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## COMPARATIVE ASSESSMENT OF FASTING AND NON-FASTING CONVENTIONAL LIPID PROFILE IN HYPERTENSIVE PATIENTS

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### ABSTRACT

**Background:** Lipid profile is a crucial test for patients visiting the cardiology clinic. An overnight fasting sample is advised which is usually burdensome and causes low compliance with illness monitoring and therapy. This study determined the relationship between fasting and non-fasting state with lipids and some antioxidants in hypertensive patients.

**Methods:** Three hundred and seventy known hypertensive patients were selected. The study was a longitudinal cohort and self-controlled. Three blood samples per participant were collected after 8-12 h fasting, 1 h, and 2 h after consumption of their customary meal. The lipid profile was determined daily over a period of 6 months, using the spectrophotometry method.

**Results:** The mean serum lipid levels at fasting, 1 h, and 2 h, respectively were, TC: 197.28±50.6, 189.69±49.7 and 191.09±51.4; TG: 84.0±31.3, 92.97±34.4 and 96.99±38.5; HDL-c: 58.0±15.2, 54.25±14.8 and 52.73±16.2; VLDL: 16.37±6.7, 19.47±6.4 and 20.50±6.0; LDL-c: 121.0, 120.0 and 118.0 mg/dL,  $p < 0.05$  in all parameters.

**Conclusion:** The study's findings demonstrated statistically significant ( $p < 0.05$ ) but not clinically important changes in lipid parameters, between fasting and non-fasting levels as all values fell within the acceptable range, it can therefore be concluded that conventional lipid profile analysis can be done on non-fasting samples.



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**Keywords:** Lipid profile, fasting state, postprandial, hypertension, self-controlled.

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## INTRODUCTION

The primary components of the lipid portion of the human body are saturated fat, triglycerides, and high-density lipoproteins (HDL). Saturated fat is the main element of cell membranes and an unsaturated alcohol of the steroid family is imperative for the healthy operation of all animal cells. Anytime these lipids are out of balance, the threat of cardiovascular diseases (CVDs), principally hypertension, increases (Mathew and Ramacandra, 2016; Obeagu *et al.*, 2018).

Serum lipid testing is one of the crucial procedures at the cardiology outpatient clinic. Because the levels of triglycerides in non-fasting blood vary widely, numerous guidelines advocate the use of fasting blood samples for determining cardiovascular risk (White *et al.*, 2015; Mathew and Ramachandra, 2016). Additionally, non-fasting triglyceride measurements may result in an underestimation of low-density lipoproteins cholesterol (LDL-c), when calculated from the Friedewald equation ( $LDL = Total\ cholesterol - HDL - VLDL$ ) (Martin *et al.*, 2013; Baibata *et al.*, 2015; Alsiad *et al.*, 2020).

Oldness, sex, as well as ethnic group are non-modifiable cardiovascular threat that cannot be altered, while diabetes mellitus, elevated blood cholesterol, hypertension, obesity, cigarette use, and physical inactivity are modifiable cardiovascular risk factors (Robinson and Stone, 2015; DeFilipis *et al.*, 2017).

Ghildiyal *et al.* (2020) argued in favor of non-fasting samples during screening for lipid disorders because of the stress experienced by most patients who might have eaten before a routine clinic visit and

thus, must otherwise prepare for subsequent tests visits to the doctor's office or planned stops to an outpatient phlebotomy facility. Patients find it inconvenient and unpleasant to have to fast for 12–14 h (more than 8 h), which results in low compliance with illness monitoring and treatment, exposing at-risk patients to a greater risk of unfavourable cardiovascular outcomes.

A person with the systolic and/or diastolic blood pressures  $\geq 140$  mmHg and  $\geq 90$  mmHg, respectively, is deemed to be hypertensive. Early signs typically appear before continuously increased blood pressure (BP), making it a chronic cardiovascular condition with complicated and linked etiologies. Hence, discrete BP thresholds alone cannot be used to identify hypertension. However, it is a persistent medical issue that is frequently asymptomatic and detected through screening. Heart, kidney, brain, and other organ damage brought on by the disease's progression causes early morbidity and death (Mathew and Ramachandra, 2016; Johannesen *et al.*, 2020).

