

University of diyala

Collage of medicine



The difference in lipid profile measurement in the fasting and fed state

Prepared by

هيثم حسين عطيه جواد

Supervised by

بشرى محمود حسين

Introduction

Coronary heart disease (CHD) is an important cause of illness and death in many countries universal and is valued that it will be the single largest reason of disease problem. A number of issues are supposed to increase the possibility of developing CHD. It can be separated into two, which are controllable and uncontrollable risk factors and hypercholesterolemia is one of risk factors that can be controlled.

New approvals have preferred non-fasting lipid assessment. Applied advantages to using non-fasting measurements include increasing patient convenience dodging separate return visits for laboratory draws and educating hospital and clinic efficiency. Furthermore, non-fasting triglycerides may progress cardiovascular risk prediction. On the other hand, classification of dyslipidemias was historically resultant in fasting samples, and cohort studies and clinical trials have usually performed fasting calculations

Because of this controversy, the aim of this study was to assess fasting versus non-fasting in measuring lipid profile

Patients and methods

A complete of eighty healthy volunteer females, ranging in age between 22-30 years, have been divided into 4 groups of 20 woman each . The first three groups are fasting groups that had no longer taken any weight-reduction plan for last 4, 6, 14 hours respectively whilst in the non-fating group, blood samples have been gathered after two hours of meal. The health status of our volunteer woman used to be proven by using clinical examination.

For biochemical & hematological investigations, venous blood samples have been taken from these females from January 2018 to June 2018. The biochemical investigation consists of

- ✚ Total Cholesterol(TC)
- ✚ Total Triglycerides(TG)
- ✚ High Density Lipoprotein (HDL)
- ✚ Low Density Lipoprotein (LDL)
- ✚ Very Low Density Lipoprotein (VLDL)

They had been measured the use of Synchron CX4 scientific device Beckman Coulter Inc., Brea, CA(standard scientific laboratory methods).The hematological investigation consists of

- ✚ White Blood Cell (WBC)
- ✚ Red Blood Cell (RBC)
- ✚ Hemoglobin (HB)
- ✚ Platelet Count (PLT)

the total blood was once decided by way of the usage of computerized hematological analyzers BC-3000 plus (from Hamburg, Germany).

Statistical analysis was carried out using SPSS (Statistical Package for Social Sciences) number 22 with regard to numerical features. It was described using the mean and standard deviation of the mean and was compared between the averages for the calculation of the samples under study at the level of 0.05.

Laboratory examination

Blood models were collected over venipuncture and examined by clinical laboratory. Lipid profiles were collected by an automatic biochemistry analyzer by an enzymatic assay. Comprehensively, TC was collected by the cholesterol oxidase-p-aminophenazone method. TG was collected by the glycerine phosphate oxidase peroxidase method, LDL-C was examined by the selective solubilization method, HDL-C was determined by a similar method, Lp(a) was analyzed by an immunoturbidimetry technique according to the manufacturer's guide with a normal value of <30 mg/dL. HbA1c was measured using a Tosoh Automated Glycohemoglobin Analyzer

Statistical analysis

Constant variables are revealed as mean \pm standard deviation (SD) or median [interquartile range (IQR)]. Categorical variables are stated as number (percentage). Differences between groups were investigated using Student's t-tests and χ^2 tests. Spearman rank correlation coefficients (r) were used to measure associations between fasting and non-fasting lipid profiles. Fasting and non-fasting lipid profiles were equated using a nonparametric test of two paired samples. The diff(%)s in persons with or without statins were associated by the Mann-Whitney μ test. The Bland-Altman analysis was showed to assess agreement between fasting and non-fasting lipid tests. Statistical analyses were performed using SPSS software. All tests were two-sided and defined statistical significance by $P < 0.05$.

Results

Table 1 & Fig. 1 shows that there had been significant($p < 0.05$) outcomes for the mean level of TC , TG & VLDL when fasted for 6 & 14 hours while there had been non-significant effects between groups for the mean degree of HDL & LDL.

