

Comparative Assessment of the Fasting and Non-Fasting Lipid Profile in Healthy Adult Population: An Analytical Study

Prince Chandra Shekhar¹, Nimisha Priya²

¹Assistant Professor, Department of Biochemistry, Shree Narayan Medical Institute & Hospital, Saharsa, Bihar, India

²BDS, MDS, Department of Dentistry, Private Practice, Bihar, India

Received: 07-06-2022 / Revised: 11-07-2022 / Accepted: 05-08-2022

Corresponding author: Dr. Prince Chandra Shekhar

Conflict of interest: Nil

Abstract

Aim: The study was proposed to test the feasibility, of using non-fasting sample to estimate lipid profile, by comparing the lipid profiles obtained in fasting and non- fasting.

Methods: The present study was conducted by the Department of Biochemistry, Shree Narayan Medical Institute & Hospital, Saharsa, Bihar, India, for the period of 2 years

Results: The lipid profile parameters in both groups in fasting and postprandial statuses were compared. In fasting group, the mean fasting serum total cholesterol level was 194.6 mg/dl and in postprandial group, mean serum total cholesterol level was 196.46 mg/dl (P = 0.0490). In fasting group, the mean fasting serum triglyceride level was 124.18 mg/dl and in postprandial group, mean serum triglyceride level was 127.19 mg/dl (P = 0.0001). The mean fasting High Density Lipoprotein (HDL) level was 47.09 mg/ dl and mean postprandial HDL was 45.90 mg/dl (P = 0.0798). The mean fasting serum VLDL level was 26.25 mg/dl and mean postprandial VLDL level was 27.34 mg/dl (P=0.0001). The mean fasting LDL was 124.6 mg/dl and mean postprandial LDL was 126.10 mg/dl (P = 0.0350).

Conclusion: We found that there is no significant clinical difference between fasting and non-fasting levels of total cholesterol, HDL, LDL, VLDL and TG. Thus, for estimation of lipid profile we can use the blood samples at any time or irrespective of mealtime.

Keywords: Fasting, Non-Fasting, Lipid Profile, Cardiovascular Disease, total cholesterol

This is an Open Access article that uses a fund-ing model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Cardiovascular disease (CVD) is a major health problem across the world for 30% of all deaths. [1,2] In India, 52% of deaths occur among those younger than 70 years, resulting in a considerable burden from cardiovascular diseases on working age citizens. [3] Atherosclerosis leading to cardiovascular disease remains the major cause of death and premature disability globally. Major risk factors for atherosclerosis are: advancing age, male

sex, dyslipidaemia, diabetes mellitus, hypertension, cigarette smoking, hypothyroidism, family history of CAD. Non-traditional risk factors are inflammatory markers, highly sensitive C-reactive protein (CRP), lipoprotein-associated phospholipase A2 (Lp-PLA2), lipoprotein(a), hyperhomocysteinemia, hyperuricemia. [4]

Serum lipid profile has now become almost a routine test. It is usually done in

fasting state due to certain alterations in postprandial triglyceride and subsequent calculated LDL values in non-fasting serum sample but in few recent studies it has been seen that there is no much difference between the values of fasting lipid profile and non-fasting lipid profile. [5]

Lipid profiles are batteries of tests which include the measurement of lipids and lipoproteins such as cholesterol, triglycerides (TG) and LDL-cholesterol, HDL-cholesterol, VLDL-C respectively, of an individual. Lipids and lipoproteins are intimately involved in the development of atherosclerosis, which is the underlying cause of cardiovascular disease like myocardial infarction. It can also cause cerebrovascular disease and peripheral vascular disease. [6-8]

Dyslipidaemia is considered one of major predisposing risk factor for atherosclerosis, American Academy of Clinical Endocrinologists (AACE) recommends evaluation of all adults >20 years of age for dyslipidaemia every 5 years as part of a global risk assessment. [4] Dyslipidemia can also be seen in many genetic disorders like familial hypercholesterolemia. This is caused due to defect in the expression and/or function of LDL receptor. It leads to accumulation of LDL in plasma, leading to deposition of cholesterol in skin, tendons, and arteries, where it causes accelerated atherosclerosis, resulting in premature CHD and death. [6,8]