Globally, 972 million individuals from all social classes and financial levels may be at danger due to the severe health threat for hypertension (Leng *et al.*, 2015; Ference *et al.*, 2016). The main threat for myocardial infarction, heart failure, stroke, and renal failure include hypertension and dyslipidemia. (Johanssen *et al.*, 2020; Grundy *et al.*, 2018).

Hypertensions is of two types; "main or essential" and "secondary." Although the multifactorial aetiology of essential hypertension includes modifiable factors like food, such as excessive sugar and salt intake or vitamin deficiencies, it also has a hereditary component. Over 85% of essential hypertension are caused by the



interaction of genetic background and environmental conditions. The precise genes that predispose most persons to hypertension are yet unclear. Some authorities claim that the cause of essential hypertension is unknown, while others claim that excessive sodium and inadequate potassium consumption are to blame (WHO, 2012; Mozaffarian *et al.*, 2014; GBD, 2016; Klimetidis *et al.*, 2020).

Secondary hypertension results from a specific underlying ailment of a recognised mechanism, such as lingering kidney ailment, tightening of the aorta or kidney arteries, endocrine anomalies like superfluous aldosterone, cortisol, or catecholamine (Funder *et al.*, 2016; Gornik *et al.*, 2019).

Aortic aneurysm, peripheral artery disease, lingering kidney disease, hypertensive heart disease, and coronary artery disease are all conditions that are greatly increased by persistent hypertension (Bhatt *et al.*, 2016; de Jager *et al.*, 2018). Modifying one's lifestyle can decrease blood pressure, besides is still the cornerstone of treating and averting high BP (Costa *et al.*, 2018; Fedak *et al.*, 2019). Other routine adjustments include limiting salt intake, abstaining from alcohol, consciously switching to a diet high in vegetables and fruits, losing weight, and fasting.

Measuring fasting serum lipids is a crucial step in determining cardiovascular risks. There are two basic reasons why lipids are typically drawn after a fast. Since eating can change some lipid levels, the initial step was to reduce variance. The second goal was to create a more accurate computation of LDL cholesterol, which is frequently obtained via an equation that is thought to yield significantly skewed values after eating. However, more recent research has substantially disproved these worries. The current consensus among scientists is that eating has minimal,

clinically negligible impact on the conventional lipids. Where elevated triglycerides level is noticeable after meal, physician can request for a fasting triglyceride test (Anderson *et al.*, 2016; Scartezini *et al.*, 2017). However, there is a drawback on using fasting samples, particularly for diabetics, young children, the elderly, and in population screening (Anderson *et al.*, 2016; Scartezini *et al.*, 2017). The issue now is which sort of sample, fasting or non-fasting is optimal for lipid investigation. Non-fasting/random blood sample has consideration for the time of last meal.

Random blood samples may best reflect the natural physiological state, because human beings spend most time in non-fasting state, although fasting samples have historically been the gold standard for measuring lipid profiles. Studies by Farukhi and Mora (2016) reported that non-fasting lipids are better than fasting in monitoring atherosclerotic cardiovascular disease (ASCVD). In a prospective analysis, Doran *et al.* (2014) observed no distinction between the risk correlations of non-fasting and fasting lipid levels.

The most communal lingering ailment is systemic arterial hypertension, and a key danger issue for cardiovascular disease (CVD) of endemic proportions. Management of hypertension is achieved by merging medication with lifestyle changes such as reducing salt intake, increase physical exercise, weight reduction, alcohol consumption reduction, and a healthy diet (NICE, 2015). Nevertheless, hypertensive patients often present with unfriendly glycaemic and lipid profiles compared to non-hypertensive patients. Monitoring these is important in the management of cardiovascular-related diseases.

Systemic arterial hypertension, which is also a significant threat cause for CVD of



pandemic proportions, is the most prevalent chronic disease. Combining medicine with lifestyle modifications, such as eating less salt, exercising more, losing weight, drinking less alcohol, and maintaining a nutritious diet, can help manage hypertension (NICE, 2015). In contrast to non-hypertensive patients, hypertensive patients frequently have unfavorable glycemic and lipid profiles. In the therapy of cardiovascular-related disorders, monitoring these is crucial.

