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# Evaluation of Lipid-Lowering Effects of Atorvastatin on Obese Patients Attending A Tertiary Healthcare Facility.

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## ABSTRACT

Introduction: Hyperlipidemia is a major cause of atherosclerosis and atherosclerosis-associated conditions, such as coronary heart disease (CHD), ischemic cerebrovascular disease (CVD), and peripheral vascular disease.
This is a progressive observational study which involved five obese participants on atorvastatin 10 mg daily and five obese participants who were not on atorvastatin who served as control group. A 3 ml blood sample was collected from the participants at recruitment and the participants were followed up after 30 days with another blood collection. The participants on atorvastatin had been adherent to drug before blood collection. The control participants were not taking any drug which lower serum cholesterol. The obtained blood samples were analysed in the laboratory for lipid profile parameters such as total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) by using TC, HDL and TG kits made by Randox. The results showed that the follow-up participants on atorvastatin had lipid profile of TC, TG, HDL, LDL and VLDL as 5.14±0.87 mmol/l, 1.72±0.32 mmol/l, 1.12±0.23 mmol/l, 3.24±0.79 mmol/l and 0.77±0.12 mmol/l respectively. The results for the follow-up control participants showed that TC, TG, HDL, LDL and VLDL were 4.76±0.76 mmol/l, 1.52±0.55 mmol/l, 1.15±0.15 mmol/l, 2.92±0.75 mmol/l and 0.68±0.25 mmol/l respectively. Comparison of the lipid profile of participants on atorvastatin and the control group showed that there was no significant variation in LDL (p= 0.435) at first phase and LDL (p= 0.523) at second phase respectively. There was no significant variation in reduction of LDL between the participants on atorvastatin and the control participants.

**Keywords**: BMI, Hyperlipidemia, LDL, TC and obese

## INTRODUCTION

The WHO reported that globally, about 1.9 billion adults aged 18 years and more were overweight and about 600 million were obese. Most of the world's populations are situated in countries where overweight and obesity result in death of more people than underweight. Report also showed that about 41 million children below 5 years were overweight or obese in 2014. Obesity is now an emerging disorder in developing countries, especially in urban cities. The number of children who are overweight or obese almost doubled from 5.4 million in 1990 to 10.6 million in 2014 in Africa. Overweight and obesity are associated with more deaths worldwide than underweight (WHO, 2016).

Obesity and overweight are due to an energy imbalance between calories intake and expended calories. There is increased intake of high calorie diet and increase in physical inactivity due to the emerging sedentary nature of many work designs, changing modes of transportation, and upsurge urbanization. Both societal and environmental factors influenced modification of diets and physical activities of people (WHO, 2016).

females.

The most common way to diagnose obesity is by determining the body mass index (BMI). BMI is calculated from the height and weight of an individual and it provides an estimate of the body fat. BMI of 18.5-24.9 $kgm^{-2}$ is normal weight, 25.0-29.9 $kgm^{-2}$ is overweight, 30.0-39.9 $kgm^{-2}$ is obesity; and 40.0 $kgm^{-2}$ and beyond is extremely obesity (NHLBI, 2012). Another classification indicated that 30.0–34.9 $kgm^{-2}$ is obesity I, 35.0–39.9 $kgm^{-2}$ is obesity II, 40.0 $kgm^{-2}$ (and above) is obesity III (NHMRC, 2016).

The distribution of body fat is also recognized as a factor that influences cardiovascular diseases (CVD) risk. Measurement of waist circumference is perhaps the easiest and most practical indicator of central obesity and correlates well with CVD risk. Target waist circumference should be less than 102 cm in white Caucasian men, less than 88 cm in white Caucasian females; and in Asians less than 90 cm in men and less than 80 cm in

Obesity can be prevented by advising patients to observe the followings:

- Maintain healthy eating plan by balancing energy in with energy out
- Avoiding overeating.

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- There should be adequate exercise.
- Keep track of weight, BMI and waist circumference (NHLBI, 2012).

