IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 4 Ser. 18 (April. 2019), PP 25-31
www.iosrjournals.org

Study of Fasting and Non-Fasting Serum Lipid Profile in Coronary Artery Disease

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Abstract: *IHD is a consequence of reduced coronary blood flow secondary to obstructive atherosclerotic vascular disease. Triglycerides are routinely measured in the fasting state. Atherosclerosis may be a postprandial phenomenon in which remnant lipoproteins play a dominant role. If this is true, increased levels of non-fasting triglycerides, reflecting increased levels of remnant lipoproteins, may predict risk of myocardial infarction (MI), ischemic heart disease (IHD), and death. The aim of this study was to evaluate and compare the values of fasting and non-fasting serum lipid profile among patients of coronary artery disease. This is a cross sectional study, conducted in the Department of Biochemistry in collaboration with the Department of Cardiology, RIMS. The study population consisted of 80 patients above 18 years suffering from coronary artery disease. The mean values of total cholesterol, HDL and LDL in the fasting state is minimally changed when compared to their mean values in the non-fasting state. The mean value of triglyceride was raised in the non-fasting state when compared to the fasting level. It may be concluded that non-fasting blood samples can be routinely used for assessment of plasma lipid profile.*

Key words- *Ischemic Heart Disease(IHD), Chylomicrons, Low Density Lipoproteins(LDL), High Density Lipoproteins(HDL), Triglycerides.*

Date of Submission: 15-04-2019 Date of acceptance: 30-04-2019

I. Introduction

Ischemic heart diseadse(IHD) is a consequence of reduced coronary blood flow secondary to obstructive atherosclerotic vascular disease. Thus, unless otherwise specified, IHD usually is synonymous with coronary artery disease (CAD). In most cases, the syndromes of IHD are the late manifestations of coronary atherosclerosis that has been gradually building for decades (beginning even in childhood or adolescence).[1] With moderate hypertriglyceridemia, chylomicron remnants and very low-density lipoprotein remnants are present in plasma. These smaller triglyceride-rich lipoproteins penetrate the arterial intima and appear to be preferentially trapped within the arterial wall. Triglycerides are routinely measured in the fasting state excluding remnant lipoproteins; however, except for the first hours in the early morning, most individuals are in the nonfasting state most of the time. Atherosclerosis may be a postprandial phenomenon in which remnant lipoproteins play a dominant role. If this is true, increased levels of nonfasting triglycerides, reflecting increased levels of remnant lipoproteins, may predict risk of myocardial infarction (MI), ischemic heart disease (IHD), and death.[2]

The necessity for estimation of fasting and nonfasting lipid profile level for predicting coronary artery disease and its prognosis is variable and contradictory between different studies conducted by various investigators. In some studies it is suggested that an elevated postprandial lipemic response precipitates a number of adverse metabolic events, including the production of atherogenic chylomicron remnants, the formation of the highly atherogenic small, dense low density lipoprotein particles, and a reduction in the concentration of the cardioprotective high density lipoprotein fraction[3] while some studies suggest that a high level of fasting lipid profile is a strong indicator of ischemic heart disease.[4] The present study is taken up with this view in mind to study fasting and nonfasting lipid profile among the patients with coronary artery disease and to see if there is any significant difference in the levels of fasting and nonfasting lipid profile levels.

II. Material And Methods

The study was a cross sectional study conducted in the Department of Biochemistry in collaboration with the Department of Cardiology, Regional Institute of Medical Sciences & Hospital, Imphal, Manipur for a period of 24 months from September 2016 to August 2018.

Study Design: A crsoss sectional study.

Study Location: This was a tertiary care teaching hospital based study done in Department of Biochemistry in collaboration with Department of Cardiology, RIMS, Imphal.

Study Duration: September 2016 to August 2018.

Sample size: 80 patients.

Sample size calculation: Taking the standard deviation 34.1gm and standard error 8 of postprandial serum lipid profile the sample size has been calculated by using the formula, $\text{Sample size} = 4 \times \frac{S^2}{e^2}$, where, S (standard deviation) - 34.1gm, L (margin of error) – 16,e (standard error) – 8, precision – 95%

Subjects & selection method: The study population consisted of 80 patients above 18 years suffering from coronary artery disease and the patients were chosen from those admitted in the cardiology ward of RIMS, Imphal.