- I. The risk of cardiovascular disease may be more accurately predicted by non-fasting triglycerides than by fasting triglycerides (Nordesgaard *et al.*, 2016).
- II. Blood sampling is made simple for individuals, laboratories, and medical professionals by using a non-fasting sample (Langsted and Nordestgaard, 2015).

## METHOD

### STUDY AREA

A total of 370 known volunteer hypertensive patients were enrolled, from the Cardiac Outpatient Unit of Lagos State University Teaching Hospital, Ikeja Lagos, and Participants' ages ranged from 25 to 65. The study is a longitudinal cohort study, each participant's three samples notably fasting, one-hour postprandial (1HrPP), and two hours postprandial (2HrPP) were taken separately.

### ANTHROPOMETRIC INDICES

#### HEIGHTS AND WEIGHT

Each subject's weight was measured using a bathroom scale. Before the weight measures could be obtained, each subject was asked to remove any bulky clothing, jewelry, and shoes; empty their pockets;

and stand in the middle of the bathroom scale. The weights were measured and recorded to 0.1 kg precision.

All participants were asked to stand still or upright, remove their shoes, jewelry and hair accessories, a stadiometer was used to measure their height. They stood with their feet together and their heels, bottoms, calves, and backs contacting a vertical surface. The participant looked straight ahead as the height was measured to the closest meter.

The body mass index (BMI) was calculated with the formula:  $BMI = \text{Weight}/\text{Height}^2$  ( $\text{kg}/\text{m}^2$ ) and classified as underweight ( $< 18.5 \text{ kg}/\text{m}^2$ ), healthy weight ( $18.5\text{--}24.9 \text{ kg}/\text{m}^2$ ), overweight ( $25.0\text{--}29.9 \text{ kg}/\text{m}^2$ ), obesity I ( $30.0\text{--}34.9 \text{ kg}/\text{m}^2$ ), obesity II ( $35.0\text{--}39.9 \text{ kg}/\text{m}^2$ ), and obesity III ( $\geq 40.0 \text{ kg}/\text{m}^2$ ) (WHO, 2014). Participants who fell under the obese group were excluded from participation.

#### *BLOOD PRESSURE MEASUREMENT*

Participants were comfortably seated with their arms resting while having their BP taken with a mercurial sphygmomanometer. The Omron electronic (digital) sphygmomanometer was used to measure the SBP and DBP. Each participant had their BP measured twice, with the average reading being recorded.

#### *EXCLUSION CRITERIA*

Excluded from the study were.

- I. Obese hypertensive patients/individual
- II. Pregnant hypertensive patients/individual
- III. Those who have fasted for more than 12 hours overnight.

#### *INCLUSION CRITERIA*



Included in the study were:

- I. Known to be hypertensive (long-time or newly established)
- II. Those between the ages of 25 and 65, subject
- III. Those who willingly volunteer to participate in the study.
- IV. Those without records of any other systemic illnesses.

**SAMPLE SIZE:**

The sample size (n) was determined using the formula,

$$N = \frac{Z^2pq}{d^2}$$

Where:

N = Minimum sample size

Z = Statistical level of confidence at 95% = 1.96

P = prevalence rate of hypertension in target population ((38.2% in Lagos Daniel et al

2013) = 0.382

d = precision (0.05)

q = 1-P= 1-0.382=0.618

$$. N = \frac{(1.96)^2 \times 0.382 (1-0.382)}{0.05^2}$$

$$N = \frac{3.84 \times 0.382 \times 0.618}{0.0025}$$

$$N = \frac{0.907}{0.0025}$$

$$N = 362.61$$
$$= 363$$
$$= 370$$

**DATA COLLECTION**

A thorough questionnaire was created to collect the participants' sociodemographic

**BIOCHEMICAL ANALYSIS**

**• LIPID PROFILE**

✓ Total cholesterol determination

details, including their educational background, socioeconomic standing, income, and type of employment. Anthropometric measurements and family history of any systemic illnesses were also collected. Additionally, information was gathered regarding the use of herbal remedies, anti-hypertensive drugs, and multivitamin supplements. This served as the standard for inclusion and exclusion.

