



COMPARATIVE ANALYSIS OF THE EFFECTS OF ROSUVASTATIN AND ATORVASTATIN ON THE SERUM LIPID PROFILE IN DIABETIC HEART PATIENTS

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Abstract

A condition of lipoprotein metabolism, hyperlipidaemia encompasses many disorders, including hypertriglyceridemia and hypercholesterolaemia. The World Health Organisation (WHO) recently said that by 2020, Indians will account for 60% of cardiovascular cases. The care of vascular disorders now includes the use of statins to treat hyperlipidaemia. The last five years have seen a significant expansion in statin indications due to the release of numerous multicenter prospective trials. The Indian market offers a variety of statins, including rosuvastatin, atorvastatin, simvastatin, pravastatin, pitavastatin, fluvastatin, and cerivastatin. Statins have a hypolipidemic impact because they inhibit hydroxymethylglutaryl-CoA reductase (HMG-CoA), and they lower LDL-C because they increase LDL receptor activation. This study's goal is to assess andThe World Health Organisation (WHO) recently said that by 2020, Indians will account for 60% of cardiovascular cases. The care of vascular disorders now includes the use of statins to treat hyperlipidaemia.

Introduction

The first-line treatment for reducing cholesterol is statins (1). The most common prescription medication nowadays is a statin (2). The fixed-dose regimens of more intense and less intensive statin therapy were compared in the majority of the trials (2,3).

Higher doses of statins were found to reduce C-reactive protein (CRP) more than lower doses in people with stable coronary disease (2). However, a number of studies indicate that the safety and effectiveness of different statins in hyperlipidaemia vary significantly (3).

Medical patients find it challenging (4). The most prevalent cause of blood vessel illnesses is dyslipidaemia, which has been increasing globally, leading to an increase in cardiovascular disease-related morbidity and mortality. Disturbances in lipid parameters such as total cholesterol, LDL-C, VLDL, TGs, and HDL-C lead to dyslipidaemia (5,6). Along with a number of other cardiovascular risk factors, including high blood pressure (BP), abdominal obesity, and insulin intolerance, dyslipidaemia is a component of metabolic syndrome, which raises the risk of atherosclerotic cardiovascular disease (7).

Patients with type 2 diabetes mellitus are more likely to have combined or mixed hyperlipidaemia (CHL), a lipid disorder marked by elevated triglycerides (TGs), decreased high-density lipoprotein cholesterol (HDL-C), and increased low-density lipoprotein cholesterol (LDL-C) (8,9).

The purpose of the National Cholesterol Education Program's Adult Treatment Panel III (NCEP-ATP III) is to treat patients with dyslipidaemia, which can be accomplished through the use of lipid-lowering medications, particularly statins (10). This condition is being treated using a variety of lipid-lowering medications, such as statins, fenofibrate, niacin, ezetimibe, bile sequestrants, etc. (11).

Numerous studies have been conducted on these medicines, but only a small number have been conducted among the people of North India, particularly in the Majhar region of Punjab, due to the fact that their socioeconomic background and level of life differ significantly from those of Western nations (12).

Hydroxymethylglutaryl-CoA reductase (HMG-CoA) inhibition is the cause of statins' hypolipidemic impact, whereas increased LDL receptor activity is the cause of the drop in LDL-C (13,14). Statin outcome trials have definitively shown that these medications lower LDL-C levels, which significantly lowers cardiovascular events in a large number of high-risk patients [15, 16] When compared to atorvastatin, simvastatin, or pravastatin, rosuvastatin has been thought to be more effective at lowering LDL-C levels (17). Additionally, "pleiotropic" benefits of statins, including vasodilatation, antioxidant, plaque stabilisation, antithrombotic, and anti-inflammatory actions, have been documented (18). Different parts of lipoprotein metabolism are impacted by fibrates and statins. According to recent American Diabetes Association research, it is therefore challenging to alter the lipid profile of patients with mixed hyperlipidaemia using statin or fibrate monotherapy (19).

Patients with mixed hyperlipidaemia (CHL) respond better to combined statin and fibrate therapy (20,21,22,23).

Aims and Objectives

Our study's objectives and goals were as follows:

to evaluate how well equivalent dosages of atorvastatin and rosuvastatin reduce LDL-C levels.

Compare the safety and effectiveness of atorvastatin and rosuvastatin in lowering cardiovascular events in people with high cholesterol and coronary artery disease.

Material and Methods

The current study was an open-label, parallel group, randomised investigation carried out at PIMS Udaipur, India. Following clinical and baseline studies, 100 patients with hyperlipidaemia who were taking 20 mg tablets of atorvastatin and 20 mg tablets of rosuvastatin were chosen for the study. Following the fifth week of statin medication, the patients' lipid profiles were reassessed.

Nature of the study

Open label, randomize, parallel group, comparative, prospective clinical Study.

Source of patient

Patients who visited the outpatient department (O.P.D.) of medicine were included in this study.

Research population

One hundred participants with combination hyperlipidaemia were screened for the study and divided into two groups of fifty at random.