Inclusion criteria: 80 patients who were admitted within 12 hours after onset of symptoms in ICCU or Medicine Ward of RIMS Hospital having typical ischaemic symptoms, and whose test reports for CK-MB & Trop-I showed positive results were the study population.

Exclusion criteria: Patients suffering from chronic heart failure, hepatic and renal disease, malignancy and anaemia were excluded from the study.

Procedure methodology

5 ml of venous blood was collected, each in the fasting and non-fasting state by venipuncture from antecubital vein. The blood collected in the plain vial was centrifuged for 10 minutes within 30 minutes of collection and the serum was stored immediately at < -20 °C. Other investigation parameters were collected from the documentation of routine investigations done in the hospital. Serum lipid profile estimation was done by Enzymatic Colorimetric Test with lipid clearing factor (LCF) by using kits marketed by Human Gesellschaft fur Biochemica und Diagnostica mbH through its Indian branch supply. Approval of Research Ethics Board, RIMS, Imphal was taken. Informed consent was taken from the participants before the study and confidentiality were maintained.

Statistical analysis

The results available were analysed using SPSS version 20. Chi-square test was used to ascertain the significance of differences between mean values. The level $P < 0.05$ was considered as the cutoff value or significance.

III. Result

A. Socio demographic variables

In Table 1, shows the number of cases according to their age group. Maximum of the cases were in the age group between 61 and 70 years. Mean and standard deviation value of the age of cases was 61.70 ± 11.07 years.

Table 1: Age wise distribution of cases

Age in years	Casesn (%)
30 – 40	3 (3.75)
41 – 50	13 (16.25)
51 – 60	20 (25.00)
61 – 70	27 (33.75)
71 – 80	16 (20.00)
>81	1 (1.25)
Total	80 (100.4)
Mean ± SD	61.70 ± 11.07

In Table 2, sex of cases were presented in number and percentages. Males constituted 71.25% of the cases and the rest 28.75% of cases were females.

Table 2: Distribution of cases by sex.

Sex	Cases n (%)
Male	57 (71.25)
Female	23 (28.75)
Total	80 (100)

In Table 3, address/area of inhabitance of cases were presented in number and percentages. Cases were maximum from urban area.

Table 3: Distribution of cases as per address/area of inhabitance.

Area of inhabitance	Cases n (%)
Urban	46 (57.5)
Rural	34 (42.5)
Total	80 (100)

B. Outcomes parameters

In table 4, the mean and the standard deviation values of lipid profile in the fasting and non-fasting state are shown.

Table 4: Mean and standard deviation values of lipid profile in the fasting and non-fasting state.

	Fasting (Mean ± SD)	Non-fasting (Mean ± SD)
Cholesterol	169.68 ± 47.208	169.61 ± 42.863
Triglyceride	157.00 ± 91.346	187.23 ± 86.940
HDL	37.38 ± 12.146	36.73 ± 11.173
LDL	103.83 ± 38.868	95.30 ± 34.614