➤ Collection of samples

Each participant had 5 ml of venous blood drawn after an overnight fast of 8 to 12 hours, which served as the baseline for subsequent blood tests. Participants were then instructed to eat their regular breakfast and take their anti-hypertensive medication within 15 minutes. Then, two more samples were taken from each participant at 1 and 2 hours after the meal. Two 5ml vials of plain and EDTA were used for the blood sample. After allowing samples to coagulate in plain vials, the samples were separated by centrifugation at 5000 rpm for five minutes. And the lipid profile was analyzed on the day of sample collection.

✓ Instruments and consumables used for study

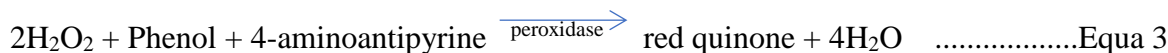
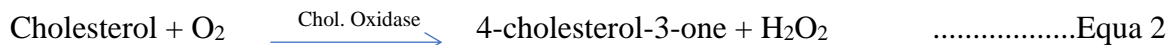
A bathroom scale, an Omron digital sphygmomanometer, a stadiometer, a tourniquet, vacutainer bottles, needles, and syringes, as well as automated pipettes, digital balances, Selectra PROXs automated machines was used in this study.



Quantitative analysis of plasma total Cholesterol by enzymatic Colorimetric assay approach illustrated by Schettler and Nussel (1975).

Assay Principle:

The sample's cholesterol was quantified after enzymatic hydrolysis and oxidation. The indicator quinoneimine is produced from 4-aminantipyrene and hydrogen peroxide in the presence of phenol and peroxide. Equation of the reaction:



The sample's cholesterol concentration directly correlates with the intensity of the color produced (Beer Lambert law).

This was determined by measuring the absorbance at 505nm.

✓ Triglycerides determination

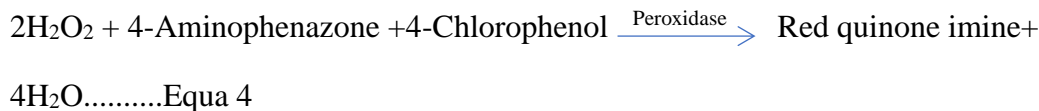
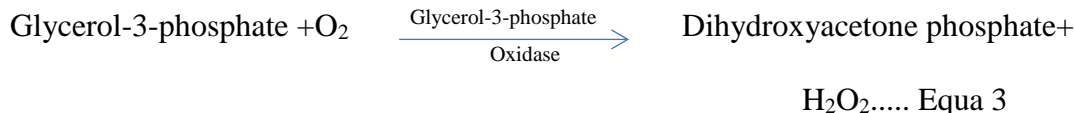
Quantitative determination of plasma triglyceride was done by enzymatic colorimetric method described by Nagele *et al.* (1984).

Assay Principle:

Quantitative determination of plasma triglyceride was done by enzymatic colorimetric method described by Nagele *et al.* (1984).

Assay Principle:

Triglycerides are identified through enzymatic hydrolysis using lipases. Peroxidase uses hydrogen peroxide, 4-aminophenazone, and 4-chlorophenol as raw materials to catalyze the production of the indicator quinoneimine. Equation for the reaction:



Absorbance was measured at 505nm.



- Determination of HDL-C

Quantitative determination of plasma triglyceride was done by enzymatic colorimetric method described by Friedwald *et al.* (1972).

Reagents: Phosphotungstate (0.55mmol/l), Magnesium chloride (25mmol/l)

Determination is based on selective precipitation method using direct HDL-c determination.

Assay principle:

The addition of phosphotungstic in the presence of magnesium ions precipitated low density lipoprotein (LDL and VLDL) and chylomicron fractions.

Following centrifugation, the HDL-c fraction's cholesterol level was calculated using the same method as for total cholesterol.