Raised BMI is an important risk factor for non-communicable diseases such as cardiovascular diseases like heart disease and stroke, which were the leading cause of death in 2012, diabetes; musculoskeletal disorders like osteoarthritis and some cancers like endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon (NHLBI, 2012). The risk for these non-communicable diseases increases, with increases in BMI. Childhood obesity is associated with a higher chance of obesity, premature death and disability in adulthood. But in addition to increased future risks, obese children experience breathing difficulties, increased risk of fractures, hypertension, and early markers of cardiovascular disease, insulin resistance and psychological effects (Pulgaron, 2013). Hyperlipidemia is a major cause of atherosclerosis and atherosclerosis-associated conditions, such as coronary heart disease (CHD), ischemic cerebrovascular disease, and peripheral vascular disease. Although the incidence of these atherosclerosis-related events has declined in the United States, these conditions still account for the majority of morbidity and mortality among middle-aged and older adults. The incidence and absolute number of annual events will likely increase over the next decade because of the epidemic of obesity and the aging of the U.S. population. Dyslipidemias, including hyperlipidemia (hypercholesterolemia) and low levels of high-density-lipoprotein cholesterol (HDL-C), are major causes of increased atherogenic risk; both genetic disorders and lifestyle such as sedentary behavior and diets high in calories, saturated fat, and cholesterol contribute to the dyslipidemias seen in developed countries around the world (Fernandez et al., 2010). Despite the efficacy of drug therapy, alterations in lifestyle have a far greater potential for reducing vascular disease risk and at a lower cost. Recognition that dyslipidemia is a risk factor has led to the development of drugs that reduce cholesterol levels. These drugs provide benefit in patients across the entire spectrum of cholesterol levels, primarily by reducing levels of low-density lipoprotein cholesterol (LDL-C). In well-controlled clinical trials, fatal and nonfatal CHD events and strokes were reduced by as much as 30 % to 40 % (Scandinavian Simvastatin Survival Study, 1994; Shepherd *et al.*, 1995; The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group, 1998; Heart Protection Study Collaborative Group, 2002; Heart Protection Study Collaborative Group, 2003; Law *et al.*, 2003). Cornerstone of management of dyslipidemia involves lifestyle changes such as diet control and exercise. Weight reduction can lower LDL cholesterol and triglyceride and raise HDL cholesterol level. Maximal improvement in dyslipidemia should be attempted with lifestyle intervention before consideration is given to lipid lowering drugs. The management approach is based on evaluation of the risk of CHD in an individual patient. An optimal LDL cholesterol goal of less than 70 mg/dL (1.8 mmol/liter) is the target (Abhimanyu and Vinaya, 2007). There are five main lipid lowering agents available for treating hyperlipidemeia namely statins, fibrates, bile acid binding agents, cholesterol absorption inhibitors and nicotinic acid and derivatives. Statins are currently the drugs of choice in the majority of patients with dyslipidaemia due to the overwhelming evidence that treatment with these agents reduces cardiovascular events. Emergence of statins which selectively inhibit 3-hydroxy-3-methylglutaryl-CoA reductase (HMG-CoA reductase) implies a significant advance in the treatment of dyslipidaemia. Their primary site of action is the inhibition of HMG-CoA reductase in the liver and the subsequent inhibition of the formation of mevalonic acid, the rate-limiting step in the biosynthesis of cholesterol. This results in a reduction in intracellular levels of cholesterol, an increase in expression of hepatic LDL receptor, and enhanced receptor-mediated catabolism and clearance of LDL-C from serum. Production of VLDL-C, the precursor of LDL-C, is also reduced. The overall effect is a reduction in TC, LDL-C, VLDL-C and triglycerides with an increase in HDL-C. The reduction in LDL-C occurs in a dose-dependent manner, with a lesser and dose-independent effect on VLDL-C and triglycerides (Wadhera et al., 2015). The efficacy of statins has been demonstrated in several landmark, randomized placebo-controlled trials. Statins are currently the lipid-lowering agents of choice in both primary and secondary prevention of CVD (Wadhera et al., 2015). Simvastatin is currently the preferred agent because of its relatively low cost, safety profile and evidence of efficacy. It is important to identify patients, who need treatment, ensure they receive an appropriate, effective dose of a statin and adhere to treatment. In spite of overwhelming evidence of benefit, effectiveness of satins is often compromised by poor adherence such that about 50 % of patients discontinuing treatment within 12 months and 75 % within 3 years of therapy. Patient factors that influence this are perception of risk, side effects of medication, expected treatment duration and socio-demographic factors (Moon and Bogle, 2006). This study aimed at evaluating lipid lowering effect of atorvastatin among obese patients in a tertiary healthcare facility.