Presently cardiovascular diseases are the leading cause of morbidity and mortality in developing countries like India. Nevertheless, in routine clinical practice the lipid profile is generally measured after 12-14 hours of fasting. Though it has been the most reliable method for testing lipid profile, it has some drawbacks. Fasting is not easy for some people especially children and diabetics. Lipid profile in fasting state acts as a barrier for population screening. Also for patients, physicians and testing laboratories, it would be more

convenient and efficient to measure lipid levels in a non-fasting setting. [9-13] So, unless there's a really good reason for fasting samples to be tested, non-fasting sampling is much more convenient for all, from a practical perspective. So, intent of our study is to check the authenticity of results obtained using non-fasting samples by correlating it with the results obtained using fasting samples.

The study was proposed to test the feasibility, of using non-fasting sample to estimate lipid profile, by comparing the lipid profiles obtained in fasting and non-fasting.

Materials and Methods

The present study was conducted by the Department of Biochemistry, Shree Narayan Medical Institute & Hospital, Saharsa, Bihar, India, for the period of 2 years. The study includes 300 patients.

Ethical clearance was obtained from the institutional ethical committee before the start of the study. The informed consent was obtained from the subjects who were willing to participate in the study.

Methodology

The subjects were selected based on the clinical history and clinical examination. The subjects for the study were randomly selected from the population, which included medical student, employees of all cadres at the hospital.

Analysis was done in clinical biochemistry laboratory. Healthy individuals aged between 20-57 yrs with BMI (Body mass index) between 18-24.99kg /m² were included in the study. Subjects with cardiovascular disease, diabetes mellitus, hypertension, malnutrition, renal diseases, cerebrovascular diseases, sepsis, medications, alcohol abuse, smoking, endocrine disorders, Pregnancy, various storage disorders, congenital biliary atresia, dyslipidemia, obesity, strenuous exercise, HRT were excluded from the study. [14,15]

Anthropometric measurements: Height was measured in cms without shoes by standard procedure and weight was measured in kg and used for calculation of body mass index.

Method of sample collection: 2ml of Blood Sample was collected, one sample after 8-12 hrs fasting and second sample in non-fasting state (random sample). lipid profile parameters were assayed in duplicate to minimize analytical variation¹⁶ in Roche Hitachi (902) autoanalyzer. Quality control: The analyzer was calibrated with materials provided by the Bio-rad. Changes in calibration curve and specificity of the analytical method were detected by using a number of accuracy control specimens, at both normal (level-I) and pathological (levels-II) of concentration, for the lipid profile analytes. During the study there were no change in the equipment, reagents, calibration standards, and controls.

Lipid profile was estimated by enzymatic kit method.

1. Estimation of Total Cholesterol in serum (enzymatic method: cholesterol oxidase/ peroxidase): the assay was carried out by using A25 bio system auto analyzer. Reference range: Upto 200mg/dl – desirable, 200–239 mg/dl–borderline high and > 240 mg/dl – High.

2. Estimation of serum Triglycerides (Enzymatic method glycerol phosphate/ peroxidase): the assay was carried out using A25 bio system auto analyzer. Reference range: Upto 150 mg/dl – Normal, 150–199 mg/dl–Borderline high,

240-249 mg/dl – High and > 500 mg/dl – Very high.

3. Estimation of serum High Density Lipoprotein-Cholesterol (direct detergent method): the assay was carried out by using A25 bio system auto analyzer. Reference range: Upto 35 mg/dl – High risk, > 60 mg/dl–Low risk.

4. Estimation of serum Very Low-Density Lipoprotein: VLDL Cholesterol is calculated by Friedewald equation (Triglycerides/5).Reference range: 5–40 mg/dl and > 40 mg/dl high

5. Estimation of serum Low Density Lipoprotein-Cholesterol. Serum LDL-Cholesterol is calculated by Friedewald equation $LDL\text{-cholesterol} = \text{Total cholesterol} - [HDL\text{-C} + (\text{triglycerides}/5)]$. Serum LDL cholesterol was estimated by direct method when TG values were >400 mg/dl.