- Determination of HDL-C and VLDL

Since the TG was less than 4.5 mmol/L, the LDL-c level was calculated using the Friedwald formula:  $LDL-C = TC - [HDL-C] - [TG/2.2]$  (Friedwald *et al.*, 1972). Otherwise, a direct measurement using a chemical masking approach would have been employed.

- Determination of very low-density lipoprotein-VLDL

On the theory that the TG: cholesterol ratio of VLDL was constant at roughly 5:1, the VLDL was calculated by dividing the triglycerides by five.

## ETHICAL APPROVAL

The LASUTH/LASUCOM ethical committee was consulted for ethical approval. Before the study began, all participants also willingly signed the informed consent after being fully told about the purpose of the research and receiving all necessary information. The data were handled confidentially, the patient's privacy was protected, and they were solely used for this study. A well-crafted questionnaire was administered to all participants.

## RESULT AND DISCUSSION

**Table 1: Socio-demographic characteristics of participants**

<b>Variables</b>	<b>Frequency n= 370</b>	<b>percentage %</b>
<b>Age (years)</b>	<b>x=56.26±8.55</b>	
31-40	28	7.7
41-50	55	14.8
51-60	181	49
61-70	106	28.5
<b>Gender</b>		
Male	174	47
Female	196	53
<b>Educational level</b>		





No formal education	86	23.3
Primary	126	34.1
Secondary	144	38.9
Tertiary	14	3.7
<b>Marital status</b>		
Single	28	7.4
Widow/widower	82	22.2
Married	197	53.3
Divorcee	63	17.1
<b>Medication</b>		
Single	129	34.8
Combined	141	65.2
<b>Socio-economic status</b>		
Low income	182	49.3
Middle income	123	33.3
High income	65	17.4
<b>Herbal consumption</b>		
Daily	152	41.1
Weekly	126	34.1
Occasionally	81	21.8
None	11	3
<b>Multivitamin supplements</b>		
Daily	115	31.1
Weekly	90	24.4
Occasionally	32	8.5
None	133	36
<b>Physical activity</b>		
Daily	52	14.1
Weekly	114	30.7
Occasionally	63	17
None	141	38.2
<b>Occupation</b>		
Full housewife/ retiree	73	19.6
Petty trader/		Artisan/ Driver
108	29.3	
Junior civil servant	104	28.1
Senior civil servant	85	23
Business owner	Nil	00

A total of 370 individuals were enlisted for this study, Regarding the participants' ages, the mean age was  $56.29 \pm 8.55$  years,. The age group 51 to 60 years had the highest frequency, which may be related to the fact that age increases the chance of developing hypertension (Shukuri *et al.*, 2019). Both men and women experience a great deal of stress at this age, including pressure to provide for the educational demands of their children, retirement age anxiety, and hormonal imbalance. This is consistent with earlier research conducted by her scientists (Dinh *et al.*, 2014).

It was noted that there were more women than men in the group (53.7% against 46.3%). This observation was probably because women participants had higher levels of awareness, more