## MATERIALS AND METHODS

**Materials**: The following materials were used, weighing scale, measuring tape, metre rule, 5 ml syringes, plain tubes, high density lipoprotein (HDL) kit, triglyceride (TG) kit, total cholesterol (TC) kit, cotton wool and methylated spirit.

Study design: A progressive observational study was used to evaluate lipid -lowering effect of prescribed atorvastatin among obese patients attending a tertiary healthcare facility.

Study location: The study was conducted among obese patients attending medical outpatient department clinic in University of Uyo Teaching Hospital.

**Study population**: The study involved obese patients from 18 years and above who have been diagnosed with hypercholesterolemia and have been receiving atorvastatin 10mg daily. This is the focused group. The control group consisted of obese patients who were not receiving any treatment for their obesity but attended the general outpatient department clinic for other purposes such as treatment of malaria and diarrhea. The blood samples of control group were collected before administration of drugs for their respective treatments.

**Sample size**: Purposive-convenience sampling was used to recruit 5 participants for the focused group and 16 participants in the control group of the study. The results of 5 participants each from the two groups were analysed for the study.

Inclusion criteria: The inclusion criteria involved both sexes, participants aged 18 years and above, were diagnosed with hypercholesterolemia and had started atorvastatin.

**Exclusion criteria**: Critically ill participants and those with the diseases such as HIV/AIDS, diabetes, peripheral vascular diseases, cerebrovascular diseases, congestive heart failure and arrhythmia were not allowed to participate in the study.

**Data collection**: A 3 ml blood was collected from the participant after recruitment and was analysed in the laboratory for the lipid profile parameters. The participants were followed up after 30 days with collection of another 3 ml blood. The blood samples were spun to obtain sera which were used for determining the amount of TC, TG, HDL, LDL and VLDL by using total cholesterol, triglyceride and HDL kits made by Randox® laboratory limited, United Kingdom.

**Laboratory Investigation**

Determination of total cholesterol was done by using pipette to add 1000 µl of cholesterol reagent into a tube known as blank tube without sample and the standard. Also pipette was used to add 10 µl of standard and 1000 µl of cholesterol reagent into another tube known as the standard tube. Pipette was also used to add 10 µl of sera and 1000 µl of cholesterol reagent into another tube known as test tube. Each tube was mixed and put in the Unispec 23D Spectrophotometer (UNISCOPE) made by Surgifriend Medicals, England. Each tube was mixed and incubated at 37 $^0$C for 5 minutes. The absorbance of standard and test tubes was read against reagent blank at wavelength of 500 nm.

Determination of triglyceride was done by starting with the reconstitution of lyophilized crystals with 15 ml of reagent which was mixed and allowed to stand for 30 mins. Then pipette was used to add 1000 µl of triglyceride into a tube known as blank tube without the standard and sera. Also, pipette was used to add 10 µl of the standard and 1000 µl of the triglyceride reagent into another tube known as standard tube. Pipette was also used to take 10 µl of the sample and 1000 µl of triglyceride reagent into another tube known as the test tube. Each tube was labeled accordingly, mixed and incubated at 37 $^0$C for 5 mins. The absorbance of standard and test tubes were read against the reagent blank at wavelength of 500 nm.

Determination of high density lipoprotein (HDL) was done by beginning with deproteinisation through preparation of a 1 in 4 dilution of the HDL reagent with distilled water such as 1 part of HDL in 4 parts of distilled water. A pipette was used to add 2 00µl of the standard into a tube without the sera, pipette was also used to add 500 µl of the diluted reagent into the same tube which was called the standard tube. A pipette was used to add 200 µl of the sera and 500 µl of the diluted reagent into another tube without the standard which was called the test tube. Both the test tube and the standard tube were covered, mixed and incubated at room temperature for 10 minutes. Thereafter, the tubes were centrifuged at 4000 rpm for 10 minutes. A pipette was used to add 1000 µl of the cholesterol reagent into a tube without both standard supernatant and sample supernatant, the tube was called the blank tube. A pipette was also used to add 100 µl of standard supernatant and 1000 µl of cholesterol reagent into another tube without the sample supernatant, the tube was called the standard tube. A pipette was used to add 100 µl of the sample supernatant and 1000 µl of the cholesterol reagent into another tube without the standard supernatant, the tube was called test. Each tube was mixed and incubated at 37 $^0$C for 5 minutes. The absorbance of standard tube and test tube were read against the reagent blank tube at a wavelength of 500 nm.

