germany[24]

Statistical Analysis

Data and various parameters will be analysed on SPSS software (USA inc.) version 23. Mean and standard deviation of all parameters will be calculated. Chi square test will be applied to non-parametric variables. Student t-test will used to compare averages in two groups

Results:

Table 1: Comparison of RBS in Control Group & Cases Group

Parameter	Group-I (Healthy Control) Mean± SD	Group -II (Premature CAD) Mean± SD	P value

RBS (mg/dl)	90.66±8.30	146.15±8.12	(p<0.001) Highly significant

Table 2: Comparison of Lipid Profile in Control Group & Cases Group

Parameters	Group-I (Healthy Control) Mean± SD	Group -II (Premature CAD) Mean± SD	P value (p<0.001) Highly significant
Total Cholesterol (mg/dl)	162.69±6.78	197.99 ±12.26	
Triglyceride (mg/dl)	118.10±7.0	160.41 ±13.12	
HDL-C (mg/dl)	45.84±4.3	30.25 ±2.70	
LDL (mg/dl)	93.22±9.63	135.65 ±11.85	

Section: Research Paper
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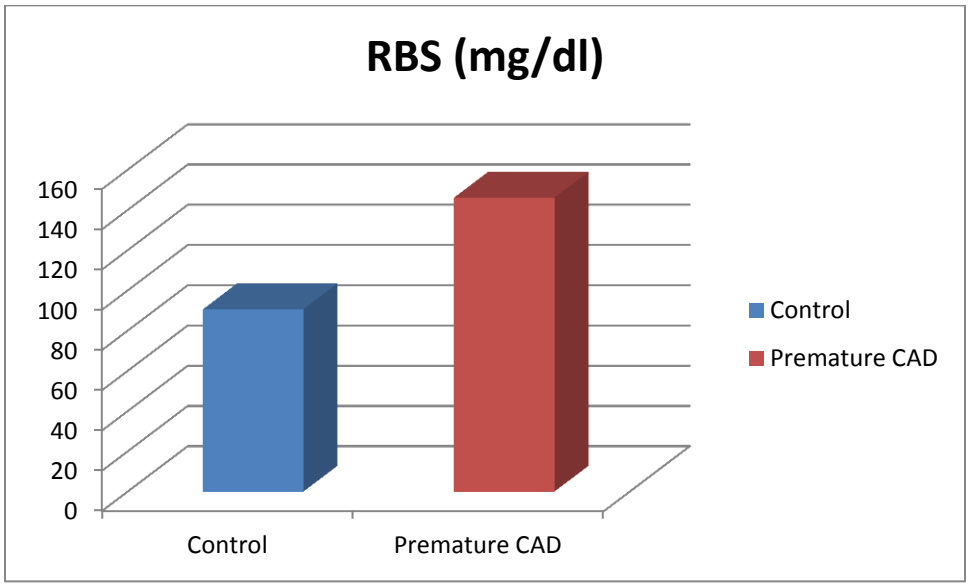

