

Serum Lipid Profile: Fasting or Non-fasting?

P. K. Nigam

Received: 29 August 2010 / Accepted: 1 December 2010 / Published online: 29 December 2010
© Association of Clinical Biochemists of India 2010

Abstract Serum lipid profile has now become almost a routine test. It is usually done in fasting state due to certain limitations in non-fasting serum sample. In the recent past efforts have been made to simplify blood sampling by replacing fasting lipid profile with non-fasting lipid profile. However, fasting specimen is preferred if cardiovascular risk assessment is based on total cholesterol, LDL cholesterol or non-HDL cholesterol. A lot has yet to be done in this area. Till then we have to believe in fasting lipid profile for assessment and management of cardiovascular disease.

Keywords Lipids · Fasting · Non-fasting

Dear Editor,

Serum lipid profile is measured for cardiovascular risk prediction and has now become almost a routine test. The test includes four basic parameters: total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides. It is usually done in fasting blood specimen. Fasting refers to 12–14 h overnight complete dietary restriction with the exception of water and medication. This may hold true due to two main reasons: (1) post prandial triglycerides remain elevated for several hours [1], (2) most reference values for serum lipids are established on fasting blood specimen. NCEP [2] and European guidelines [3] also recommend doing lipid profile in fasting blood specimen for assessment of cardiovascular risk. However, these guidelines allow total and HDL cholesterol in the non-fasting specimen as these lipids are not much different in fasting and non-fasting

specimens. In addition, non-HDL cholesterol (total cholesterol – HDL cholesterol), a secondary target of therapy in adult treatment panel III, may also be used in the non-fasting state [2].

Basically fasting state is essential for triglycerides estimation because as mentioned above it remains high for several hours after meal and the Friedewald equation, used for calculation of LDL cholesterol (LDL cholesterol = total cholesterol – HDL cholesterol – [triglycerides/5]), uses fasting triglycerides value. If non-fasting triglycerides value is used in this equation the LDL cholesterol, the primary target of lipid lowering therapy, will be underestimated. However, this problem can be overcome to some extent by using direct LDL cholesterol estimation as this can be done in non-fasting specimen. Unfortunately, this method for direct measurement sometimes also gives underestimation of LDL cholesterol [4, 5]. This may misclassify many individuals into a lower NCEP category and thereby these individuals may miss drug intervention for prevention of cardiovascular events. Moreover, the lack of association of non-fasting direct LDL cholesterol with cardiovascular disease in women raises questions regarding the clinical utility of a direct assay for LDL cholesterol in non-fasting sample [5]. Further, direct assays are costly and so add to health care cost.

During the last few years efforts have been made to simplify blood sampling by replacing fasting lipid profile with non-fasting lipid profile as it has been found that lipids, lipoproteins and apolipoproteins were not much different in fasting and non-fasting state with the exception of triglycerides which were higher in non-fasting state and all these were associated with cardiovascular risk prediction [6]. However, a fasting sample is preferred if cardiovascular disease (CVD) risk assessment is based on total cholesterol, LDL cholesterol or non-HDL cholesterol but

P. K. Nigam (✉)
Department of Cardiology, CSM Medical University, U.P.,
Lucknow 226003, India
e-mail: p_nigam1@yahoo.com

HDL cholesterol, triglycerides, total/HDL cholesterol ratio and apolipoprotein A-1 predict CVD when measured non-fasting [7]. The most interesting part is that non-fasting triglycerides levels may be even better predictor of cardiovascular risk as compared to fasting triglycerides [8, 9]. Although the terms non-fasting and postprandial can be considered synonyms but there is some difference as non-fasting sample means blood draw at any time without knowledge of the time of previous meal while post prandial implies a sample at a fixed time after a standard meal. Moreover, triglycerides increase step wise after fat diet, therefore, non-fasting triglycerides would vary depending on time after meal with highest levels 4–5 h post prandially [9]. Further, the cut off levels of non-fasting triglycerides for cardiovascular risk have not yet been defined. It is important to compare serum lipid profile in fasting and at different time interval after a representative meal in terms of prediction of cardiovascular risk. As is true for fasting triglycerides, postprandial lipemia can be affected by ethnicity, alcohol consumption, and menopausal status, and thus these factors should be considered in clinical practice [10]. Thus, a lot has yet to be done in this area and then if the use of non-fasting lipid profile could be included in recommended guidelines then the sampling for lipid profile would be simplified and this will improve the compliance for lipid lowering treatment. Till then we have to believe in fasting lipid profile for assessment and management of cardiovascular risk.

