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Fasting and Postprandial Level of Lipid Profile in Cardiovascular Disease (CVD) At CCM Medical College Durg (CG)

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Abstract:

Purpose: *- The present study examines the Fasting and Postprandial Level of Lipid Profile in Cardiovascular Disease at CCM medical college Durg.*

Methods: *- A total number of 227 male subjects were selected at CCM Medical College Hospital Durg. 127 subjects are suffering from cardiovascular disease average range of 32 to 60 years. 100 subjects age, sex matched was selected for control group. Controls were clinically and physically normal and healthy*

Results: *- The Fasting levels of total cholesterol, triglycerides and VLDL-C in patients of CVD are significantly higher as compared to normal healthy controls ($p < 0.001$). The level of Fasting HDL-C in CVD patients is lower as compared to normal healthy controls but not significantly and the LDL-C is increased significantly in CVD patients as compared to healthy controls in fasting state. Ail parameters of lipid profile is significantly higher in CVD patients than controls ($p < 0.001$). In our study the lipid ratios of TC: HDL-C: LDL-C: HDL-C found higher in both fasting and postprandial (table 2.1 & 2.3) state in patients with cardiovascular disease than control and also showed higher in CVD patient whereas raised in both group patients than control group patients.*

Conclusion: *- On the basis of our study results we concluded that the monitoring and management of dyslipidemia in cardiovascular disease patients and the data suggest that assessment and treatment. Impaired lipoprotein metabolism is one of the major an etiological factors for the pathogenesis of CVD. Assessment is usually made in the fasting state but during the postprandial period the massive amount of lipid fluxes through the intra vascular compartment. Thus, the higher TG and VLDL-C and lower HDL-C levels are better indicators of CVD than the classical risk factors like total cholesterol and LDL-C supporting the hypothesis that postprandial lipoprotein metabolism and their catabolic rate play a crucial role in the development and progression of atherosclerosis.*

Keywords: ***- Total Cholesterol, Triglyceride, High density lipoprotein, Low density lipoprotein, Very Low density lipoprotein, CVD (Cardiovascular disease), CHD (Coronary Heart Disease) and atherosclerosis***

Introduction

Cardiovascular diseases (CVD) encompass any medical conditions related to the heart and blood vessels. CVD is the main cause of disability and premature death worldwide, and is projected to remain the leading cause of death. An estimated 17.5 million people died from his disease in 2005, representing 30% of all global deaths.

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Of these deaths, 7.6 million were due to coronary heart disease (CHD) and 5.7 million because of stroke. If immediate and proper attention is not paid, by 2015 an estimated 20 million people will die from CVD, including stroke.[1] Coronary heart disease (CHD) is widely prevalent both in the developed and developing countries and continues to be a leading cause of mortality despite recent advances in diagnostic facilities and treatment modalities. It is a multifactorial disease where atherosclerosis and dyslipidemia are the prominent causes involved.[2] Hypercholesterolemia and hypertriglyceridemic are considered the independent risk factors but most of the earlier studies in this area have considered only the fasting

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lipids and lipoproteins. Recently it has been proposed that postprandial lipoproteins may be better indicators of deranged lipoprotein metabolism and hence of atherosclerosis and CHD.[3] postprandial hypertriglyceridemic (PHTG) and delayed triglyceride (TG) rich lipoprotein clearance have been found to impair endothelial function significantly either directly or by increasing superoxide anions. As these lipoproteins are rich in cholesterol as well as triglyceride content, their uptake by macrophages can result in formation of cholesterol laden foam cells. It has also been reported that magnitude and duration of postprandial lipidaemia is positively related to the pathogenesis and progression of CHD.[4,5]

Risk Factors of Cardiovascular Diseases

The majority of Cardiovascular diseases (CVD) is caused by risk factors that can be controlled, treated or modified, such as high blood pressure, cholesterol, overweight/obesity, tobacco use, lack of physical activity and diabetes. In terms of attributable deaths, the leading CVD risk factor is raised blood pressure (to which 13 % of global deaths is attributed), followed by tobacco use (9 %), raised blood glucose (6 %), physical inactivity (6 %) and overweight and obesity (5 %).