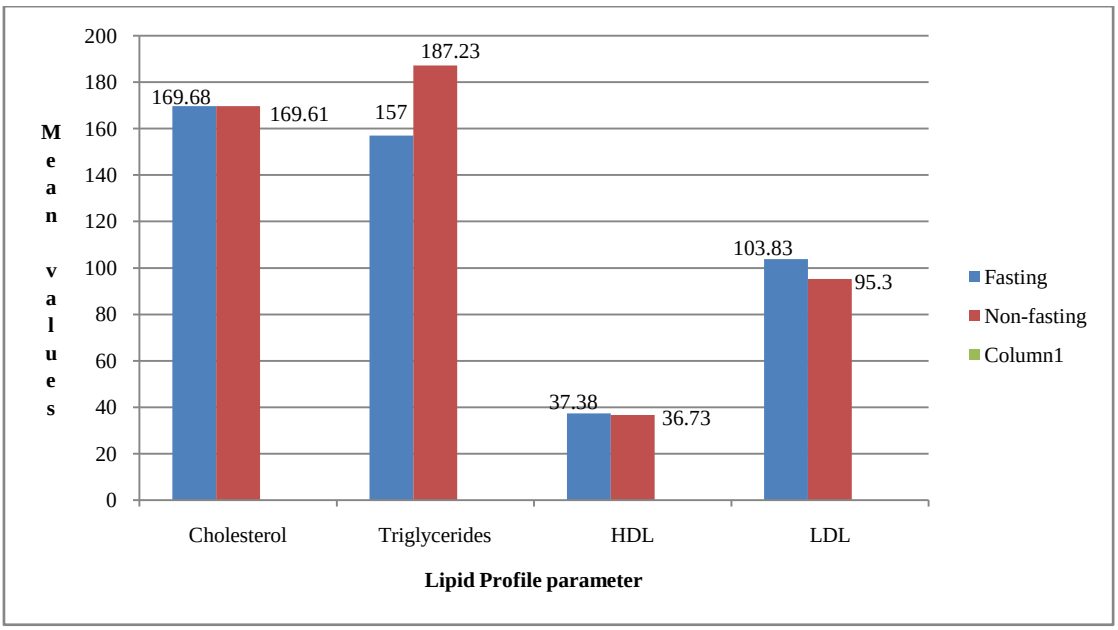

Figure 4: Bar diagram showing comparison between the mean values of fasting and non-fasting lipid profile.

Table 5, shows the number of cases having normal or raised fasting and non-fasting cholesterol level. The comparison between the fasting and non-fasting cholesterol level was done by chi-square test. Most of the cases (60) had not much change in the fasting and the non-fasting cholesterol level. This finding was statistically significant ($p<0.05$).

Table 5: Comparison between fasting and non-fasting cholesterol levels in cases.

		Non-fasting cholesterol		p-value
Fasting cholesterol		<200mg/dl	>200mg/dl	0.000
	<200mg/dl	60	1	
	>200mg/dl	4	15	

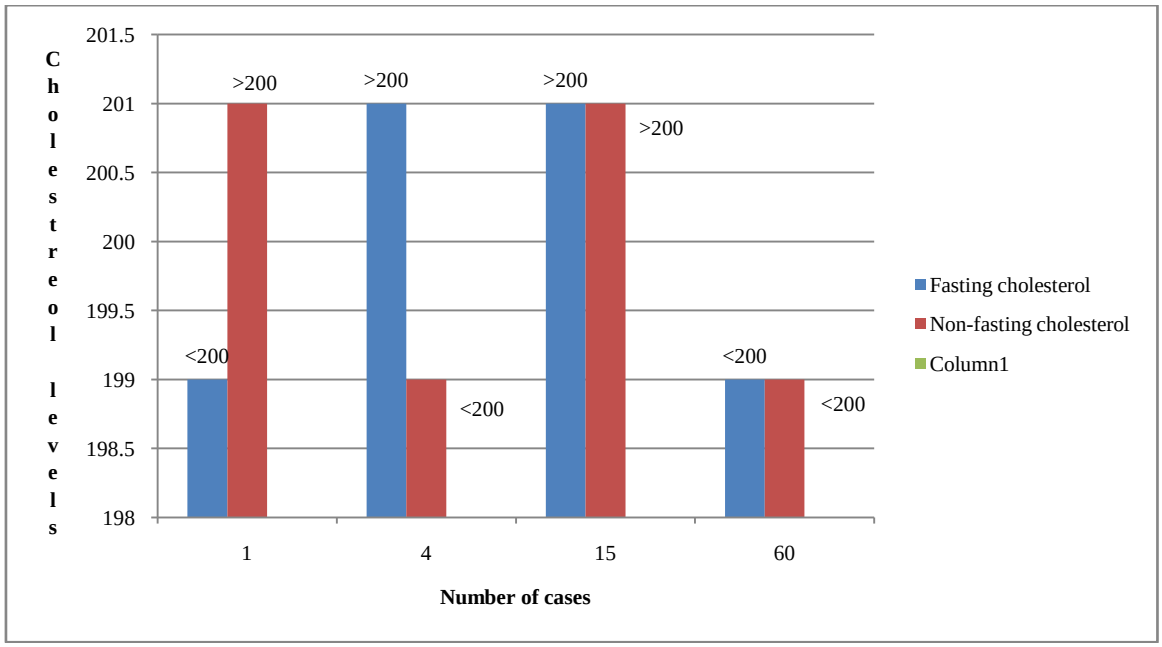

Figure 5: Bar diagram showing comparison between fasting and non-fasting cholesterol levels.

