

Comparison of Fasting and Postprandial Lipid Profile in Patients of Coronary Heart Disease

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Abstract

Lipid profile and blood glucose were estimated in fasting and postprandial samples of 40 patients of coronary heart disease and 40 healthy controls along with other routine investigations and compared statistically. Serum triglycerides, total cholesterol, VLDL-cholesterol were significantly high in patients than controls in both fasting and postprandial states ($p < 0.001$) while HDL-cholesterol was found to be decreased significantly in fed state only ($p < 0.05$). LDL-cholesterol was found to be decreased in patients postprandially as compared to controls but not significantly while fasting levels were raised in patients than controls ($p < 0.001$). It is concluded that there is comparatively more transfer of cholesterol and cholesterol esters from HDL to LDL in postprandial state leading to their low levels and this along with higher triglycerides and VLDL levels are better indicators of coronary heart disease.

Introduction

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 dyslipidaemia are the prominent causes involved.¹

Hypercholesterolaemia and hypertriglyceridaemia are considered the independent risk factors but most of the earlier studies in this area have considered only the fasting 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.² Postprandial hypertriglyceridaemia (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.³⁻⁶ Therefore, the present study was undertaken to evaluate the role of postprandial lipid profile as an indicator of the efficiency of lipoprotein metabolism and its relationship with development of CHD.

Material and Methods

This study was carried out on 40 patients of CHD and 40 age and sex matched healthy volunteers after obtaining their informed consent. The diagnosis of CHD was based on previous history of myocardial infarction, ECG evidence, echocardiography, coronary artery bypass grafting surgery or coronary angiogram. These patients were free of any

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clinical event for a period of at least six months prior to the study. Venous blood sample was collected aseptically for each subject after a twelve hours overnight fast and then two hours and four hours after a mixed diet. Lipid profile and blood sugar were done in fasting samples and postprandial (PP) samples- blood sugar in 2 hour PP and lipids in 4 hours PP samples. In addition, routine investigations like haematological profile, blood urea, serum electrolytes, serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), serum uric acid levels were also carried out in fasting samples of all the subjects. Total cholesterol (TC), HDL- cholesterol (HDL-C) and TG were estimated enzymatically while VLDL and LDL were calculated using Friedewald equation.⁷⁻¹⁰ Atherogenic index (AI) was calculated using the formula, AI = TC-HDL-C/HDL-C.⁷ Blood sugar was analysed using glucose oxidase-peroxidase (GOD-POD) method,¹¹ blood urea by diacetylmonoxime method,¹² serum sodium/ potassium by flame photometry [Fp20],¹³ SGOT/PT by kinetic method^{14,15} and uric acid by enzymatic method¹⁶ using autoanalyser Konelab 60 for all parameters except for blood sugar and urea which were analysed using semiautoanalyser Erba-Chem.

Results

There were 36 males and 4 females in both

Table 2 : Blood Sugar and Lipid profile in patients of coronary heart disease and controls

Parameter (mg/dl)	Control group (mean ± SD)		Study group (mean ± SD)	
	Fasting	Postprandial	Fasting	Postprandial
Blood sugar	70.4±7.5	116.4±11.3	82.1±15.0	114.5±20.3
Triglycerides	119.2±32.9	137.9±37.4	173.4±81.6	227.4±110.8
Total cholesterol	174.6±31.5	172.0±33.3	196.6±35.7	186.1±34.1
HDL-C	41.4±5.5	39.5±5.0	40.7±5.0	36.9±4.6
LDL-C	109.5±28.5	105.1±30.4	120.4±32.8	104.6±31.6
VLDL-C	23.8±6.5	27.4±7.5	34.6±16.2	46.1±21.4

Table 1 : Routine investigations in patients of coronary heart disease and controls

Biochemical parameter	Control group (mean±SD)	Study group (mean±SD)	p value
Blood urea (mg/dl)	22.1±3.7	23.2±3.0	>0.01
Serum electrolytes (meq/l)			
Serum sodium	139.9±5.1	138.4±5.2	>0.01
Serum potassium	3.9±0.4	3.8±0.5	>0.01
SGOT (IU)	24.6±6.5	39.3±15.6	<0.001
SGPT (IU)	23.9±8.7	34.8±15.3	<0.001
Serum uric acid (mg/dl)	5.2±0.9	5.0±1.7	<0.001

the study group and the control group. Haemoglobin levels in the control group ranged between 9.5-14.0 g% (mean= 11.6 g%) while in the study group, it ranged between 9.0-14.0 g%. Total leucocyte count ranged from 5000-15000 /mm³ in both the groups with a mean value of 9725.0/mm³ in controls and 9987.5/mm³ in the study group. Levels of blood urea, serum electrolytes, SGOT, SGPT and serum uric acid are given in Table 1 while fasting and post-prandial blood sugar and lipid profile are given in Table 2.

The atherogenic index was found to be 3.8 ± 0.9 in the study group as compared to 3.2 ± 0.6 in the control group with p value of < 0.01.

Discussion

In the present study, the patients of CHD

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 CHD. Various authors also have reported an increased risk of CHD in upper percentiles of fasting glucose distribution.^{17, 18}

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 CHD patients as compared to controls in fasting state. AI is significantly higher in patients of CHD 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.¹⁹ Similar results have been reported by some other authors.²⁰⁻²³

Postprandially, TG levels in CHD 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.⁴ 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.^{5,6,24-26} In another study, it was found that

atherosclerosis was associated with PP TG levels independently of fasting TG suggesting that lipoprotein characteristics specific to PP state are atherogenic.²⁷ Roche *et al* have shown that magnitude and duration of PP lipemia is positively related to the pathogenesis and progression of CHD. An elevated lipemic response precipitates a number of adverse metabolic events by activating the coagulation factor VII and plasminogen activator inhibitor.^{28,29} Postprandial state modulates both metabolism and composition of apo B-100 containing lipoprotein particles and it is probable that the intravascular cholesterol redistribution due to postprandial lipidaemia modifies plasma lipoproteins such that there is an increased generation of potentially atherogenic TG rich lipoproteins and small dense LDL.²⁹ Delayed lipid clearance from body might reveal a state of fat intolerance linked to an elevated risk of CHD that is under genetic control and cannot be detected by simple measurement of fasting lipids.

In fed state, with the influx of TG rich lipoproteins from the intestines and subsequent lipolysis of triglycerides, there is transfer of cholesterol esters from HDL and LDL to these particles through the action of CETP (Cholesterol ester transfer protein). This results in a decrease in LDL-C and HDL-C in the fed state as compared with the fasting state as is seen in the present study. Decreased HDL-C in patients indicate decreased rate of reverse cholesterol transport and therefore accumulation of TG-rich lipoproteins leading to increased risk of atherosclerosis and CHD in patient group.

Thus, higher TG and VLDL-C and lower HDL-C levels are better indicators of CHD 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.

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Abbreviations

CHD, Coronary heart disease; PHTG, postprandial hypertriglyceridemia; PP, Postprandial; AI, Atherogenic index; CETP, Cholesterol ester transfer protein.

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