The mean level of TC was once 159.25 mg/dl, 142.25 mg/dl, 150.25 mg/dl, 172.50 mg/dl for fasting intervals of four hours, 6 hours and 14 hours and the non-fasting respectively and mean degree of TG was once 72.75mg/ dl, 59.0 mg/dl, 62.50mg/dl, 97.00 mg/dl for fasting hours of 4hours, 6 hours ,14 hours and the non- fasting respectively . Regarding VLDL, its mean stage used to be 16mg/dl,14.25mg/dl,12.25mg/dl and 20.75 mg/dl for fasting durations of 4 hours, 6 hours and 14 hours and non-fasting respectively. From the above measurements, we concluded that fasting at least 6 hours gave giant variations ($P < 0.05$) in assessing TC,TG & VLDL.

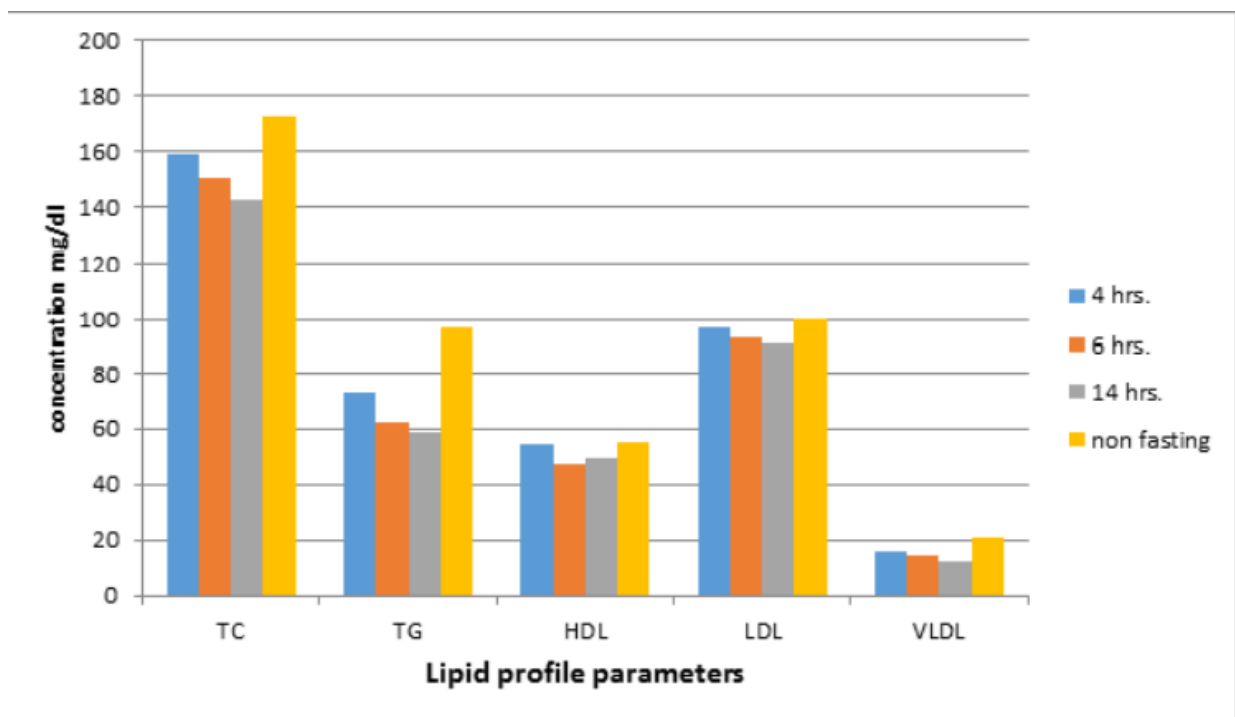


Fig 1 Lipid profile parameters under different fasting hours and non-fasting in healthy female

There had been non-significant variations between fasting & non-fasting in measuring HB , RBC, WBC and PCT as established in desk two & Fig.2