Table 6, shows the number of cases having normal or raised fasting and non-fasting triglyceride level. The comparison between the fasting and non-fasting triglyceride level was done by chi-square test. Most of the cases (29) had not much change in the fasting and the non-fasting triglyceride level. This finding was statistically significant ($p<0.05$).

Table 6: Comparison between fasting and non-fasting triglyceride levels in cases.

		Non-fasting Triglycerides		p-value
Fasting Triglycerides		<150mg/dl	>150mg/dl	0.000
	<150mg/dl	29	21	
	>150mg/dl	2	28	

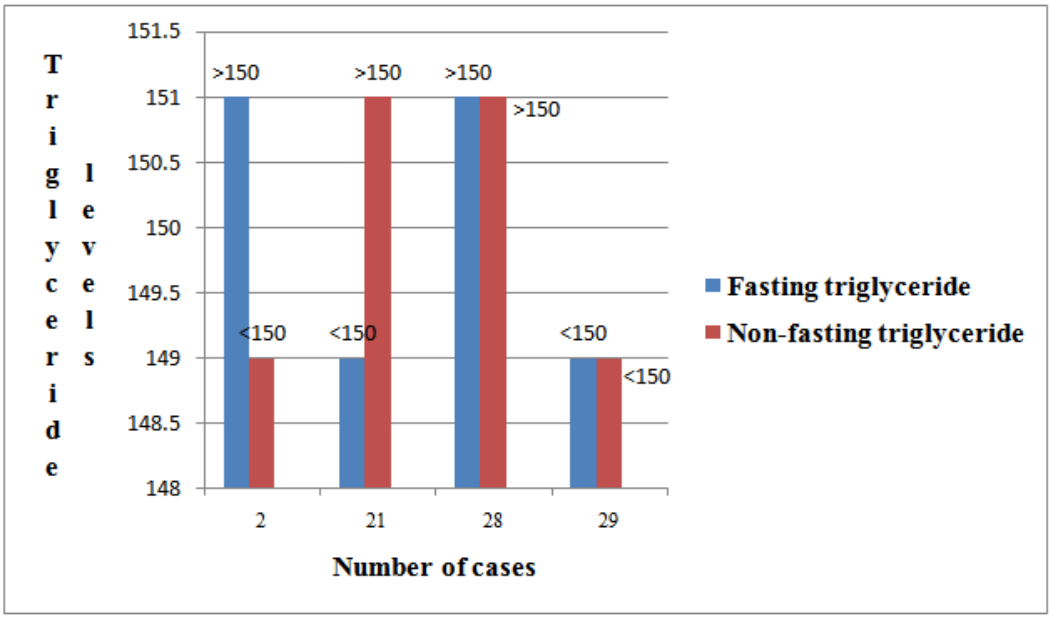

Figure 6: Bar diagram showing comparison between fasting and non-fasting triglyceride levels.

Table 7, shows the number of cases having normal or raised fasting and non-fasting HDL level. The comparison between the fasting and non-fasting HDL level was done by chi-square test. Most of the cases (44) had not much change in the fasting and the non-fasting HDL level. This finding was statistically significant ($p<0.05$).

Table 7: Comparison between fasting and non-fasting HDL levels in cases**.**

		Non-fasting HDL		p-value
Fasting HDL		<40mg/dl	>40mg/dl	0.000
	<40mg/dl	44	6	
	>40mg/dl	3	27	