Statistical analysis

SPSS- version 17 was used to analyze the data. The parameters which followed a Gaussian probability Kolmogorov-Simrnov test for normality were analysed by parametric test. The parameters which did not follow a Gaussian probability curve were analysed by non-parametric test. The comparison study of fasting and non-fasting lipid profile was analyzed by student paired ‘t’ test. For total cholesterol and LDL-C parametric students paired ‘t’ test was done. Triglycerides, HDL-C, VLDL-C were fit for non-parametric test, Wilcoxon signed rank test. Correlation was done by Pearson’s correlation.

Results

Table 1: Comparison between laboratory findings of the lipid parameters in fasting and postprandial status in study population

Parameters	Fasting [mean (± SD)]	Postprandial [mean (± SD)]	P value
TC	194.6 (±10.46)	196.46 (±11.22)	0.0490
TG	124.18 (±4.56)	127.19 (±4.36)	0.0001
HLDL	47.09 (±5.48)	45.90 (±5.70)	0.0798
VLDL	26.25 (±0.90)	27.34 (±0.84)	0.0001
LDL	124.6 (±10.88)	126.10 (±10.8)	0.0350

The lipid profile parameters in both groups in fasting and postprandial statuses were compared. In fasting group, the mean fasting serum total cholesterol level was 194.6 mg/dl and in postprandial group, mean serum total cholesterol level was 196.46 mg/dl ($P = 0.0490$). In fasting group, the mean fasting serum triglyceride level was 124.18 mg/dl and in postprandial group, mean serum triglyceride level was

127.19 mg/dl ($P = 0.0001$). The mean fasting High Density Lipoprotein (HDL) level was 47.09 mg/dl and mean postprandial HDL was 45.90 mg/dl ($P = 0.0798$). The mean fasting serum VLDL level was 26.25 mg/dl and mean postprandial VLDL level was 27.34 mg/dl ($P=0.0001$). The mean fasting LDL was 124.6 mg/dl and mean postprandial LDL was 126.10 mg/dl ($P = 0.0350$).

Table 2: Percentage difference between fasting and non-fasting in lipid profile in study population

Parameters	Fasting [mean (\pm SD)]	Postprandial [mean (\pm SD)]	Percentage difference
TC	194.6 (\pm 10.46)	196.46 (\pm 11.22)	0.2
TG	124.18 (\pm 4.56)	127.19 (\pm 4.36)	21.8
HDL	47.09 (\pm 5.48)	45.90 (\pm 5.70)	7.12
VLDL	26.25 (\pm 0.90)	27.34 (\pm 0.84)	20.5
LDL	124.6 (\pm 10.88)	126.10 (\pm 10.8)	1.80

A strong positive correlation was observed between fasting total cholesterol and non-fasting total cholesterol, fasting triglyceride and non-fasting triglyceride, and fasting LDL-C and non-fasting LDL-C. A positive correlation was observed between fasting HDL-C and non-fasting HDL-C and fasting VLDL and non-fasting VLDL-C. Shows there is 0.2%, 21.8%, 20.5% increase in non-fasting total cholesterol, triglyceride and VLDL-C as compared to fasting, respectively. There is 7.12% and 1.80 % decrease in non-fasting HDL-C and LDL-C as compared to fasting, respectively.

Discussion

Lipid profile test is routinely done in fasting blood specimen. It includes four basic parameters: total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C). [16] TC and LDL-C levels were slightly reduced in non-fasting as compared to fasting specimen. The possible cause for small reduction in TC & LDL-C levels in non-fasting specimen is most likely haemodilution following fluid intake in

association to the meal. [17] Triglycerides increased and HDL-C decreased in non-fasting as compared to fasting specimen. These changes are mostly possible due to food intake rather than fluid intake. Although triglyceride increase are owed directly to fat intake, the parallel reduction in HDL cholesterol is possibly due to bidirectional lipid exchange between triglyceride-rich lipoproteins and HDL particles. [18]