Risk Factors:

Hypertension (high blood pressure)

High blood pressure is defined as a repeatedly elevated systolic pressure of 140 or higher OR a diastolic pressure of 90 or higher. Hypertension is one of the most important causes of premature death worldwide and the problem is growing; in 2025, an estimated 1.56 billion adults will be living with hypertension. Hypertension is the leading cause of CVD worldwide.[6,7]

Tobacco Use

Smoking is estimated nearly 10% of all CVD. The risk of developing CVD is higher in female smokers, young men, and smokers. Within two years of quitting, the risk of coronary heart disease is substantially reduced, and within 15 years the risk of CVD returns to that of a non-smoker.[6]

Raised blood glucose (diabetes)

CVD accounts for about 60 % of all mortality in people with diabetes. The risk of cardiovascular events is form two the three higher in people with type 1 or type-2 diabetes and the risk is disproportionately higher in women. In some age groups, people with diabetes have a two-fold increase in the risk of stroke. Cardiovascular risk increased with raised glucose values.[6]

Physical Inactivity

Insufficient physical inactivity is the fourth leading risk factor for mortality. People who are insufficiently physical active have 20 to 30 minutes of moderate intensity physical active most day of the week. In 2008, 31.3 percent of adults aged 15 or older (28.2 percent women) were insufficient physical active.[6]

Unhealthy Diet

Higher dietary intake of saturated fat, trans-fats and low intake of fruits, vegetables and fish are linked to cardiovascular risk. Frequent consumption of high-energy foods, such as processed foods that are high on a fats and sugars, promotes obesity compared to low-energy foods.[2] High consumption of saturated fats and trans-fatty acid is linked to heart disease; elimination of trans-fat and replacement of saturated with polyunsaturated vegetable oils lowers coronary heart disease risk.[6]

Cholesterol/lipids

Raised blood cholesterol increased the risk of the heart disease and smokers. Globally, one third of ischemic heart disease is attributable of high cholesterol. In 2008, the prevalence of raised total cholesterol among adults defined as total cholesterol of 6.2 240 mg/dl or higher was 9.7% (8.5 % in males and 10.7 % in females). The prevalence of raised total cholesterol noticeably increases according to the income level of the country.[6]

While cardiovascular disease can refer to many different types of heart or blood vessel problems, the term is often used to mean damage caused to your heart or blood vessel by atherosclerosis a build-up of fatty plaques in your arteries. This is a disease that affects your arteries. Arteries are blood vessels that carry oxygen and nutrients from your heart to the rest of your body. Healthy arteries are flexible and strong. Over time, however, too much pressure in the arteries can make the walls thick and stiff sometimes restricting blood flow to the organs and tissues. This process is called hardening of the arteries called as arteriosclerosis. Arteriosclerosis is the most common form of this disorder. Arteriosclerosis is also the most common cause of cardiovascular disease, and it's often caused by an unhealthy diet, lack of exercise, being overweight and smoking. All of these are major risk factors for developing arteriosclerosis and, in turn, cardiovascular disease.

Role of Fasting and Postprandial Lipid

Human nature and unlimited access to food are a combination that causes Western people to live in constant postprandial state, i.e. the time period between food ingestion. However, most of the studies on lipoprotein metabolism have concentrated on the post absorbed fat has been reached. Such studies monitor the endogenous pathway of lipid metabolism. Studies in the postprandial state, however, take into account the metabolic steps in both the exogenous and the endogenous pathway.

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In current laboratory practice lipid profile are measured in the fasting state specifically to exclude the possibility of postprandial effects. Controversy exists, however, regarding the clinical usefulness of fasting lipid profile as an independent predictor of risk of cardiovascular disease (CVD). Measurement of lipid profile in the fasting state has systematically underestimated the impact of hypercholesterolemia, hypertriglyceridemia in clinical practice. Increased levels of cholesterol and triglyceride rich remnant lipoproteins in fasting plasma are associated with increased risk of coronary artery disease.[8] Despite many studies showing that postprandial triglyceride-rich lipoproteins play a central role in the pathogenesis of atherosclerosis, suitably standardized methods to measure postprandial lipidaemia or remnant lipoprotein in the clinical setting are lacking.

