Special Feature: Short Communication

Non-fasting samples for estimation of serum lipid levels in patients with coronary artery disease

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ABSTRACT

Background: Evaluation of dyslipidaemia requiring fasting serum sample may not be possible in patients hospitalized in the acute phase. Evaluation of lipids in non-fasting sample was found to be useful but there are not many reports from India.

Methods: One hundred and forty five patients with coronary artery disease (CAD) patients grouped as stable angina (SA), unstable angina (UA) and myocardial infarction (MI) and 66 healthy subjects were studied. Total serum cholesterol, triglycerides (TG), high-density lipoprotein cholesterol (HDL) and non-HDL were estimated in a non-fasting serum sample.

Results: Increase in total serum cholesterol, TG, non-HDL along with a decrease in HDL levels was observed in CAD patients. The same trend was observed across the spectrum of CAD (SA, UA and MI).

Conclusions: Non-fasting sample can also be utilized in order to evaluate CAD patients hospitalized in the acute phase where waiting for obtaining a fasting sample may delay institution of specific treatement.

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INTRODUCTION

Coronary artery disease (CAD) is a leading cause of death[1] and by 2020, 60% of the world's heart disease is expected to occur in India.[1] The clinical spectrum of CAD includes stable angina (SA), unstable angina (UA), non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI). Atherosclerosis is the underlying cause of CAD. The major risk factors for atherosclerosis include hypertension tobacco smoking, dyslipidaemia, insulin resistance, diabetes mellitus (DM) and obesity. Evaluation of dyslipidaemia is usually done in fasting state in most of the laboratories in and includes estimation of serum total cholesterol, triglycerides (TG), high-density lipoprotein cholesterol (HDL) and low-density lipoprotein cholesterol (LDL). Of these, LDL is calculated using the Friedwald's formula[2] or estimated by direct methods, which are costly.

However, CAD patients are often hospitalized in the acute phase. Lipid estimations can pose a problem, if condition of fasting state is to be adhered to. To avoid this problem, random evaluation of lipids (non-fasting lipids) can be useful, which includes total cholesterol, HDL and non-HDL. Estimation of non-HDL provides information of atherogenic particles including IDL, VLDL, lipoprotein (a) and LDL.[3] The benefits of non-HDL estimation may also be cost-effective as it can be calculated from a standard lipid panel without additional expense.[4] Non-HDL concept was recommended as secondary target by Third

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National Cholesterol Education Programme-Adult Treatment Panel (NCEP-ATP-III)[3] and its importance as a measure of vascular risk has been reported.[5]

Serum TG is an independent risk factor for CAD.[6] More emphasis on the measurement and management of those with increased serum TG is seen in NCEP-ATP III[3] compared with earlier reports. TG rich lipoproteins or remnant lipoproteins are currently recognized to be atherogenic.[7] Some studies have reported association of serum triglycerides estimated in a non-fasting sample with increased risk of CVD.[8] There are not many Indian studies and the need to explore this in Indian population has been identified.[9] Hence this study was taken up to know the levels of lipids in random blood samples.

MATERIAL AND METHODS

The study was conducted in the Department of Biochemistry, Mamata Medical College and General Hospital, Khammam, Telangana, India. The patients attending outpatient department and those admitted to wards of Cardiology and General Medicine departments of Mamata General Hospital and superspeciality hospital were included in this study. Ethical clearance was obtained from Institutional Ethical Committee. Study group comprised of 145 patients diagnosed to have CAD based on clinical and biochemical criteria using electrocardiogram (ECG), echocardiogram, cardiac biomarkers (myocardial enzymes and troponin) and tread-mill test (TMT). Patients were divided into groups based on diagnosis, as SA, UA and MI. Subjects with a past history of CAD, altered kidney function (random urinary protein > 16 mg/dL and serum creatinine > 1.1 mg/dL), history of alcoholism and intake of lipid lowering drugs were excluded from the study. Sixty six age- and gender-matched subjects were recruited as control group using the same criteria. Informed consent was obtained from all the subjects. Five mL of peripheral venous blood was collected from all the subjects by venipuncture in a random fashion at the time of presentation to the hospital. Serum was separated and analyzed the same day. Blood lipids analyzed included serum total cholesterol, TG, HDL and non-HDL. Cholesterol was estimated by cholesterol oxidase method, TG by oxidase and peroxidase method, HDL by phosphotungstate precipitation method using commercial kits (Accurex, Mumbai) whereas non-HDL was calculated by subtracting HDL from total cholesterol value. Comparison of the groups was done using, one-way analysis of variance (ANOVA) using Bonferenni post-hoc test for significance between groups. Statistical analysis was done using Statistical Analysis System (SAS), version 9.3. A p value of 0.05 was considered significant.