Determination of the very low density lipoprotein was done by dividing the value of triglycerides by 2.22 as follow:

$$VLDL = \frac{(triglyceride)}{2.22}$$

equation 1

Determination of low density lipoprotein was done by using Friedewald equation as follow:

$$LDL = TC - VLDL - HDL$$

equation 2

$$LDL = TC - \left(\frac{(TG)}{2.22}\right) - HDL$$

equation 3

**Data management and analysis**

Data obtained was stored in the computer with MS Office 2008. Data was analyzed using descriptive statistics such as frequency tables and percentages to describe the distribution of the participants with respect to the measured characteristics. Inter-group comparison of data was done in the first and second phases of blood collection while intra-group comparison was done for the second phase. Further analysis was performed on the data using paired-T test at $p \leq 0.05$ level of significance and other advance statistical methods in the Statistical Package for the Social Sciences (SPSS) software version 17.0 (SPSS Inc. Chicago, III, USA).

**RESULTS**

The results of the participants during the study showed that the mean age, BMI and waist circumferences of the focused group, that is participants on atorvastatin were 47.50±12.23 years, 31.54±6.13 kg/m$^2$ and 0.98 m respectively. The waist circumference of participants in the control group varies significantly with the participants in the focused group (Table 1). The baseline of lipid profile of participants on atorvastatin 10 mg daily, the focused group, included TC, TG, HDL, LDL and VLDL as 4.77±1.48 mmol/l, 1.04±0.28 mmol/l, 1.36±0.38 mmol/l, 3.71±1.1 mmol/l and 0.70±0.29 mmol/l respectively (Table 2). Three participants on atorvastatin had elevated TC, three participants had lower HDL, four participants had elevated LDL while two participants had elevated VLDL (Table 2). The baseline of lipid profile of the control group that were followed up were TC, TG, HDL, LDL and VLDL as 4.84±1.18 mmol/l, 2.11±1.17 mmol/l, 0.81±0.20 mmol/l, 3.07±0.96 mmol/l and 1.00±0.62 mmol/l respectively (Table 3).

The results showed that the follow-up participants on atorvastatin lipid profile of TC, TG, HDL, LDL and VLDL were 5.14±0.87 mmol/l, 1.72±0.32 mmol/l, 1.12±0.23 mmol/l, 3.24±0.79 mmol/l and 0.77±0.12 mmol/l respectively. Four participants had elevated TC, two participants had elevated TG and three participants had elevated VLDL (Table 4). The results for the follow-up control group showed that TC, TG, HDL, LDL and VLDL were 4.76±0.76 mmol/l, 1.52±0.55 mmol/l, 1.15±0.15 mmol/l, 2.92±0.75 mmol/l and 0.68±0.25 mmol/l (Table 5).

The results for comparison of the lipid profile of participants on atorvastatin, focused group and the control group showed that HDL (p= 0.04) and LDL (p= 0.435) in first phase and HDL (p= 0.815) and LDL (p= 0.523) in second phase respectively. HDL varies significantly between the two groups in the first phase (Table 6).

Both TC and TG were increased in the obese participants on atorvastatin, focused group while TC and TG were reduced in participants in the control group. LDL was reduced in both obese participants on atorvastatin, focused group and participants in the control group. HDL varied significantly between the focused group and the control group (Table 7).