A number of reports which will be discussed later point to an association between impaired metabolism of postprandial triglyceride-rich lipoproteins and the presence and development of CAD. A mechanistic hypothesis linking the postprandial generation of TRL remnant to the development of atherosclerosis was formulated almost 20 years ago by Zolversmit.[9] Firstly, the perturbations of lipoprotein in metabolism in the postprandial state are very complex, with changes in both composition and concentration of potentially atherogenic lipoproteins. Secondly, there is no consensus of how a standardized postprandial state should be elicited. Thirdly, it is probable that the close statistical association between fasting plasma triglycerides and the accumulation of TRL in the postprandial state will lead to a situation similar to the one for the association between plasma triglycerides and CAD, i.e. conventional statistical methods seem to be inappropriate or misleading in the study of causal relationship between risk factor and disease.[10] Therefore, the present study was undertaken to evaluate the role of fasting and postprandial lipid profile as an indicator of the efficiency of lipoprotein metabolism and its relationship with development of CVD

Material and methods

A total number of 227 male subjects were selected at CCM Medical College Hospital Durg. 127 subjects are suffering from cardiovascular disease average range of 32 to 60 years. 100 subjects age, sex matched was selected for control group. Controls were clinically and physically normal and healthy.

Table1. Distribution of Study group subjects

Groups	No of Subjects
Normal Healthy Group	100
CVD patients	127
Male CVD patients	86
Female CVD patients	41

Sample Collection

Fasting and postprandial 5 ml blood sample was collected in plain dry test tube. Serum sample was obtained by centrifugation and sample were immediately separated into another plain dry test tube and stored at -200 C. Serum sample was used to estimate serum total Cholesterol (TC), serum Triglyceride (TG), serum low density lipoprotein (LDL-C) and serum High density lipoprotein (HDL-C) by using fully autoanalyser by enzymatic , colorimetric method. In addition, routine investigations like hematological profile, blood urea, serum electrolytes, serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and serum uric acid levels were also carried out in fasting samples of all the subjects. Blood sugar was analyzed using GOD-POD method, blood urea by DAM method, serum sodium/ potassium by flame photometry, SGOT, SGPT by kinetic method and uric acid by enzymatic method.

Data were expressed as mean ±SD. Mean values were assessed for significance by unpaired student –t test. A statistical analysis was performed using the Stastical Package for the Social Science program (SPSS, 21.0). Frequencies and percentages were used for the categorical measures. Probability values $p < 0.05$ were considered statistically significant.

Observation and Results

There were 86 males and 41 females patients selected for study group and 100 normal health for control group. Hb level in the control group ranged between 10-14 gm%, while in the study group, it ranged between 9.0-14.0 gm%. Total leukocyte count ranged from 5000-15000/mm in both the groups with a mean value of 9725.0/mm in controls and 9963.9/mm in the study group. Levels of blood urea, serum electrolytes, SGOT, SGPT and serum uric acid are given in following table.

Table 2. Routine investigations in patients of cardiovascular disease and controls

Biomedical Parameters	Control Subjects (Mean ±SD)	CVD patients (Mean ±SD)	"P" value
SGOT (IU/L)	22.70 ±5.92	41.23 ±17.2	<0.001
SGPT (IU/L)	17.68 ±5.60	38.9 ±15.61	<0.001
Blood Urea (mg/ dl)	19.52 ±4.16	21.38 ±4.59	<0.01
Uric acid (mg/ dl)	6.12 ± 1.03	5.97 ±089	<0.01

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Sodium (meq/ l)	139.9 ± 5.35	138.52 ±5.34	<0.01
Potassium (meq/ l)	4.12 ±0.57	3.98 ±0.68	<0.01
Fasting Blood Glucose (mg/ dl)	82.29 ± 17.92	116.4±18.3	<0.001
Postprandial Blood Glucose(mg/ dl)	97.49 ± 23.94	124.28 ±22.37	<0.001

Table3. Lipid Profile investigation in CVD patients and control group subjects.