RESULTS

A significant increase of serum cholesterol, TG, non-HDL along with a significant decrease in HDL were observed in CAD patients when compared with controls. In the spectrum of CAD (SA, UA and MI) (Table 1); mean cholesterol, total serum TG, non-HDL values showed incremental increase and mean HDL showed incremental decrease (Table 2). Among the cases, 72% had serum cholesterol levels more than 170 mg/dL (near optimal level for Indians). It was 61 % in SA cases, 75 % in ACS cases (72 % in UA and 77 % in MI). When HDL serum was taken as target, 57% of CAD cases had levels less than 40 mg/dL, while 39% of patients with SA and 62% of patients with ACS (59 % and 64 % in UA and MI respectively) had HDL levels less than 40 mg/dL. When non-HDLwith target value of 130 mg/dL was considered, 72% of CAD cases had levels more than 130 mg/dL, while 45 % of patients with SA and 76% of patients with ACS (72 % in UA and 78 % in MI) had non-HDL levels more than 130mg/dL.

Table 1: Comparison of serum lipid profile between cases and controls

Parameter (mg/dL)	Controls(n=66)	Cases(n=145)	p-value
Total serum cholesterol	162.5 ± 25.9	201.9 ± 56.8	p < 0.001
TG	122.3 ± 31.8	162.1 ± 53.8	p < 0.001
HDL	43.1 ± 4.7	39.01 ± 5.6	p < 0.001
Non-HDL	119.3 ± 24.9	161.9 ± 54.1	p < 0.001

Data are provided as mean ± standard deviation
TG = serum triglycerides; HDL = serum high-density lipoprotein cholesterol; non-HDL = serum non-HDL

Table 2: Comparison of serum lipid profile in patients with stable angina, unstable angina and acute myocardial infarction

Parameter (mg/dL)	SA	UA	MI
Total serum cholesterol	180.8 ± 37.2	186.8 ± 55.3*	215.8± 60.0†‡
TG	124.3 ± 30.4	159.34±35.0*	177.6±59.6†
HDL	41.1 ± 5.2	39.34 ± 6*	38.1 ± 5.1†
Non-HDL	137.5 ± 36.4	148.37 ± 50.7	176.4 ± 56.7†‡

Data are presented as mean ± standard deviation
* p < 0.01 significantly different from SA patients; † p<0.01 significantly different from SA patients;
‡p < 0.01 significantly different from UA patients
TG = serum triglycerides; HDL = serum high density lipoprotein cholesterol; non-HDL = serum non-HDL
SA = stable angina; UA = unstable angina; MI = myocardial infarction

DISCUSSION

In a previously reported cross-sectional study,[10] non-fasting total serum cholesterol, serum non-HDL LDL, apolipoprotein B (Apo-B), serum triglycerides, ratio of total serum cholesterol to HDL, and ratio of Apo-B to apolipoprotein A1 (Apo-A1) from the highest versus lowest tertile predicted 1.7- to 2.4-fold increased risk of cardiovascular events. However, it has been reported that serum HDL, triglycerides and total/HDL ratio predict CVD when measured nonfasting while total serum cholesterol, serum LDL, and non-HDL measurement, provided less useful CVD risk information when measured in a nonfasting sate.[11] Atherosclerosis which underlies CAD, is a multifocal, chronic, immunoinflammatory and fibro proliferative disease of large and medium sized arteries fuelled by lipids. Among the multiple cardiovascular risk factors, elevated cholesterol is most unique in being sufficient to drive the development of atherosclerosis. The risk of elevated cholesterol is augmented by lowered HDL and Apo-A1 lipoprotein which confer protection against atherothrombosis.[12] NCEP-ATP-III guidelines suggested non-HDL as a screening test which includes atherogenic lipoproteins, LDL and VLDL.[13] Postprandial lipaemia is gaining importance with recent reports showing nonfasting TG to independently predict atherosclerosis.[8] With each input of postprandial TG-rich lipoproteins (TRL), cholesteryl esters will be transferred from HDL to TRL through the action of cholesteryl ester transfer protein. Translocation of cholesteryl esters from HDL to TRL can contribute to the atherogenic potential of these particles because the cholesteryl esters originating from HDL and now transported in TRL will remain with these particles along their lipolytic cascade and the endocytic pathways of their remnants. These particles can penetrate into the arterial wall, deliver cholesterol into the intima, and eventually lead to development of atherosclerosis.[14]

The findings of the present study thus suggest that non-fasting serum TG can also be utilized in order to evaluate CAD along with non-HDL.

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