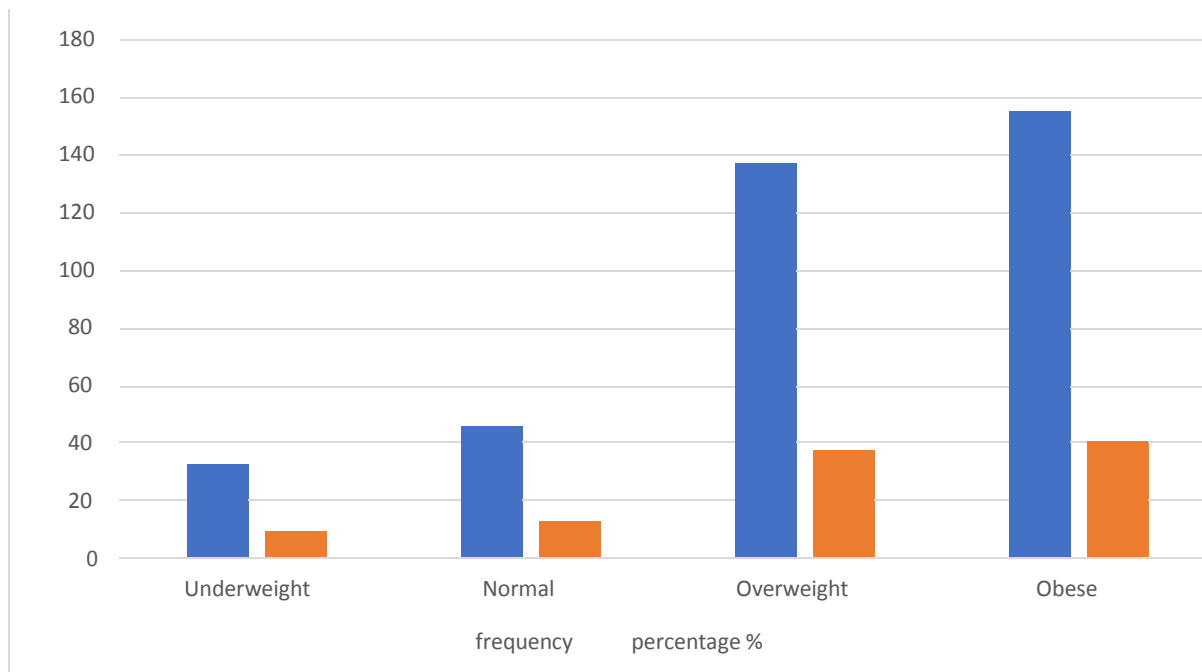


eager to submit to screening, and were more patient than men. The observation of high incidence amongst females was consistent with earlier research by Everett and Zajacova (2015) and Reckelhoff (2017). In this region of the world, it is culturally expected that men should oversee providing for the needs of the entire family, the observed high incidence of hypertension amongst females may be linked to diets heavy in fat and sugar.

Although men are more liable than women to develop hypertension at premenopausal years, hypertension is the principal basis of death in women (Wei *et al.*, 2017; Wenger *et al.*, 2018). However, the observation contrasts with Olooto *et al.* (2020) who observed a higher incidence in men than women.

The study also considered the individuals' level of education and observed a high prevalence of hypertension among people with secondary education. This finding contrasted that of a prior study by Ikeoluwapo *et al.* (2016), which found that primary school dropouts had the highest frequency of hypertension. Meanwhile, low education is linked to an increased chance of developing pre-hypertension, according to a related study by Zhang *et al.* (2018). This study revealed that low-income earners had the highest frequency of hypertension (49.3%). This finding agrees with earlier work by Bing *et al.* (2015), who found socioeconomic disparities among patients with some deadly chronic conditions like cancer and heart disease. The results of this study, however, disagree with that of the study by Choi *et al.* (2017) in terms of socioeconomics. A study on the relationship between the prevalence of hypertension and a few potentially modifiable characteristics like education, occupation, and income level showed that socioeconomic position has a great impact on the frequency and severity of hypertension (Setiawan *et al.*, 2017; Zhou *et al.*, 2018; Schultz *et al.*, 2018). The use of herbal mixtures by the participants as an alternative form of therapy was also investigated in this study. It was shown that 41.1% of the hypertensive participants consumed daily. This agrees with the findings of Daniel *et al.* (2013) amongst people living in urban slums. Because most of them are low-wage earners, this practice may reflect the high total cost of antihypertensive medications. According to Cuschieri *et al.* (2017), lack of access to suitable antihypertensive medications links low socioeconomic status with worse BP control and a higher risk of consequences. If there is no longer a barrier preventing access to proper hypertension therapy, this correlation may not exist.

Participants' regular multivitamin supplementation was considered in this study, 31.1%, 24.4%, and 8.5% take them daily, weekly, and occasionally, respectively. While 36% of the participants do not engage in multivitamin supplements at all. The amount of physical activity, including its frequency in terms of daily, weekly, occasional, and none, was also considered. The percentage of exercising participants, 14.1%, 30.7%, and 17% reported exercising daily, weekly, and rarely, compared to 38.2 percent who reported doing no physical activity at all.