Table 1: Demographic profile of participants

| Participants | Number of participants | Age (years) | BMI (Kgm$^{-2}$) | Waist circumference (m) |
|---|---|---|---|---|
| Focused group | 5 | 47.50±12.23 | 31.54±6.13 | 0.98±0.08 |
| Control group | 5 | 48.75±12.23 | 39.6±2.86 | 1.15±0.06 |
| *p-value | | 0.464 | 0.219 | 0.022 |

*$p \leq 0.05$ was considered significant

Table 2: Baseline lipid profile of the focused group

| Code | BMI (Kgm$^{-2}$) | TC (mmol/l) | TG (mmol/l) | HDL (mmol/l) | LDL (mmol/l) | VLDL (mmol/l) | Dose (mg) |
|---|---|---|---|---|---|---|---|
| A01 | 26.1 | 4.3 | 1.5 | 1.6 | 2.02 | 0.68 | 10 |
| A02 | 26.3 | 5.26 | 0.87 | 1.41 | 3.47 | 0.89 | 10 |
| A03 | 31.3 | 6.00 | 0.93 | 0.80 | 4.80 | 0.40 | 10 |
| A04 | 32.9 | 5.90 | 1.10 | 1.80 | 3.65 | 0.45 | 10 |
| A05 | 41.1 | 2.4 | 0.80 | 1.22 | 4.61 | 1.10 | 10 |
| Mean ± SD | 31.54±6.13 | 4.77±1.48 | 1.04±0.28 | 1.36±0.38 | 3.71±1.10 | 0.70±0.29 | |

| | | | | | | |
|---|---|---|---|---|---|---|
| Normal range | 18.5-24.9 | <5.17 | <1.71 | ≥1.55 | <3.36 | <0.77 |

Table 3: Baseline lipid profile of control group

| Code | BMI (kgm-2) | TC (mmol/l) | TG (mmol/l) | HDL (mmol/l) | LDL (mmol/l) | VLDL (mmol/l) |
|---|---|---|---|---|---|---|
| C02 | 26 | 4.00 | 1.36 | 0.93 | 2.46 | 0.61 |
| C03 | 32.6 | 4.02 | 1.87 | 1.04 | 2.14 | 0.84 |
| C06 | 25 | 4.54 | 1.82 | 0.71 | 3.01 | 0.82 |
| C07 | 35.8 | 4.87 | 1.64 | 1.14 | 2.99 | 0.74 |
| C08 | 39.1 | 5.79 | 0.88 | 1.13 | 4.26 | 0.40 |
| C09 | 43.6 | 3.03 | 1.26 | 0.85 | 1.61 | 0.57 |
| C10 | 31.2 | 3.22 | 1.11 | 0.98 | 1.74 | 0.50 |
| C12 | 38.3 | 5.13 | 2.45 | 0.68 | 3.35 | 1.10 |
| C13 | 35.4 | 4.19 | 1.72 | 0.94 | 2.47 | 0.78 |
| C14 | 38 | 4.99 | 0.93 | 1.04 | 3.53 | 0.42 |
| C15 | 43 | 6.31 | 1.80 | 0.99 | 4.51 | 0.81 |
| C16 | 36.9 | 5.72 | 1.68 | 0.80 | 4.17 | 0.75 |
| C17 | 36 | 4.35 | 2.10 | 0.58 | 2.82 | 0.95 |
| C18 | 41 | 5.91 | 3.89 | 0.85 | 3.31 | 1.75 |
| C19 | 31.1 | 2.94 | 0.10 | 0.75 | 1.74 | 0.45 |
| C20 | 32 | 3.88 | 1.37 | 1.09 | 2.17 | 0.62 |
| Mean* | 35.31±5.41 | 4.55±1.04 | 1.62±0.81 | 0.90±0.16 | 2.89±0.91 | 0.75±0.32 |
| Mean** | 39.6±2.86 | 4.84±1.18 | 2.11±1.17 | 0.81±0.20 | 3.07±0.96 | 1.00±0.62 |
| Normal range | 18.5-24.9 | <5.17 | <1.71 | ≥1.55 | <3.36 | <0.77 |

*Baseline data of control participants at recruitment phase. **Baseline data of control participants that were followed up