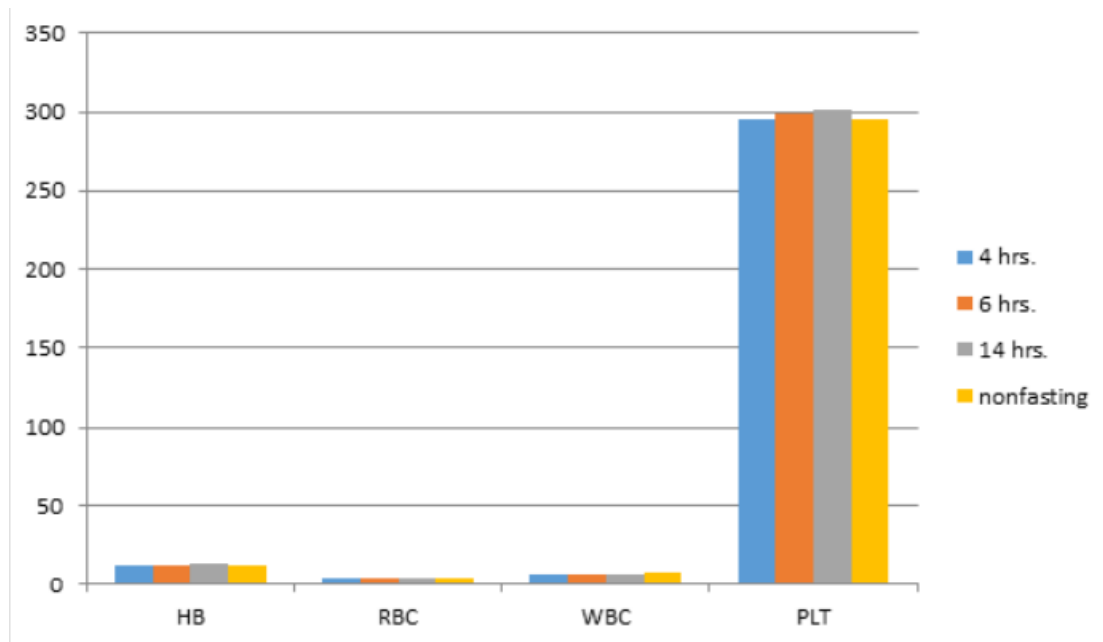


Figure 2 : Blood parameters under different fasting hours and non-fasting in healthy female

Discussion

Venipuncture is occupied in testing the lipid profile so we can predict cardiovascular risk and/or screen responses to lipid-lowering therapy. Some rules continue to promulgate the predictable practice of measuring the lipid profile in the fasting state. While other cultures & laboratories modified non-fasting lipid profiles. Since 2009, non-fasting lipid testing has become the clinical standard in Denmark, based on approvals from the Danish Society for Clinical Biochemistry that all laboratories in Denmark use random non-fasting lipid profiles as the standard, while offering clinicians the option of remeasuring triglyceride concentrations in the fasting state if non-fasting values are 4 mmol/L (350 mg/dL). Still, the UK NICE rules have allowed non-fasting lipid testing in the prime prevention setting since 2014

For cardiovascular risk valuation, sign is lacking that fasting is greater to non-fasting when assessing the lipid profile. However, there are advantages to using non-fasting samples over fasting in the measuring of lipid profile

Comparison biochemical and hematological outcomes, fasting is not desired for measuring complete blood picture as there were no substantial differences between fasting & non-fasting in measuring Hb, RBC, WBC, PLC. For lipid profile calculation, fasting at least 6 hours was suggested for measuring TC, TG & VLDL while it was not essential for assessing HDL & LDL. It was decided by Cohn et al. & Mihos et al. the reason that favored fasting lipid profiles is the growth in triglyceride concentration seen through a fat tolerance test. Alternatively, LDL cholesterol is often considered by the Friedewald formula, which has been supposed to be affected considerably by food intake. So if this formula is active, there may be some exaggeration of LDL cholesterol when chylomicrons are existing. Furthermore to that non-fasting disorder may a bit lower plasma LDL cholesterol concentrations due to liberal intake of fluids, and therefore principal to minor misclassification of cardiovascular risk, as well as to error in initiating or altering lipid-lowering medication especially to diabetic person

Since non-fasting may decline the correctness in diagnosing some forms of hyperlipidaemia, the study recommended that laboratories & organizations should also offer measurement of fasting triglycerides according to clinical situations, as in the case of very high non-fasting triglyceride concentration.

