Original Research Article

Comparison of fasting and post prandial lipid profile in patients of IHD

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Abstract

Background: Ischemic heart disease (IHD) is widespread in both developed and developing countries and remains the main cause of death despite recent advances in diagnostic facilities and treatment regimens. 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. **Aims and objectives:** To compare the fasting and post prandial lipid profile in patients of IHD attending tertiary care institute. **Materials and method:** In the present study 30 patients of IHD reporting to Department of Medicine, SRTR Medical College, Ambajogai were enrolled after taking informed consent. Study duration was from January 2018 to December 2018. The diagnosis of CHD was based on previous history of myocardial infarction, ECG evidence, echocardiography, coronary artery bypass grafting surgery or coronary angiogram. All these patients were free of any clinical event for a period of at least six months prior to the study. All the enrolled patients were underwent detail clinical examination and the findings were enrolled in the prestructured proforma. Venous blood sample was collected aseptically for each subject after a twelve hours overnight fast and then two 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. **Results:** Tobacco chewing (40%) was the most common risk factor observed followed by smoking (26.67%) and hypertension (23.33%). It was observed that mean fasting sugar level of the study subjects was 85.41±21.24mg/dl while post prandial sugar level was 112.65±24.52mg/dl. Mean fasting triglycerides was 211.02±63.8mg/dl and mean post prandial triglycerides 275.65±47.9mg/dl and the difference was statistically significant. Fall in post prandial total cholesterol (229.72±53.8mg/dl) was observed as compared to fasting level (243.87±64.3mg/dl) but the difference observed was not statistically significant. Mean fasting HDL was 42.76±4.7mg/dl while mean post prandial HDL was 40.65±3.5mg/dl. It was seen that mean fasting LDL was 156.48±23.8mg/dl and mean post prandial LDL was 145.76±31.4mg/dl. It was observed that post prandial VLDL levels (41.76±28.54) were raised as compared to fasting VLDL levels (37.93±17.5) but the difference was not statistically significant. **Conclusion**: Thus we conclude that there was that there was no significant clinical difference between fasting and nonfasting levels of total cholesterol, HDL, and LDL except Triglycerides where TG was raised statistically significant post prandialy.

Key words: IHD, lipid profile, Fasting, post prandial.

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INTRODUCTION

Ischemic heart disease (IHD) is widespread in both developed and developing countries and remains the main cause of death despite recent advances in diagnostic facilities and treatment regimens. It is a multifactorial disease in which atherosclerosis and dyslipidemia are the most important causes.[1]While hypercholesterolemia and hypertriglyceridemia are considered independent risk factors, most of the first research done in this area only examined fasting lipids and lipoproteins. Recently, it has been suggested that postprandial lipoproteins may be a better indicator of the metabolism of perturbed lipoproteins and thus atherosclerosis and CNS metabolism.[2]Human

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food consumption is usually evenly distributed throughout the day (i.e, in the form of three meals and snacks between). Fasting stage occurs after 8 hours of fast[3]. Thus, most humans find themselves in the nonfasting state for the majority of a 24-hour period, perhaps with the exception of the early morning hours. Despite this fact, plasma lipids, lipoproteins, and apolipoproteins for cardiovascular risk prediction are usually measured in the fasting state.[3-5] A main reason is the increase in triglyceride levels seen during a fat tolerance test, in which patients typically consume 1 g fat per 1 kg body weight.[6,7] However, levels of nonfasting triglycerides are better at predicting future cardiovascular events than levels of fasting triglycerides.[8,9] Furthermore, it is possible that nonfasting levels of lipids, lipoproteins, and apolipoproteins differ only minimally from levels in the fasting state simply because most people consume far less fat at ordinary meals than during a fat tolerance test. 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.[10] 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.[11-14] 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 IHD.

MATERIALS AND METHOD

The present study was conducted in Department of Medicine, SRTR Medical College, Ambajogai were enrolled after taking informed consent. Study duration was from January 2018 to December 2018. with the aim to study and compare the fasting and post prandial lipid profile in patients of IHD. For the purpose of study 30 patients of IHD reporting to the study institute were enrolled in the study after taking 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. All these patients were free of any clinical event for a period of at least six months prior to the study. All the enrolled patients were underwent detail clinical examination and the findings were enrolled in the prestructured proforma. Venous blood sample was collected aseptically for each subject after a twelve hours overnight fast and then two 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, etc 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.[15-18] The collected data was entered in Microsoft excel and was anazlyed and presented with appropriate tables and graphs.

RESULTS

Table 1: Distribution of patients according to gender

Gender	No. of patients	Percentage
Male	19	63.33
Female	11	36.67
Total	30	100

It was observed that in the present study total 63.33% patients were male and 36.67% were female with male: female ratio of 1.73:1.

Table 2: Distribution of patients according to risk factors of IHD

Risk factors *	No. of patients	Percentage
Smoking	8	26.67
Diabetes mellitus	6	20.00
Hypertension	7	23.33
Tobacco chewing	12	40.00
Family history of PCAD	3	10.00

* Multiple responses recorded

While studying various risk factors of IHD in the study population it was observed that tobacco chewing (40%) was the most common risk factor observed followed by smoking (26.67%) and hypertension (23.33%).