Table 4: Follow-up lipid profile of the focused group

| Code | BMI ($kgm^{-2}$) | TC (mmol/l) | TG (mmol/l) | HDL (mmol/l) | LDL (mmol/l) | VLDL (mmol/l) | Dose (mg) |
|---|---|---|---|---|---|---|---|
| A01 | 26.1 | 3.65 | 2.04 | 1.04 | 2.02 | 0.59 | 10 |
| A02 | 26.3 | 5.29 | 1.32 | 1.12 | 3.35 | 0.82 | 10 |
| A03 | 31.3 | 5.95 | 2.06 | 0.79 | 4.23 | 0.93 | 10 |
| A04 | 32.9 | 5.36 | 1.52 | 1.43 | 3.15 | 0.78 | 10 |
| A05 | 41.1 | 5.47 | 1.70 | 1.22 | 3.48 | 0.77 | 10 |
| Mean | 31.54±6.13 | 5.14±0.87 | 1.72±0.32 | 1.12±0.23 | 3.24±0.79 | 0.77±0.12 | |
| Normal range | 18.5-24.9 | <5.17 | <1.71 | ≥1.55 | <3.36 | <0.77 | |

Table 5: Follow-up lipid profile of the control group

| Code | BMI ($Kgm^{-2}$) | TC (mmol/l) | TG (mmol/l) | HDL (mmol/l) | LDL (mmol/l) | VLDL (mmol/l) |
|---|---|---|---|---|---|---|
| C03* | 32.6 | 4.66 | 1.10 | 1.13 | 3.03 | 0.5 |
| C08 | 39.1 | 4.66 | 0.75 | 1.35 | 2.97 | 0.34 |
| C09 | 43.6 | 3.48 | 1.28 | 1.29 | 1.61 | 0.58 |
| C12 | 38.3 | 5.15 | 1.66 | 1.07 | 3.33 | 0.75 |
| C17 | 36.2 | 5.39 | 2.26 | 0.98 | 3.39 | 1.02 |
| C18 | 41.3 | 5.15 | 1.67 | 1.09 | 3.31 | 0.75 |
| Mean | 39.7±2.84 | 4.76±0.76 | 1.52±0.55 | 1.15±0.15 | 2.92±0.75 | 0.68±0.25 |
| Normal range | 18.5-24.9 | <5.17 | <1.71 | ≥1.55 | <3.36 | <0.77 |

*Data was not used for the statistics

Table 6: Comparison of lipid profile of the two groups

| S/N | Phase | Lipid profile | Focused group | Control group | *p-value |
|---|---|---|---|---|---|
| 1 | First | BMI | 31.54±6.13 | 39.60±2.86 | 0.064 |
| 2 | | TC | 4.77±1.48 | 4.84±1.18 | 0.951 |
| 3 | | TG | 1.04±0.28 | 2.11±1.17 | 0.156 |
| 4 | | HDL | 1.36±0.38 | 0.81±0.20 | 0.040 |
| 5 | | LDL | 3.71±1.10 | 3.07±0.96 | 0.435 |
| 6 | | VLDL | 0.70±0.29 | 1.00±0.62 | 0.301 |
| 1 | Second | BMI | 31.54±6.13 | 39.70±2.84 | 0.059 |
| 2 | | TC | 5.14±0.87 | 4.76±0.76 | 0.462 |
| 3 | | TG | 1.72±0.32 | 1.52±0.55 | 0.569 |
| 4 | | HDL | 1.12±0.23 | 1.15±0.15 | 0.815 |
| 5 | | LDL | 3.24±0.79 | 2.92±0.75 | 0.523 |
| 6 | | VLDL | 0.77±0.12 | 0.68±0.25 | 0.384 |

*p≤ 0.05 was considered significant

Table 7: Rate of reduction of lipid profile in second phase

| S/N | LIPID PROFILE | FOCUSED GROUP | CONTROL GROUP | *p-value |
|---|---|---|---|---|
| 1 | TC | (-)0.37±1.53 | 0.07±0.88 | 0.648 |
| 2 | TG | (-)0.68±0.64 | 0.59±0.98 | 0.172 |
| 3 | HDL | 0.24±0.24 | (-)0.33±0.1 | 0.006 |
| 4 | LDL | 0.47±0.31 | 0.14±0.68 | 0.594 |
| 5 | VLDL | (-)0.07±0.34 | 0.32±0.43 | 0.144 |