Groups		TC (mg/ dl)	TG (mg/ dl)	LDL-C	VLDL-C	HDL-C
Control Group (Mean ±SD)	Fasting	168.6±30.9	109.2±35.4	127.5±40.9	26.8±7.1	42.4±4.9
	Postprandial	166.0±34.3	119.9±38.4	119.1±36.3	32.4±8.4	38.3±5.4
Patient Group (Mean ±SD)	Fasting	206.6±45.8	193.4±97.6	159.7±48.8	38.6±18.2	39.7±4.2
	Postprandial	199.1±39.3	248.9±122.8	124.6±39.6	56.1±31.4	32.8±5.6

Table 3:- Shows that the Fasting levels of total cholesterol, triglycerides and VLDL-C in patients of CVD are significantly higher as compared to normal healthy controls ($p < 0.001$). The level of Fasting HDL-C in CVD patients is lower as compared to normal healthy controls but not significantly and the LDL-C is increased significantly in CVD patients as compared to healthy controls in fasting state. Ail parameters of lipid profile is significantly higher in CVD patients than controls ($p < 0.001$).

Discussion

In the present study, the patients of CVD had significantly higher levels of fasting blood glucose than the healthy controls ($p < 0.001$) although these are within the normal range suggesting that elevated, non-diabetic fasting glucose level may be associated with CVD. Various authors also have reported an increased risk of CVD in upper percentiles of fasting glucose distribution.[11,12] Fasting levels of triglycerides, VLDL-C and total cholesterol in patients of CHD are significantly higher as compared to those in controls ($p < 0.001$). Fasting HDL-C in CHD patients is lower as compared to that in controls but not significantly. LDL-C is increased significantly in CVD patients as compared to controls in fasting state. AI is significantly higher in patients of CVD than controls ($p < 0.001$). In a prospective cardiovascular Munster study, elevated TG has been found to be significant and independent risk factor for major coronary events even after adjustment for LDL-C and HDL-C levels and other risk factors.[13] Similar results have been reported by some other authors.[14,15] Postprandially, TG levels in CVD patients are found to be raised significantly as compared to controls ($p < 0.05$) and fasting state ($p < 0.001$). Total cholesterol is high postprandially as compared to controls ($p < 0.001$) but decreased as compared to fasting in both controls ($p > 0.05$) and study group ($p < 0.001$). Similar findings have been reported by Ernst JS et al but they observed significant decrease in both the groups.[5] PP HDL-C is lower in study group as compared to control group ($p < 0.05$). TG rich lipoproteins in PP state act adversely on vascular endothelium through increasing superoxide anion radicals or by direct impairment of vascular endothelium by decreasing coronary bioactivity.

The multiple risk factor intervention trial with fasting and nonfasting TG levels at baseline were followed up for CHD incidence and death on total of 2809 (of 12 866) men randomized during 1973 through 1975[16]. Proportional hazards regression models were used to assess association of fasting and nonfasting TG levels with CHD. They found that the average fasting and nonfasting TG levels were 187 and 284 mg/dl, respectively. Prevalence of hypertriglyceridemia (200 mg/dl or more) was 31% for fasting and 61% for nonfasting. There were 175 nonfatal or fatal CHD events during 8 years and 328 CHD deaths during 25 years. Compared with TG levels less than 200 mg/dl, risk factor-adjusted hazard ratios for CHD mortality for hypertriglyceridemia were 1.24 (P=.09) for fasting and 1.26 (P=.07) for nonfasting. For nonfatal or fatal CHD, fasting and nonfasting TG levels were similarly predictive with hazard ratios of 1.64 (P=.004) for fasting and 1.46 (P=.03) for nonfasting. These associations for fasting TG levels were assessed to be underestimated by 56% because of regression dilution bias, with attenuation likely greater for nonfasting Tg levels. He concluded the greater ease of obtaining nonfasting then fasting measurements, greater prevalence of hypertriglyceridemia with nonfasting then fasting values, and similarly increased risk for each indicate that nonfasting TG levels may be more useful than fasting ones for risk stratification.

Koba S et al.[17] measured mean LDL particle diameter by gradient gel electrophoresis in 571 patients with CHD and in 263 healthy subjects who served as control patients. Patients with CHD were classified into acute coronary syndrome (ACS), stable CHD and vasospastic angina. High-density lipoprotein cholesterol and apolipoprotein-A1 and -B were significantly different between patients with CHD and controls. LDL size in patients with CHD was markedly smaller than that in controls in both men and women (25.5 ± 0.7 vs. 25.9 ± 0.4 and 25.7 ± 0.7 vs 26.0 ± 0.5nm, respectively). LDL cholesterol was significantly higher in patients with ACS than in other groups. Plasma levels of high-density lipoprotein cholesterol decreased as the number of diseased vessels or angiographic coronary severity evaluated by Gensini score increased but the LDL size was comparable irrespective of the type of CHD and the extent

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severity of the lesions. Multiple logistic regression analysis revealed that small dense LDL was independently associated with the incidence of CHD in male and females. Their studies suggested that the small dense LDL is strongly associated with various type of CHD, independent of traditional and nontraditional coronary risk factor, but is not related to the severity and extent of the coronary lesions.