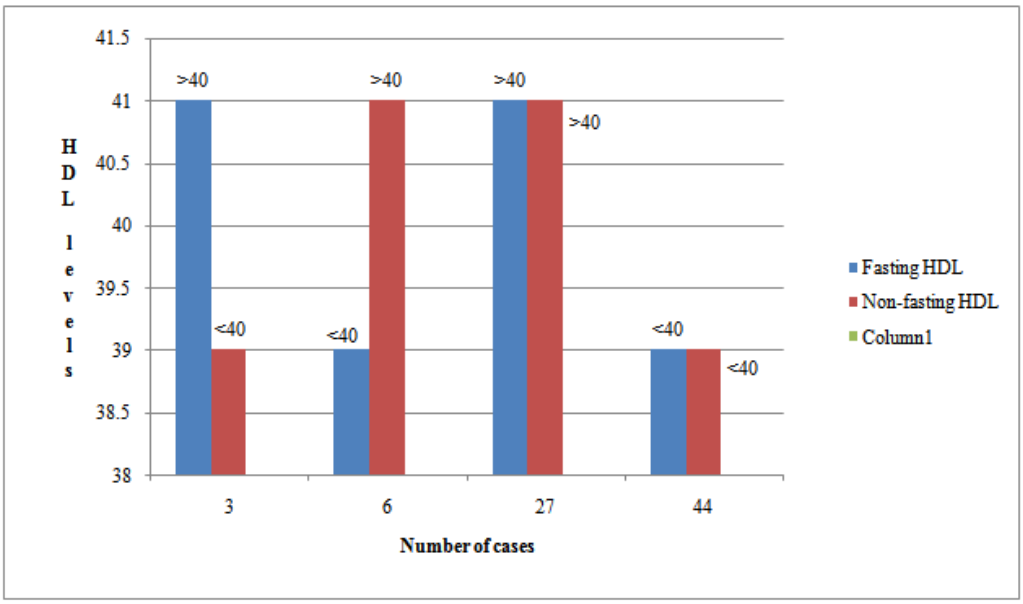

Figure 7: Bar diagram showing comparison between fasting and non-fasting HDL levels.

Table 8, shows the number of cases having normal or raised fasting and non-fasting LDL level. The comparison between the fasting and non-fasting LDL level was done by chi-square test. Most of the cases (59) had not much change in the fasting and the non-fasting LDL level. This finding was statistically significant ($p<0.05$).

Table 8: Comparison between fasting and non-fasting LDL levels.

		Non-fasting LDL		p-value
Fasting LDL		<130mg/dl	>130mg/dl	0.000
	<130mg/dl	59	1	
	>130mg/dl	8	12	

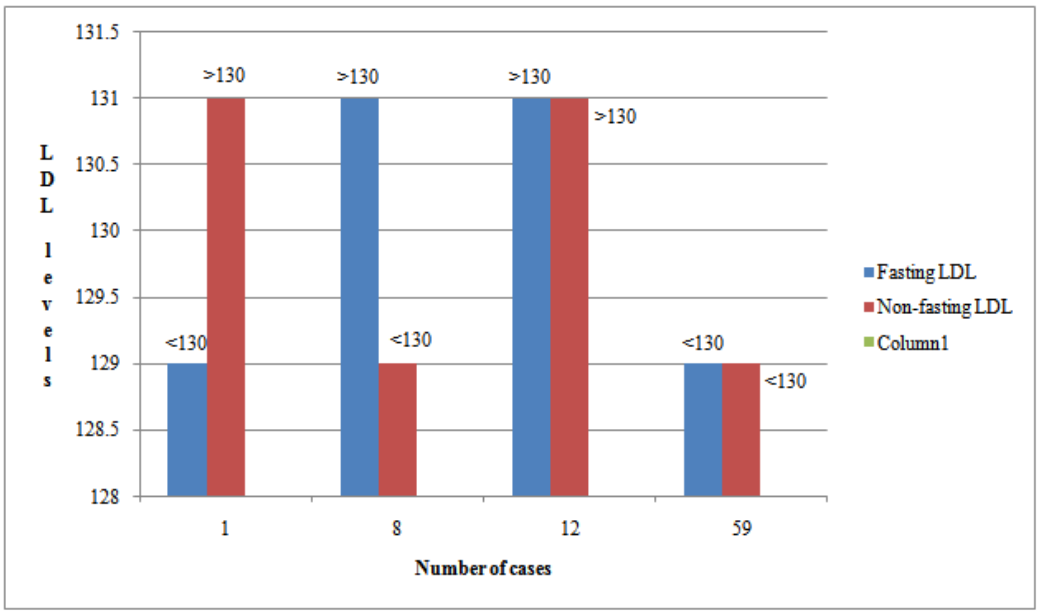

Figure 8: Bar diagram showing comparison between fasting and non-fasting LDL levels.