Inclusion criteria

The trial will cover both male and female patients aged 35 to 85 who have triglycerides (TG) greater than 200 mg/dL and low density lipoprotein cholesterol (LDL-C) greater than 100 mg/dl. Every patient in the study had coronary artery disease, diabetes mellitus, obesity, and hypertension.

Exclusion criteria

Patients with Renal and hepatic failure, Pregnancy and lactation, Hypothyroidism, Malignancy, Myopathy, Patients undergoing bypass surgery and those with concurrent medications like warfarin, verapamil,

Methodology

Using a randomisation chart, the 100 patients who were enrolled in the trial were divided into two groups of fifty (n = 50) each. Prior to assigning the treatment, baseline measurements of plasma lipid levels, such as TC, TG, HDL, LDL, and VLDL, were obtained for both groups.

Next, 20 mg of Tab. Atorvastatin was given to Group I, and 20 mg of Tab. Rosuvastatin was given to Group II. For five weeks, one tablet was given to each group once a day at night. After five weeks, the patients were evaluated, and a serum lipid profile was completed.

Biochemical Examination

Collected 5 ml blood in plain vial from subject and the serum is separated. Following investigations were carried out:

Lipid Profile Determination of serum profile:

Estimation of Serum Total Cholesterol: by kit reagents (supplied by ERBA Diagnostics) on Semi autoanalyzer. **Estimation of HDL Cholesterol:** Estimation of serum HDL cholesterol will be carried out on semi automatic analyzer

Method: Burstein method (Burstein et al 1974).

Estimation of Triglycerides: The estimation of serum Triglycerides was carried out on semi automatic analyzer. **Method:** Modified Wako, McGowan and Fossati method. (Wako et al 1983).

Estimation of Low Density Lipoprotein Cholesterol: Serum LDL is calculated by Friedwald's equation $LDL\ cholesterol = Total\ Cholesterol - (HDL\ Cholesterol + VLDL\ Cholesterol)$

LDL was estimated by direct method when TG values were $> 400\ mg/dl$. **Estimation of Very Low – Density Lipoprotein Cholesterol.**

VLDL Cholesterol is calculated by Friedwald's equation

A total serum cholesterol level of $>200\ mg/dl$, an LDL cholesterol level of $>130\ mg/dl$, and a serum triglyceride level of $<350\ mg/dl$ had to be measured within the first 24 hours following the onset of acute coronary syndrome, or up to six months earlier if no sample had been taken during that time.

The patients were divided into two groups for the study: 50 people in Group A took 20 mg of ATORVASTATIN, and 50 people in Group B took 20 mg of ROSUVASTATIN. Both groups were monitored after five weeks.

Results & Discussion

The goal of the current study is to produce the best and most efficient outcomes possible. To demonstrate the important effects of therapies, the five lipid parameters are statistically assessed.

Table 1 provides an explanation of the findings. The mean age of patients receiving rosuvastatin is 59.76, while the mean age of subjects receiving atorvastatin is 60.42. The mean age of the treatment groups does not significantly differ. ($p>0.05$). It indicates that patients in both treatment groups are of the same age.

Both treatment groups have an equal gender ratio, and there is no discernible difference in the genders getting the treatment. ($p>0.05$)

The two treatment groups' mean glucose levels do not differ much. ($p>0.05$). The rosuvastatin group's mean glucose level is 275.3, while the atorvastatin group's is 258.84. The mean serum cholesterol levels in the two treatment groups do not differ significantly ($p>0.05$). The rosuvastatin group's mean serum cholesterol level is 247.68, while the atorvastatin group's is 258.84. The mean

HDL cholesterol levels in the two treatment groups do not differ significantly ($p>0.05$). Atorvastatin and rosuvastatin groups have mean HDL cholesterol levels of 42.7 and 44.12, respectively. The two treatment groups' mean LDL cholesterol levels do not differ much. ($p>0.05$). The rosuvastatin group's mean LDL cholesterol level is 146.57, while the atorvastatin group's is 158.74. The mean triglyceride levels in the two treatment groups do not differ significantly. ($p>0.05$). The rosuvastatin group's mean triglyceride level is 284.94, while the atorvastatin group's is 287. The two treatment groups' mean VLDL levels do not differ significantly. ($p>0.05$). Atorvastatin and rosuvastatin groups have mean VLDL levels of 57.4 and 56.98, respectively. Based on the Serum Lipid Profile Distribution Following therapy in respect to groups The mean serum cholesterol levels of the two treatment groups varied significantly ($p<0.05$). The rosuvastatin group's mean serum cholesterol is 176.5, while the atorvastatin group's is 193.08. Additionally, after therapy, the mean serum cholesterol of the rosuvastatin 20 mg group was lower than that of the atorvastatin group. The mean HDL cholesterol levels in the two treatment groups varied significantly ($p<0.05$). Atorvastatin and rosuvastatin groups have mean HDL cholesterol levels of 44.42 and 46.74, respectively. Additionally, after therapy, the mean HDL cholesterol was lower in the rosuvastatin 20 mg group compared to the atorvastatin group. The mean LDL cholesterol levels in the two treatment groups varied significantly ($p<0.05$).