In cardiology, diabetology, thyroid clinics, etc. estimation of lipid profile is now a days common and frequently done test. Serum lipid profile has now become almost a routine test. It is usually done in fasting state due to certain alterations in postprandial triglyceride and subsequent calculated LDL values in non-fasting serum sample but in few recent studies it has been seen that there is no much difference between the values of fasting lipid profile and non-fasting lipid profile. [5]

In our study, on comparison of lipid profile parameters in both fasting and postprandial statuses, the mean level of Total cholesterol (194.6vs.196.46 mg/dl),

TG (124.18 vs. 127.19 mg/dl), HDL (47.09 vs. 45.90mg/dl), VLDL (26.25 vs. 27.34 mg/dl) and LDL (124.6 vs. 126.10 mg/dl) was not significantly different. Thus, there was no significant clinical difference between fasting and non-fasting levels of total cholesterol, TG, HDL, VLDL and LDL. This change in levels of lipids, at most in response to normal food intake is minimal and unimportant. Evidence suggests differences in cholesterol levels based on fasting versus not fasting are not clinically significant. In 2013, the American College of Cardiology and the American Heart Association released guidelines noting that non-fasting lipid tests can be used for assessing cardiovascular risk, but still recommended a fasting lipid panel prior to statin initiation. [19] Anne Langsted et al. and Samia Mora et al. demonstrated that fasting lipid levels are not superior to non-fasting levels for cardiovascular risk prediction. [9,10]

In present study, when we compared fasting lipid profile, we observed significant difference in the fasting TG, HDL-C, VLDL-C and non-fasting TG, HDL-C, VLDL-C, but there was no significant difference in the total cholesterol and LDL-C in total study population. There was significant increase in the non-fasting triglycerides compared to fasting triglycerides. Difference in the fasting and non-fasting triglyceride was also shown in studies done by Anne Langsted et al, [9] Jouko Sundvall et al, [20] who observed increase in Triglycerides in response to normal food intake even after the correction albumin levels.

This increase in triglycerides is attributable directly to fat intake. Anette Varbo and colleague [21] concluded that step wise increasing non-fasting TG was related with high risk of ischemic stroke in both males and females. Sandeep Bansal et al [22] showed that, to evaluate the cardiovascular risk triglyceride in non-fasting state may

be superior to triglyceride in fasting state. Measuring non-fasting TG levels may provide additional information for determining cardiovascular risk. [23]

The advantages of a determining lipid profiles in a non-fasting blood sample are that patients who have not fasted do not have to make another appointment to have their blood drawn, By not requiring an overnight fast, the crowd of patients showing up in the morning for a blood test is lessened and physicians are spared from having to track down repeat tests.

Conclusion

Fasting samples are preferable for serum lipid profile measurement in all individuals with serum triglyceride levels greater than 350 mg/dL (in whom the Friedwald equation for calculating LDL should not be used). But, non-fasting samples for lipid profile can be used for cardiovascular risk determination in the general people as it reduces patient's inconvenience and promotes patient acquiescence towards lipid profile checking. Thus, we can use the nonfasting blood samples to estimate lipid profile in follow-up the dyslipidemic patients. There is no need to make fasting lipid profile mandatory. This study suggests efforts should be made to simplify blood sampling by replacing fasting lipid profile with non-fasting lipid profile. It is necessary to determine cut-points of non-fasting lipid values. Life-threatening or extremely abnormal test results deserve special attention and reactions of the clinical biochemical laboratory.

References

1. Gaziano TA. Economic burden and the cost-effectiveness of treatment of cardiovascular disease in Africa. *Heart*. 2008;94(2):140-144.
2. World Health Organization 2009. Cardiovascular Diseases. <http://www.who.int/mediacentre/factsheets/fs17/en/index.html><http://www.who.int>