IV. Discussion

Serum lipid profile is one of the major risk factor for coronary heart disease and fasting lipid profile is done invariably in patients, both who are at risk and who develop coronary heart disease. This study was done with the aim to see whether non-fasting lipid profile could replace fasting, since most hours of the day of an individual is spent in a non-fasting state and non-fasting lipid profile would simplify blood sampling procedure

In this study, most of the cases were elderly males. This is evident from table 1, which shows that maximum number of cases i.e. 33.75% were in the age group between 61 – 70 years followed by 25% in 51 – 60 age group, 20% in 71 – 80 age group, 16.25% in 41 – 50 age group, 3.75% in 30 – 40 age group and 1.25% in >80 years age group. Mean age of cases was 61.7 years. Table 2 shows that most of the cases constituted of male population i.e. 71.25% and the rest 28.75% were females. These findings were consistent with the observation of a study done by Goulart AC et al[5], in which the mean age was 62.7years and 58.5% were men. With aging, there is an incremental acquisition of several CVD risk factors in an individual's lifespan. When these risk factors are incorporated in a multivariable regression model, age still remains an independent risk factor. The burden of CVD risk associated with rising age can be reduced partly by the modification of traditional coexisting CVD risk factors.[6]

It is seen from Table 4, that the mean values of total cholesterol, HDL and LDL in the fasting state is minimally changed when compared to their mean values in the non-fasting state. The mean value of triglyceride was raised in the non-fasting state when compared to the fasting level. The mean values of total cholesterol, triglyceride, HDL and LDL in the fasting state was 169.68mg/dl, 157mg/dl, 37.38mg/dl and 103.83mg/dl respectively and in the non-fasting state it was 169.61mg/dl, 187.23mg/dl, 36.73mg/dl and 95.30mg/dl respectively. These findings were in accordance to a study done by Langsted A et al[7] who in their study has shown that maximum changes after normal food and fluid intake from fasting levels were − 0.2 mmol/L for total cholesterol, − 0.2 mmol/L for low -density lipoprotein cholesterol , − 0.1 mmol/L for HDL cholesterol, and 0.3 mmol/L for triglyceride and concluded that lipid profiles at most change minimally in response to normal food intake in individuals in the general population and nonfasting lipid profiles predicted increased risk of cardiovascular events.

In this study when the values of total cholesterol, HDL and LDL were compared in the fasting and the non-fasting state, most of the cases showed minimal alteration in their values which was not clinically significant and this finding was similar to a study done by Nordestgaard BG et al[8] who indicated that the maximal mean changes at 1–6 h after habitual meals are not clinically significant [+0.3 mmol/L(26 mg/dL) for triglycerides;− 0.2 mmol/L(8 mg/dL) for total cholesterol ; − 0.2 mmol/L(8 mg/dL) for LDL cholesterol; +0.2 mmol/L (8 mg/dL) for calculated remnant cholesterol ; − 0.2 mmol/L (8 mg/dL) for calculated non-HDL cholesterol]; concentrations of HDL cholesterol, apolipoprotein A1, apolipoprotein B, and lipoprotein(a) are not affected by fasting/non-fasting status in addition, non-fasting and fasting concentrations vary similarly over time and are comparable in the prediction of cardiovascular disease.

Serum triglyceride level remains elevated for a few hours after meal and has been implicated to be potentially atherogenic. The present study showed that non-fasting triglyceride level was raised when compared to their fasting level and this was similar to the findings suggested by Bansal S et al[9], who in his study mentioned that postprandial lipids may play an important role in the pathogenesis of cardiovascular disease because postprandial triglyceride-rich remnant lipoproteins can penetrate the endothelial cell layer and reside in the subendothelial space, where they can contribute to the formation of foam cells, a hallmark of early atherosclerosis and concluded that nonfasting triglyceride levels were associated with incident cardiovascular events. Fatima S et al[10] found in her study that the diagnostic accuracy of non-fasting lipid profile was significantly higher than fasting lipid profile for the assessment of lipoprotein coronary risk on the basis of non-HDL-C, which seemed to be significant test for ruling out hyperlipidemia

V. Conclusion

The results of this study shows that the levels of non-fasting triglyceride, which is more atherogenic, when compared to the fasting levels was raised in the cases. There was not much difference between the fasting and the non-fasting levels of serum cholesterol, HDL and LDL, and this was not clinically important. This is the first study in Indian Manipuri population with coronary artery disease to compare the fasting and non-fasting lipid profile levels. This study demonstrates that lipid profile test can be done in the non-fasting state and the potentially atherogenic non-fasting triglyceride will be more informative in predicting the risk of coronary artery disease.

So, it may be concluded that non-fasting blood samples can be routinely used for assessment of plasma lipid profile.

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Dr. Tina Das. "Study of Fasting and Non-Fasting Serum Lipid Profile in Coronary Artery Disease." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 04, 2019, pp 25-31.