Fig 1: Shows Random Blood Sugar level in control & Case group

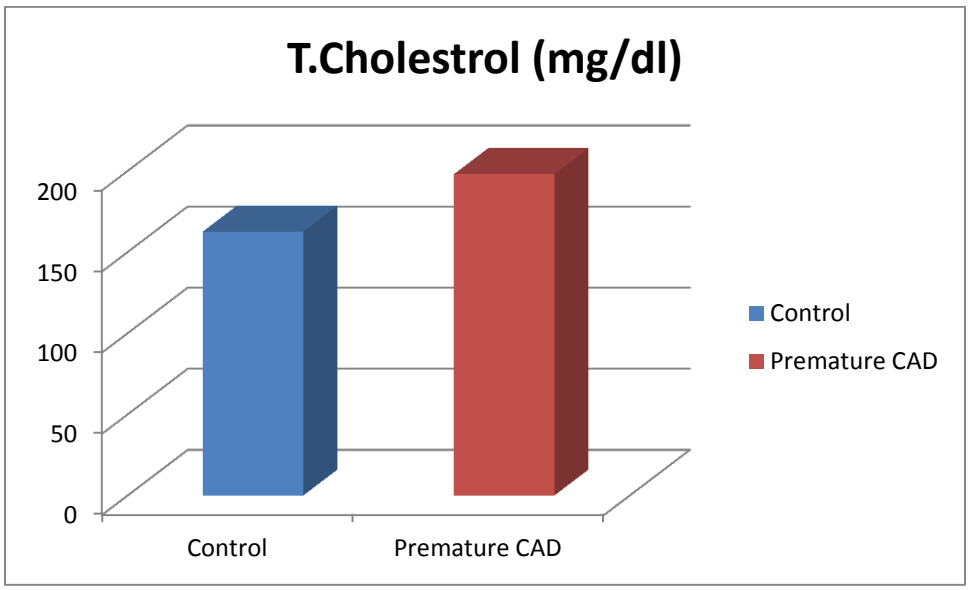

Fig 2: Shows Total Cholesterol level in control & Case group

Section: Research Paper
ISSN 2063-5346

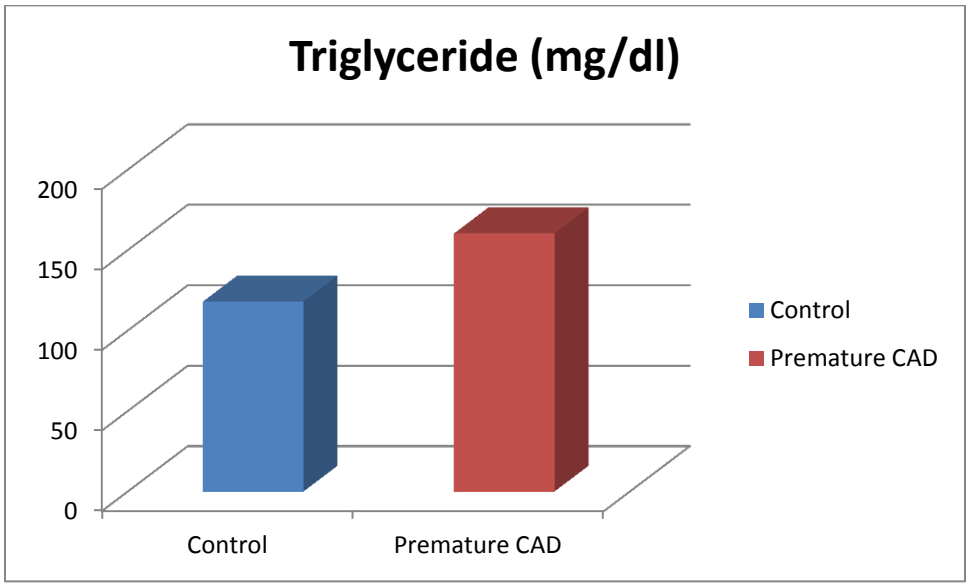

Fig 3: Shows Serum Triglyceride level in control & Case group

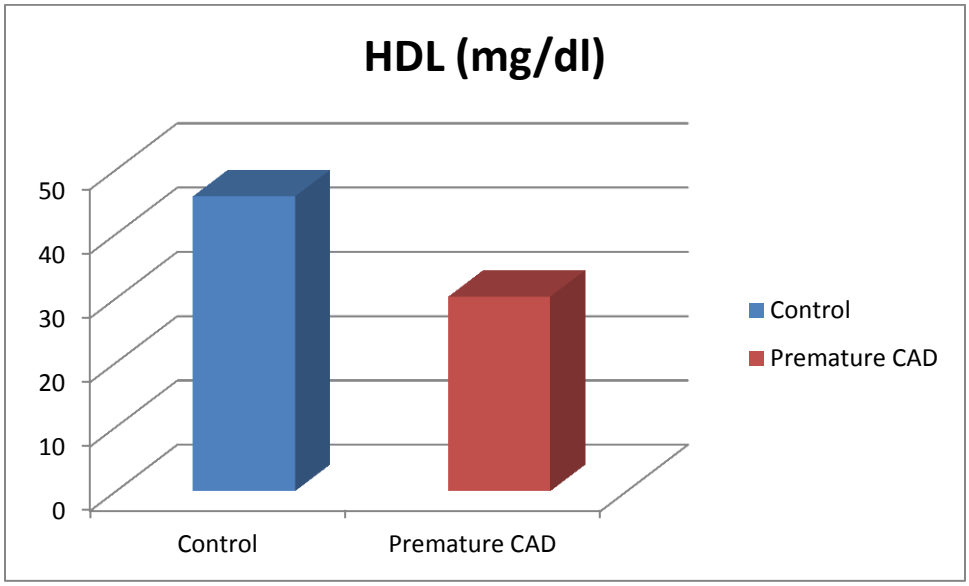

Fig 4: Shows Serum HDL level in control & Case group

Section: Research Paper
ISSN 2063-5346

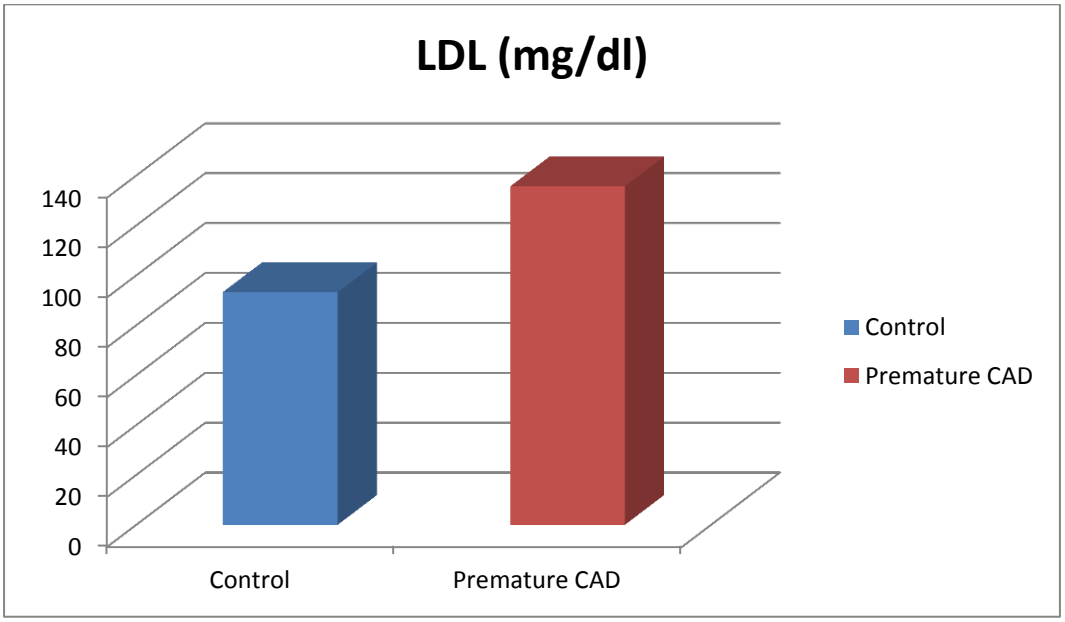

Fig 4: Shows Serum LDL level in control & Case group

Discussion:

The pathogenesis of CAD is strongly associated with dyslipidemia. It has been known for many years that abnormal lipid parameters are risk factors for coronary artery disease. Several studies have demonstrated the importance of the conventional lipid parameters TC, LDL, HDL, and TG in the pathogenesis of atherosclerosis, an underlying pathology in coronary artery disease.

The levels of plasma glucose in the cases were significantly higher than in the controls. The results shown in Table 1 of RBS in controls & compared the result with CAD patients. The increased plasma glucose caused increased generation of reactive oxygen species.

Cheraghi M et al 2019; conducted a study of 50 CHD patients and 50 healthy volunteers. RBS levels activities were significantly greater in CHD patients than in healthy controls.[25]

Sowmya K et al 2011; conducted a study group of 60 subjects, of which 30 were healthy individuals & 30 angiographically proven coronary artery disease patients were taken as cases. Plasma glucose was increased in CAD patients as compared to healthy controls.[26]

Our results for plasma glucose are supported by Cheraghi M *et al* & suggest greater risk of atherosclerosis and cardiovascular disease. The results shown in Table 2 of lipid profile in controls & CAD patients. Serum total cholesterol, LDL-cholesterol, triglycerides were significantly increased whereas HDL cholesterol levels were decreased in CAD Patients as compared to healthy controls.