**Figure 1** : BMI of participant

Figure 4.1 shows the classification of BMI of participants according to WHO and found that the obese class has the highest percentage (42%), followed by overweight (38%), normal weight, and underweight 12% and 8% respectively. This was in accordance with the study of Arshad *et al.*, (2019) who reported only 2% of their subjects were underweight, 11% were normal weight and 15% were overweight and obese 72% of the total investigated population had diabetes mellitus. This was in line with the study of another researcher (Bansal and Upadhyay 2018). However, this is in contrast with the findings of a previous study in Yemen that overweight and obesity accounted only for just 26.2% of their subjects with T2DM aged 20-65 (Al-sharafi and Gunaid 2014)

From the figure, it can be deduced that management of obesity can reduce hypertension by 50% in the participants. This also implied that hypertension among the participants is BMI induced. This is in agreement with the report of Shisana (2013) that the average BMI of the women in his study was in the obese category at baseline and increased at follow-up, with more than 50% of the participants classified as obese. High body weight and obesity, by measuring BMI, are the main causes of these disorders, hence, the continuous weight management. Research is actively being conducted on the relationship between hypertension and BMI class (Yang *et al.*, 2013).



**Table 3: Serum lipid profile among the participants at different time intervals**

Parameters	Fasting IQR	1HrPP IQR	2HrPP IQR	F	P
TC (mg/dL)	197.0 (166-231)	183.0 (162-224)	175.0 (158-226)	32.038	0.001**
TG (mg/dL)	82.0 (70-96)	89.0 (71-118)	92.0 (67-143)	18.391	0.001**
HDL-c (mg/dL)	54.0 (43-64)	54.0 (44-66)	53.0 (44-66)	2.306	0.316
LDL-c (mg/dL)	113.4 (84-134)	109.7 (77-145)	103.6 (68-139)	15.713	0.001**
VLDL (mg/dL)	17.0 (13-19)	17.8 (12-21)	17.6 (13-26)	6.829	0.033*

Values are expressed as mean and standard deviation (mean+/- SD), level of statistical significance was set at  $p < 0.05$ .

There have been a series of arguments on whether the analysis of cholesterol should be on the non-fasting or fasting samples. From this study, a reduction in TC, HDL, and LDL concentrations; and rise in TG and VLDL concentrations were observed among the participants (Fasting: 197.0, 82.0, 54.0, 113.4, 17.0; 1HrPP: 183.0 mg/dL, 89.0 mg/dL, 54.0 mg/dL, 109.7 mg/dL, and 17.8 mg/dL; 2HrPP: 175.0 mg/dL, 92.0 mg/dL, 53.0 mg/dL, 103.6 mg/dL, and 17.6 mg/dL)

However, when compared to fasting with postprandial result, a significant difference ( $p < 0.05$ ) was observed in TC and LDL-c while HDL-c, showed no significant difference ( $p > 0.05$ ). On the other hand, a substantial rise in triglycerides was observed ( $p < 0.05$ ). This finding is consistent with those made by researchers in other investigations (Nordestgaard *et al.*, 2016; Devaraj *et al.*, 2017; Ghildiyal *et al.*, 2020). In the non-fasting condition, the observed decrease in TC and LDL-c values is most likely the result of haemodilution after fluid intake with the meal. The detected rise in triglycerides is directly attributable to dietary fat ingestion. Given that water consumption is often permitted when fasting, these events may occur during the conventional fasting state that is frequently employed for lipid profiles.

According to Nordestgaard *et al.* (2017), the maximum mean difference between a nonfasting and a fasting lipid profile should be the following.: total cholesterol and LDL-c should be minus 8 mg/dL (-8 mg/dL), triglycerides +26 mg/dL, VLDL +8 mg/dL, while HDL-c remains unchanged 3-4 h after meal. Furthermore, a slight variation between fasting and nonfasting lipid profile values was seen in response to regular food intake in earlier research by White *et al.* (2015) and Farukhi and Mora (2017). Based on these findings by Devaraj *et al.*, (2017); Nordestgaard *et al.*, (2017), Hospitals in Copenhagen and other parts of Denmark adopted using non-fasting lipid profiles in regular testing with the caveat that a repeat on the fasting triglyceride should be considered if non-fasting value is more than 4 mmol/L (352 mg/dL).