Conclusion

Diagnostic precision of non-fasting lipid profile was found significantly higher than fasting lipid profile ($p=0.004$) for the calculation of lipoprotein coronary risk on the basis of non-HDL-C, which appeared to be important test for ruling out hyperlipidemia.

Financial Disclosure

There is no financial disclosure.

Conflict of Interest

None to declare.

References

1. World Health Organization (WHO). Cardiovascular Diseases. Fact sheet N°317. Updated January 2015.
2. Bansal S, Buring JE, Rifai N, Mora S Sacks FM, Ridker PM. Fasting compared with non- fasting triglycerides and risk of cardiovascular events in women...JAMA 2007;298:309-16.
3. Sidhu D, Naugler C. Fasting time and lipid levels in a community-based population: a cross-sectional study. Arch Intern Med. 2012;172:1707– 1710.
4. Brenner H, Heiss G. The intraindividual variability of fasting triglyceride: a challenge for further standardization. Eur Heart J. 1990;11:1054–1058.
5. Knapp RG, Miller MC. Clinical epidemiology and biostatistics. National Medical Series(NMS) from Williams and Wilkins.Baltimore1992;3:34.
6. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH,Goldberg AC, Gordon D, Levy D, Lloyd-Jones DM, McBride P, Schwartz JS,Shero ST, Smith SC Jr, Watson K, Wilson PW. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;63:2889–2934.
7. Langsted A, Nordestgaard BG. Nonfasting lipids, lipoproteins, and apolipoproteins in individuals with and without diabetes: 58 434 individuals from the Copenhagen General Population Study. Clin Chem 2011;57:482–489
8. NICE clinical guideline CG181. Lipid modification: cardiovascular risk assessment and the modification of blood lipids for the primary and secondary prevention of cardiovascular disease.2015.

9. Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and nonfasting lipid levels: influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. *Circulation* 2008;118:2047–2056.
10. Gaziano JM. Should we fast before we measure our lipids? *Arch Intern Med* 2012; 172:1705–1706.
11. Cohn JS, McNamara JR, Schaefer EJ. Lipoprotein cholesterol concentrations in the plasma of human subjects as measured in the fed and fasted states. *Clin Chem* 1988;34:2456–2459.
12. Mihas C, Kolovou GD, Mikhailidis DP, Kovar J, Lairon D, Nordestgaard BG, Ooi TC, PerezMartinez P, Bilianou H, Anagnostopoulou K, Panotopoulos G. Diagnostic value of postprandial triglyceride testing in healthy subjects: a metaanalysis. *Curr Vasc Pharmacol* 2011;9:271–280.
13. Tanno K, Okamura T, Ohsawa M, Onoda T, Itai K, Sakata K, Nakamura M, Ogawa A, Kawamura K, Okayama A. Comparison of low-density lipoprotein cholesterol concentrations measured by a direct homogeneous assay and by the Friedewald formula in a large community population. *Clin Chim Acta* 2010;411:1774–1780.
14. Watts GF, Cohn JS. Whither the lipid profile: feast, famine, or no free lunch? *Clin Chem* 2011;57:363–365.
15. Langsted A, Nordestgaard BG. Nonfasting lipids, lipoproteins, and apolipoproteins in individuals with and without diabetes: 58 434 individuals from the Copenhagen General Population Study. *Clin Chem* 2011;57:482–489.
16. Lund SS, Jensen T. Using nonfasting lipids— hemodilution or convenience? *Clin Chem* 2011;57:1336–1338