As a result of greater triglyceride concentrations, VLDLc is produced more rapidly, and clearance of triglycerides is reduced. Patients with CAD have abnormal lipid profiles, which confirms the severity of

the disease. **Gupta S et al 2009;** Conducted a study of patients with coronary artery disease (n = 100) & normal healthy controls (n = 50). The results of the study clearly indicate dyslipidemia in CAD patients; when compared with controls. The abnormal lipid profile levels in CAD patients confirm the severity of the disease.[27] **Sowmya K et al** 2011; conducted a study group of 60 subjects, of which 30 were healthy individuals & 30 angiographically proven coronary artery disease patients were taken as cases. Total cholesterol , Triglyceride & LDL cholesterol were significantly increase in Premature CAD as compared to the control group (P<0.0001). While High-density lipoprotein cholesterol (HDL-c) significantly decreased in Premature CAD patients as compared to Healthy controls (p<0.0001).The study clearly represent the dyslipidemia .[26] Our results for serum lipid profile are supported by **Gupta S et al**. The abnormal lipid profile in CAD patients caused dyslipidemia & suggests greater risk of atherosclerosis

Conclusion:'

Our study shows highly significant elevation of glucose, cholesterol, and triglyceride & LDL cholesterol in Study group (premature CAD patients) as compared to the Healthy control group. The increase low density lipoprotein (LDL) cause oxidation through free radicals; the oxidation results in the attachment of monocytes to the vessel wall and also damages to the endothelium, resulting in endothelial dysfunction. The decrease activity of HDL-C leading to accelerated atherogenesis and Causes ischemic heart related complications.

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