- /cardiovascular-diseases/priorities/en/index.html. Accessed June 17, 2010.
- Goyal A, Yusuf S. The burden of cardiovascular disease in the Indian subcontinent. *Indian J Med Res.* 2006 Sep 1;124(3):235-44.
 - Jellinger PS, Smith DA, Mehta AE, Ganda O, Handelsman Y, Rodbard HW, Shepherd MD, Seibel JA, Kreisberg R, Goldberg R. American Association of Clinical Endocrinologists' guidelines for management of dyslipidemia and prevention of atherosclerosis. *Endocrine practice.* 2012 Mar 1;18:1-78.
 - Abdel-aziza WF, Soltana GM, Ahmed AM. Comparison between fasting and non-fasting lipid profile in patients receiving treatment with statin therapy. *Menoufia Med J.* 2016;30(2): 614-8.
 - Ashwood ER, Aldrich JE, Tietz NW. *Tietz fundamentals of clinical chemistry.* Saunders; 1996.
 - M, Cox DL, Nelson. *Principles of biochemistry.* England: Sara Tenney: 5th edn.; 1990; 836–843.
 - Fauci, Braunwald, Kasper, Hauser, Longo, et al. *Principles of Internal Medicine.* In: *Journal of Cardiovascular Translational Research.* vol.2. New York: Mc Graw Hill ; 2008., p. 341–348.
 - Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and non-fasting lipid levels: influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. *Circulation* 2008;118(20):2047-56.
 - Mora S, Rifai N, Buring JE, et al. Fasting compared with non-fasting lipids and apolipoproteins for predicting incident cardiovascular events. *Circulation* 2008;118(10): 993-1001.
 - Watts GF, Cohn JS. Whither the lipid profile: feast, famine, or no free lunch? *Clin Chem* 2011;57(3):363-5.
 - Gaziano JM. Should we fast before we measure our lipids? *Arch Intern Med* 2012;172(22):1705-6.
 - Khera AV, Mora S. Fasting for lipid testing: is it worth the trouble? *Arch Intern Med* 2012;172(22):1710-2.
 - Tandon N. Intermediary metabolism of lipids and lipid transport. Monograph: Lipid disorders: Implication and management (ed) Tripathy BB, Das S., Indian college of physicians, mumbai. 2002:15-32.
 - A Kakade S et al. Evaluation of reference intervals of serum lipid profile from healthy population in Western Maharashtra. *Indian J Clin Biochem.* 2009;24(1):30–35.
 - Folsom AR, Kuba K, Leupker RV, Jacobs DR, Frantz ID. Lipid concentrations in serum and EDTA-treated plasma from fasting and non-fasting normal persons, with particular regard to high-density lipoprotein cholesterol. *Clin Chem.* 1983; 29(3): 505–508.
 - Simundic AM, Cornes M, Grankvist K, Lippi G, Nybo M. Standardization of collection requirements for fasting samples: for the Working Group on Preanalytical Phase (WG-PA) of the European Federation of Clinical Chemistry and Laboratory Medicine (EFLM). *Clinica chimica acta.* 2014 May 15;432:33-7.
 - Zilversmit DB. Atherogenic nature of triglycerides, postprandial lipidemia, and triglyceride-rich remnant lipoproteins. *Clin Chem* 1995;41 (1): 153-8.
 - Farukhi Z, Mora S. Re-assessing the role of non-fasting lipids: a change in perspective. *Ann Transl Med.* 2016;4 (21):431.
 - Sundvall J, Leiviskä J, Laatikainen T, Peltonen M, Salomaa V, Vanhala M, Korpi-Hyövälti E, Lauronen J, Alftan G. The use of fasting vs. non-fasting triglyceride concentration for estimating the prevalence of high LDL-cholesterol and metabolic

- syndrome in population surveys. *BMC medical research methodology*. 2011 Dec;11(1):1-6.
21. Varbo A, Nordestgaard BG, Tybjaerg-Hansen A, Schnohr P, Jensen GB, Benn M. Nonfasting triglycerides, cholesterol, and ischemic stroke in the general population. *Annals of neurology*. 2011 Apr;69(4):628-34.
 22. 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 Jul 18;298(3):309-16.
 23. Olaleye A. A., Ejikeme B. N., Samuel E. E. E., Okeke N. E., Olinya B. I., Onyekelu E. O., Edene, C. N., Obasi J. C., & Nwafor A. V. Custodian Rape of a Minor in the Warring Zone of Ezza, Effium Area of Ebonyi State: *Journal of Medical Research and Health Sciences*, 2021; 4(9): 1468–1475.