In the explanations of their findings, Mente *et al.* (2017) and Lee *et al.* (2020) speculated that consuming more carbohydrates may be linked with lower levels of TC, HDL-c, LDL-c, and noticeably raised levels of triglycerides. This finding is consistent with the outcomes of this study's comparison of lipid profile values from the fasted and non-fasted states. However, Liu



*et al.* (2021) showed no appreciable distinction between the cholesterol levels in people who were fasting and those who were not.

The DASH diet, when followed, decreased total cholesterol, LDL, and HDL-c while having no negative effects on triacylglycerol. It also caused a decrease in HDL-c. The low HDL-c levels seen in part of the study group could potentially be brought on by this. This study's findings of increased triglycerides and decreased HDL-c may be related to increased carbohydrate intake at the time of sample collection. Another possible explanation for this is that hypertension patients are typically advised to avoid fatty meals, which has led to a shift toward foods high in protein and carbohydrates instead of lipids, which increases the risk of cardiovascular disease. Triglyceride results were consistent with all earlier research in which non-fasting state readings were considerably higher. Consuming edible vegetable oils may have a variety of effects on BP and serum lipid profiles, according to several research (Lai *et al.*, 2014; Petropoulos *et al.*, 2017; Gholamian-Dehkordi *et al.*, 2017). Since carbohydrates are the main meal in this region of the world, the observed changes in lipid values between fasting and non-fasting samples could not be entirely unconnected to the subjects' regular eating habits. The results of this study disagree with those of Mandle *et al.*, (2019), which found that measurements of the lipid profile parameters in the non-fasting state were rarely useful for estimating cardiovascular risk or for other clinical purposes because all the observed parameters increased significantly in non-fasting state compared to fasting state.

**Table 5: Pairwise analysis of parameters among the control and hypertensive participants**

	TC	TG	HDL-c	LDL-c	VLDL
Fasting vs 1hr	0.001**	0.001**	0.110	0.050*	0.001**
Fasting vs 2hrs	0.001**	0.002*	0.087	0.001*	0.001**
1hr vs 2hrs	1.000	0.241	1.000	1.000	0.186

P < 0.05 was considered statistically significant

The pairwise values showed a substantial difference between fasting and 1hr and fasting and 2-HrPP for all the lipid markers, except in HDL-c where no significant difference was observed throughout the time interval. However, there was no significant difference between 1hrpp and 2hrpp in all the lipid markers.

## CONCLUSION

The acceptable changes in lipid parameter levels from fasting status were decreased by 0.2 mmol/L (-7.73 mg/dL) for serum total cholesterol, decreased by 0.2 mmol/L (-7.73 mg/dL) for serum LDL, decreased by 0.1 mmol/L (-3.87 mg/dL) for serum HDL, and increased by 0.3 mmol/L (26.57 mg/dL) for serum triglycerides.

The findings in this study showed that the postprandial lipid profile result was within the permissible minimal change when compared with the fasting result. The

acceptable changes in lipid parameter levels from fasting status were decreased by 0.2 mmol/L (-7.73 mg/dL) for serum total cholesterol, decreased by 0.2 mmol/L (-7.73 mg/dL) for serum LDL, decreased by 0.1 mmol/L (-3.87 mg/dL) for serum HDL, and increased by 0.3 mmol/L (26.57 mg/dL) for serum triglycerides.

According to the findings of this study, socioeconomic level and educational attainment were significant risk factors for the development of hypertension.

The findings of this investigation showed that lipid profile analysis can be performed



on samples that had not been fasted because the changes were within the permissible range between fasting and postprandial (upper and lower levels of normal). In addition, since the conventional standard lipid profile comprises all the parameters (TC, TG, HDL-c, LDL-c, and VLDL) none can be measured in isolation.

Therefore, it can be concluded based on the result of the current study, that lipid profiles can be analysed using non-fasting samples as well, however, triglycerides can be repeated with fasting sample, where the non-fasting value exceeds 4 mmol/L (352 mg/dL).

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