In our study the lipid ratios of TC: HDL-C: LDL-C: HDL-C found higher in both fasting and postprandial (table 2.1 & 2.3) state in patients with cardiovascular disease than control and also showed higher in CVD patient whereas raised in both group patients than control group patients.

Conclusion

On the basis of our study results we concluded that the monitoring and management of dyslipidemia in cardiovascular disease patients and the data suggest that assessment and treatment. Impaired lipoprotein metabolism is one of the major an etiological factors for the pathogenesis of CVD. Assessment is usually made in the fasting state but during the postprandial period the massive amount of lipid fluxes through the intra vascular compartment. Thus, the higher TG and VLDL-C and lower HDL-C levels are better indicators of CVD than the classical risk factors like total cholesterol and LDL-C supporting the hypothesis that postprandial lipoprotein metabolism and their catabolic rate play a crucial role in the development and progression of atherosclerosis.

References

[1] WHO Preventing Chronic disease a vital investment (2005).
[2] Rosenson RS. Hypertriglyceridemia and coronary heart disease risk. Cardiol Rev 1999; 7: 342-8.
[3] Zilversmit DB. Atherogenesis: A postprandial phenomenon. Circulation 1979; 60: 473-85.
[4] Bae JH, Schwemmer M, Lee IK, et al. Postprandial hypertriglyceridemia induced endothelial dysfunction in healthy subjects is independent of lipid oxidation. Int J Cardiol 2003; 87: 259-67.
[5] Ernst JS, Marie CA, Judith R, et al. Comparison of fasting and postprandial plasma lipoproteins in subjects with and without coronary heart disease. Am J Cardiol 2001; 88: 1129-33.
[6] Mendis S, Puska P, Norrving B. Global Atlas on cardiovascular disease preventation and control Published by the World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization. 2011.
[7] WHO. Hypertension fact sheet. 2012.
[8] Cohn JS. Postprandial lipemia and remnant lipoproteins. Clin Lab med 2006; 26(4):773-786.
[9] Zilversmit DB. Atherogenesis a postprandial phenomenon. Circulation. 1979; 60:473-485.
[10] Hamsten A, Karpe F. Triglycerides and coronary heart disease has epidemiology given us the right answer? In Betteridge DJ. Ed. Lipids Current perspective 1996; 43-68.
[11] Jarret RJ. The cardiovascular risk associated with impaired glucose tolerance. Diabet Med 1996; 13: 15-19.
[12] Balkau B, Bertrais S, Ducimetiere P, et al. Is there a glycemic threshold for mortality risk? Diabetes Care 1999; 22: 696-99.
[13] Assmann G, Cullen P, Schulta H. The Munster Heart Study (PROCAM) - results of follow up at 8 years. Eur Heart J 1998; 19: A2-A11.
[14] Cullen P. Evidence that triglycerides are independent coronary heart disease risk factors. Am J Cardiol 2000; 86: 943-949.
[15] Alaupovic P, Mack WJ, Knightgibson C, et al. The role of triglyceride rich lipoprotein families Bombay Hospital Journal, Vol. 50, No. 3, 2008 449 in the progression of atherosclerotic lesions as determined by sequential coronary angiography from a controlled clinical trial. Arterioscler Thromb Vasc Biol 1997; 17: 715-722.
[16] Eberly LE, Stamler M, Ooi DS et. al. Postprandial Remnant-like lipoproteins in hypertriglyceridemia. J Clin Endocrinology Metab 2001 86(7): 3134-3142.
[17] Koba S, Hirano T, Kondo T et.al. significance of small dense low-density lipoproteins and other risk factor in patients with various type of coronary heart disease. Am Heart J 2002 44(6).

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