In addition to fasting/non-fasting state there are other factors (pre-analytical) which may affect lipid components:

1. A change from an upright to a supine position due to dilutional effect can reduce the cholesterol levels by 10% and triglycerides by 12% [11].
2. Prolonged tourniquet application (2–5 min) can increase cholesterol from 5 to 15% [12, 13].
3. Cholesterol is slightly higher in winter than in summer and the opposite is true for triglycerides [11, 13, 14].
4. The disease conditions like nephrotic syndrome increase total cholesterol, LDL cholesterol and VLDL cholesterol [15] and hypothyroidism increases LDL cholesterol and total cholesterol. Infection and inflammation may decrease total cholesterol and HDL cholesterol and increase triglycerides [16]. Lipids alter following myocardial infarction [17, 18] and these changes may persist for several weeks. That is why it is better to do lipid profile in such patients within 24 h of myocardial infarction. The study by Nawaz et al. [19] showed that all individual values of the lipid profile in patients admitted with acute illness vary significantly during and after hospital stay, whereas the ratio of total cholesterol to HDL remains relatively stable.

It is therefore, important that all these factors should be kept in mind while interpreting the lipid profile.

References

1. Campose H, Khoo C, Sacks FM. Diurnal and acute pattern of postprandial apolipoprotein B-48 in VLDL, IDL and LDL from normolipidemic human. *Atherosclerosis*. 2005;181:345–51.
2. 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) final report. *Circulation*. 2002;106:3143–3421.
3. De Backer G, Ambrosioni E, Borch-Johnson K, Brotons C, et al. European guidelines on cardiovascular disease and prevention in clinical practice. *Atherosclerosis*. 2003;171:145–55.
4. Sahu S, Chawla R, Uppal B. Comparison of two methods of estimation of low density lipoprotein cholesterol, the direct versus Friedewald estimation. *Indian J Clin Biochem*. 2005;20: 54–61.
5. Mora S, Rifai N, Buring JE, Ridker PM. Comparison of LDL cholesterol concentration by Friedewald calculation and direct measurement in relation to cardiovascular events in 27,331 women. *Clin Chem*. 2009;55:888–94.
6. Nordestgaard BG, Langsted A, Freiberg JJ. Nonfasting hyperlipidemia and cardiovascular disease. *Curr Drug Targets*. 2009;10:54–61.
7. Mora S, Rifai N, Buring JE, Ridker PM. Fasting compared with nonfasting lipids and Apolipoproteins for predicting incident cardiovascular events. *Circulation*. 2008;118:993–1001.
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;298:309–16.
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. Ridker PM. Fasting versus nonfasting triglycerides and prediction of cardiovascular risk: do we need to revisit the oral triglyceride tolerance test? *Clin Chem*. 2008;54:11–3.
11. Narayana S. Pre and post analytical errors in lipid determination. *Indian J Clin Biochem*. 1996;11:12–6.
12. Young DS. Biological variability. In: Brown SS, Mitchell FL, Young DS, editors. *Chemical Diagnosis of Disease*. New York: Elsevier; 1979. p. 1–113.
13. Cooper GR, Myers GL, Smith J, Schlant RC. Blood lipid measurements: variations and practical utility. *JAMA*. 1992;267: 1652–60.
14. Narayanan S. Physiological variables in blood sampling. *Mitt Klin Chem*. 1993;24:130–4.
15. Joven J, Villabona C, Vilella E. Abnormalities of lipoprotein metabolism in patients with nephrotic syndrome. *N Engl J Med*. 1990;323:579–84.
16. Alvarez C, Ramos A. Lipids, lipoproteins and apolipoproteins in serum during infection. *Clin Chem*. 1986;32:142–5.
17. Ryder REJ, Hayes TM, Mulligan JP, Kingswood JC, Williams S, Owens DR. How soon after myocardial infarction should plasma lipid values be assessed? *Br Med J*. 1984;289:1651–3.
18. Nigam PK, Narain VS, Hasan M. Serum lipid profile in patients with acute myocardial infarction. *Indian J Clin Biochem*. 2004; 19:67–70.
19. Nawaz H, Comerford BP, Njike VY, Dhond AJ, Plavec M, Katz DL. Repeated serum lipid measurements during the perihospitalization period. *Am J Cardiol*. 2006;98:1379–82.