Table 3: Distribution of patients according to Biochemical parameter

Biochemical parameter	No. of patients	Percentage
Blood sugar (F)	85.41	21.24
Blood sugar (PP)	112.65	24.52
Blood urea (mg/dl)	21.83	2.87
Serum sodium	141.74	4.38
Serum potassium	4.18	1.21

It was observed that mean fasting sugar level of the study subjects was 85.41±21.24mg/dl while post prandial sugar level was 112.65±24.52mg/dl. Mean blood urea, serum sodium and potassium was 21.83±2.87mg/dl, 141.74±4.38meq/l and 4.18±1.21 meq/l respectively.

Table 4: Distribution of patients according to fasting and postprandial lipid profile

Parameter	Fasting	Postprandial	Statistical significance
Triglycerides	211.02±63.8	275.65±47.9	Significant
Total cholesterol	243.87±64.3	229.72±53.8	Not Significant
HDL	42.76±4.7	40.65±3.5	Not Significant
LDL	156.48±23.8	145.76±31.4	Not Significant
VLDL	37.93±17.5	41.76±28.54	Not Significant

It was observed that mean fasting triglycerides was 211.02±63.8mg/dl and mean post prandial triglycerides 275.65±47.9mg/dl and the difference was statistically significant. Fall in post prandial total cholesterol (229.72±53.8mg/dl) was observed as compared to fasting level (243.87±64.3mg/dl) but the difference observed was not statistically significant. Mean fasting HDL was 42.76±4.7mg/dl while mean post prandial HDL was 40.65±3.5mg/dl. It was seen that mean fasting LDL was 156.48±23.8mg/dl and mean post prandial LDL was 145.76±31.4mg/dl. It was observed that post prandial VLDL levels (41.76±28.54) were raised as compared to fasting VLDL levels (37.93±17.5) but the difference was not statistically significant.

DISCUSSION

The present study was conducted in the department of medicine of tertiary care institute with the aim to compare the fasting and post prandial lipid profile in patients of IHD. Total 30 cases of IHD were studied and it was observed that total 63.33% patients were male and 36.67% were female with male: female ratio of 1.73:1.While studying various risk factors of IHD in the study population it was observed that tobacco chewing (40%) was the most common risk factor observed followed by smoking (26.67%) and hypertension (23.33%). It was observed that mean fasting sugar level of the study subjects was 85.41±21.24mg/dl while post prandial sugar level was 112.65±24.52mg/dl. Thus the patients of CHD had significantly higher levels of post prandial blood glucose as compared to fasting glucose level. Vijay Shankar[19] observed 82.1±15.0mg/dl fasting glucose and 114.5±20.3mg/dl post prandial glucose with statistically significant difference. Jarret RJ [20] and Balkau B[21] also observed similar findings in their study. Mean blood urea, serum sodium and potassium was 21.83±2.87mg/dl, 141.74±4.38meq/l and 4.18±1.21 meq/l respectively. It was observed that mean fasting triglycerides was 211.02±63.8mg/dl and mean post prandial triglycerides 275.65±47.9mg/dl and the difference was statistically significant. Similar findings have been reported by Ernst JS *et al* [ref1-4]. 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.[13,14,22-24]

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.[25] 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.[26,27]Fall in post prandial total cholesterol (229.72±53.8mg/dl) was observed as compared to fasting level (243.87±64.3mg/dl) but the difference observed was not statistically significant. Mean fasting HDL was 42.76±4.7mg/dl while mean post prandial HDL was 40.65±3.5mg/dl. It was seen that mean fasting LDL was 156.48±23.8mg/dl and mean post prandial LDL was 145.76±31.4mg/dl. It was observed that post prandial VLDL levels (41.76±28.54) were raised as compared to fasting VLDL levels (37.93±17.5) but the difference was not statistically significant. The postprandial VLDL levels showed a similar pattern to TG as mentioned above. Similarly Ayyappan *et al*.[28] in their study observed that VLDL had a significant postprandial rise and was considered as a component of postprandial lipemia as well. Boccalondro *et al*[29] have shown that patients with coronary artery diseases have a prolonged postprandial lipemia compared to healthy individuals. It is well documented that Hyperlipidemia is a risk factor for cardiovascular disease and fasting lipoprotein measurements, according to ATP III recommendation guidelines, is currently considered the standard of care when assessing a patient's lipid profile.[30] In a clinical setting this creates an inconvenience for patients and providers alike. However recent studies have raised doubt as to the need to measure fasting lipids and thus changing clinical practice.

CONCLUSION

Thus we conclude that there was that there was no significant clinical difference between fasting and nonfasting levels of total cholesterol, HDL, and LDL except Triglycerides where TG was raised statistically significant post prandialy.

REFERENCES

1. Rosenson RS. Hypertriglyceridemia and coronary heart disease risk. Cardiol Rev 1999; 7: 342-8.
2. Zilversmit DB. Atherogenesis: A postprandial phenomenon. Circulation 1979; 60: 473-85.
3. Rifai N, Warnick GR. Lipids, lipoproteins, apolipoproteins, and other cardiovascular risk factors. In: Burtis CA, Ashwood ER, Bruns DE, eds. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 4th ed. Philadelphia, Pa: Elsevier Saunders; 2006:903–982.

4. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486 –2497.
5. De Backer G, Ambrosioni E, Borch-Johnsen K, Brotons C, Cifkova R, Dallongeville J, Ebrahim S, Faergeman O, Graham I, Mancia G, Manger C, V, Orth-Gomer K, Perk J, Pyorala K, Rodicio JL, Sans S, Sansoy V, Sechtem U, Silber S, Thomsen T, Wood D. European guidelines on cardiovascular disease prevention in clinical practice: Third Joint Task Force of European and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. Eur Heart J. 2003;24:1601–1610.
6. Cohn JS, McNamara JR, Cohn SD, Ordovas JM, Schaefer EJ. Postprandial plasma lipoprotein changes in human subjects of different ages. J Lipid Res. 1988;29:469–479.
7. Schaefer EJ, Audelin MC, McNamara JR, Shah PK, Tayler T, Daly JA, Augustin JL, Seman LJ, Rubenstein JJ. Comparison of fasting and postprandial plasma lipoproteins in subjects with and without coronary heart disease. Am J Cardiol. 2001;88:1129 –1133.
8. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA. 2007;298:309 –316.
9. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA. 2007;298:299 –308.
10. Zilversmit DB. Atherogenesis: A postprandial phenomenon. Circulation 1979; 60: 473-85.
11. 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.
12. 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.
13. Plotnick GD, Corretti MC, Vogel RA. Effect of antioxidant vitamins on the transient impairment of endothelium dependent brachial artery vasoactivity following a single high fat meal. J Am Med Assoc 1997; 278: 1682-6.
14. Kugiyama K, Doi H, Motoyama T, *et al*. Association of remnant lipoprotein levels with impairment of endothelium dependent vasomotor function in human coronary arteries. Circulation 1998; 97: 2519-26.
15. Orekhov AN, Tertov VV, Mukhin DN. Desialyated low density lipoprotein- naturally occurring modified lipoprotein with atherogenic potency. Atherosclerosis 1991; 86: 153-61.
16. Gordel T, Castelli WP, Hjortlaud MC. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. Am J Med 1977; 62: 707-14.
17. McGowan MW, Artiss JD, Strandbergh DR. A peroxidase coupled method for the colorimetricdetermination of serum triglycerides. Clin Chem 1983; 29: 38-542.
18. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol without the use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499-504.
19. Vijay Shankar, Harnam Kaur, Kiran Dahiya, MS Gupta. Comparison of Fasting and Postprandial Lipid Profile in Patients of Coronary Heart Disease. Bombay Hospital Journal,2008; 50(3):445-49.
20. Jarret RJ. The cardiovascular risk associated with impaired glucose tolerance. Diabet Med 1996; 13:15-9.
21. Balkau B, Bertrais S, Ducimetiere P, *et al*. Is there a glycemic threshold for mortality risk? Diabetes Care 1999; 22: 696-99.
22. Shaikh M, Wootton R, Nordestgaard BG, *et al*. Quantitative studies of transfer in vivo of low density, Sf 12-60 and Sf 60-400 lipoproteins between plasma and arterial intima in humans. Arterioscler Thromb 1991; 11 : 569-77.
23. Patsch JR, Miesenbock G, Hopferwieser T, *et al*. Relation of triglyceride metabolism and coronary artery disease: studies in the postprandial state. Arterioscler Thromb Vasc Biol 1992; 12 : 1336-45.
24. Bjorkegren J, Boquist S, Samnegard A, *et al*. Accumulation of apolipoprotein C-1 rich and cholesterol rich VLDL remnants during exaggerated postprandial triglyceridemia in normolipidemic patients with coronary artery disease. Circulation 2000; 101 : 227-30.
25. Sharrett AR, Heiss G, Chambless LE, *et al*. Metabolic and lifestyle determinants of postprandial lipemia differ from those of fasting triglycerides. The Atherosclerosis risk in communities (ARIC) study. Arteioscler Thromb Vasc Biol 2001; 21 : 275-81.
26. Roche HM, Gibney MJ. The impact of postprandial lipemia in accelerating atherothrombosis. J Cardiovasc Risk 2000; 7 : 317-24.
27. Karpe F. Postprandial lipoprotein metabolism and atherosclerosis. J Intern Med 1999; 246 : 341-55.
28. Ayyappan S, Kalyananibehra A and Ilanchezhian T: Comparison of post prandial lipid profile at an interval of 2 hours and 4 hours in patients of coronary heart disease. Int J Pharm Sci Res 2017; 8(4): 1846-49
29. Boccalandro F, Farias J, Boccalandro C, Vaisman D. Frequency of postprandial lipemia after a first acute coronary event (unstable angina pectoris or non-ST-segment elevation acute myocardial infarction) and the effects of atenolol on the lipemia. Am J Cardiol. 2002; 90(2):153-6.
30. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on a. Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult b. Treatment Panel III) final report. Circulation. 2002; 106 (25): 3143-421.