(-): increase, *p≤ 0.05 was considered significant

**DISCUSSION**

The study evaluated the extent of reduction of lipid by atorvastatin in obese patients and compared it with the reduction of lipid in other obese patients who were not receiving atorvastatin or other agents which could reduce serum lipid. The study observed that there were no significant difference in the age and BMI of the participants on atorvastatin and the control participants. The control participants were classified as type II obesity while the participants on atorvastatin were classified as type I obesity (Mardolkar M *et al*., 2017). There was a significant variation in the waist circumference of the two groups. The control participants with significant increase in waist circumference suggested that they were at risk of cardiovascular diseases and mortality except they were treated with antilipid agents. Seidell (2010) had earlier concluded in his study that waist circumference alone could replace waist-hip ratio and BMI as a single risk factor for all cause of mortality. Gelber *et al*., (2008) indicated that the magnitude of associations of BMI, waist circumference, waist-hip ratio and waist height ratio with cardiovascular diseases risk were similar. Previous study had indicated that a high waist circumference was associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and CVD in patients with a BMI of 25 - 34.9 kg/m$^2$ (Chan *et al*., 1994). The control participants did not receive treatment for the hypercholesterolemia, hence they were used as the control for the study.

It was observed that the TC and TG on individual basis increased among participants on atorvastatin after a month of follow-up suggesting that the use of atorvastatin alone to control hypercholesterolemia without other measures such as diet control, exercise will not be sufficient to control hypercholesterolemia. This observation was in contrast to the observation in previous study which indicated that atorvastatin had efficacy to reduce both TC and LDL (Adam, 2015). However, only two of the participants on atorvastatin had elevated LDL during the follow-up period probably suggesting a lowering effect on LDL. Those whose LDL were not controlled would probably be due to inability to modify their lifestyle on diet and exercise. A previous study suggested that future global threat of coronary heart diseases' risk factor such as hypercholesterolemia was modifiable through lifestyle (Mannu *et al*., 2013).

Though the individual lipid profile parameters of some participants were higher than the limit, it was observed that the group mean of lipid profile parameters such as TC, LDL and VLDL of participants on atorvastatin were under control at the time of follow-up while TG was slightly elevated. This simply suggested that lifestyle modification is very important to control hypercholesterolemia in addition to the use of statin. Previous study had concluded that atorvastatin reduced LDL and TG (Stein *et al*., 1998).

The group mean for lipid profile parameters such as TC, TG, LDL and VLDL in the control group were under control at the time of follow-up. Lifestyle modification such as diet control and increased physical activity were major determinants for reduction of LDL without use of statin. In health conditions such as hypertension, dyslipidaemia and diabetes mellitus, previous study had shown that nutritionally balanced diets which meet recommendations of international health organizations would improve risk factors for cardiovascular diseases such as blood pressure, lipid metabolism, carbohydrate metabolism and BMI (McCarron *et al*., 1997).

It was observed that HDL parameter was lower than the limit in both the focused group and the control group suggesting that atorvastatin might not be responsible for lowering HDL in this population. Previous study affirmed that atorvastatin did not significantly affect the variability of HDL and TG measurements (Adams *et al*., 2015).

The lipid profile parameter HDL was significantly higher in participants on atorvastatin than the control participants at the time of recruitment. Previous study concluded that small doses of atorvastatin increases HDL while higher doses cause the effect to diminish (Adams *et al*., 2015).

During follow-up, HDL and LDL were the only lipid profile parameters that were lowered in participants on atorvastatin while TC, TG, LDL and VLDL were reduced in the control group suggesting that non-pharmacological approach such as lifestyle modification should be used first to control hypercholesterolemia before use of statin. A study conducted on the effect of lifestyle modification concluded that target cholesterol levels according to various guidelines could be achieved by lifestyle modification such as diet, weight reduction and increased physical activity designed to reach those goals specified (Mannu *et al*., 2013). A continued lowering of HDL values in the focused group would predispose the obese participants on atorvastatin to increased atherogenic risk (Fernandez *et al*., 2010).

Conclusion: This study revealed that there was no significant variation in reduction of LDL of participants on atorvastatin and the control participants.

Recommendation: Pharmacist intervention through pharmaceutical care plan is hereby recommended for patients on atorvastatin to achieve effective control of hypercholesterolemia.

Conflict of interest: There was none among the authors.

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