The rosuvastatin group's mean LDL cholesterol is 98.97, while the atorvastatin group's is 115.77. Additionally, after treatment, the mean LDL cholesterol was lower in the rosuvastatin 20 mg group compared to the atorvastatin group. The mean triglyceride levels in the two treatment groups differ significantly. ($p<0.05$). The rosuvastatin group's mean triglyceride level is 153.92, while the atorvastatin group's is 164.44. Additionally, after treatment, the mean rosuvastatin 20 mg group had lower triglycerides than the atorvastatin group. The mean VLDL levels in the two treatment groups differ significantly. ($p<0.05$). The rosuvastatin group's mean VLDL level is 30.78, while the atorvastatin group's is 32.88. Additionally, after therapy, the mean rosuvastatin 20 mg group had lower VLDL than the atorvastatin group.

There is no discernible difference in the mean serum cholesterol level between the two genders in the distribution of serum lipid profiles prior to treatment with atorvastatin 20 mg ($p>0.05$). Male and female mean serum cholesterol levels are 255.85 and 262.63, respectively. Both genders' mean HDL cholesterol levels varied significantly ($p<0.05$). Male HDL cholesterol is 41.17, while female HDL cholesterol is 44.63. Additionally, prior to therapy, men had lower mean HDL cholesterol than women. The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 156.88 and 161.1, respectively.

The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 288.96 and 284.5, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 57.79 and 56.9, respectively.

Based on the Serum Lipid Profile Distribution Following therapy with 20 mg of atorvastatin based on gender The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 190.92 and 195.81, respectively.

Both genders' mean HDL cholesterol levels varied significantly ($p<0.05$). Male HDL cholesterol is 42.96, while female HDL cholesterol is 46.27. Additionally, prior to therapy, men had lower mean HDL cholesterol than women. The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 115.12 and 116.6, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 164.21 and 164.72, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p>0.05$). Male and female mean serum cholesterol levels are 32.84 and 32.94, respectively.

The distribution of the serum lipid profile before and after treatment with 20 mg of atorvastatin reveals a significant difference in the mean serum cholesterol level before and after therapy ($p<0.05$). and the atorvastatin group's mean serum cholesterol level decreased following treatment.

The mean HDL cholesterol level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean HDL cholesterol level is higher following treatment. The mean LDL cholesterol level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean LDL cholesterol level is lower following treatment.

The mean triglyceride level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean triglyceride level is lower following treatment. The mean VLDL level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean VLDL level is lower following treatment. There is no discernible difference in the mean serum cholesterol level between the two genders in the distribution of serum lipid profiles prior to treatment with rosuvastatin ($p > 0.05$). Male and female mean serum cholesterol levels are 244.83 and 252.31, respectively. The mean HDL cholesterol level does not significantly differ between the sexes ($p > 0.05$). Male HDL cholesterol is 44.32, while female HDL cholesterol is 43.78. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 143.54 and 151.5, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 284.83 and 285.1, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 56.96 and 57.02, respectively.

Serum lipid profile distribution Following rosuvastatin medication, there is no discernible difference in the mean serum cholesterol levels between the two genders ($p > 0.05$). Male and female mean serum cholesterol levels are 177.96 and 174.1, respectively. The mean HDL cholesterol level does not significantly differ between the sexes ($p > 0.05$). Male HDL cholesterol is 46.93, while female HDL cholesterol is 46.42. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 99.5 and 98.11, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 157.64 and 147.84, respectively. The mean serum cholesterol levels of the two genders do not differ significantly ($p > 0.05$). Male and female mean serum cholesterol levels are 31.52 and 29.56, respectively.

The distribution of the serum lipid profile before and after rosuvastatin treatment reveals a significant difference in the mean serum cholesterol level before and after treatment ($p < 0.05$). and the atorvastatin group's mean serum cholesterol level decreased following treatment. The mean HDL cholesterol level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean HDL cholesterol level decreases following treatment.

The mean LDL cholesterol level before and after therapy differs significantly ($p < 0.05$), and the atorvastatin group's mean LDL cholesterol level is lower after treatment. The mean triglyceride level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean triglyceride level is lower following treatment. The mean VLDL level before and after treatment differs significantly ($p < 0.05$). and the atorvastatin group's mean VLDL level is lower following treatment.

There is no discernible difference between the two treatment groups' percentage changes in serum cholesterol levels before and after, according to biomedical parameters ($p > 0.05$). while the mean changes for the rosuvastatin group are 28.29 and the atorvastatin group is 24.78. The percentage changes in HDL cholesterol levels before and after were observed to differ significantly between the two treatment groups ($p < 0.05$). and the mean changes are -6.11 for the rosuvastatin group and -4.09 for the atorvastatin group. Additionally, the atorvastatin group has a higher mean HDL cholesterol level. The percentage changes in LDL cholesterol levels (before to after) in both treatment groups did not differ significantly ($p > 0.05$). and the mean changes in the rosuvastatin group are 31.2 and in the atorvastatin group are 25.12. The percentage changes in triglyceride levels (before to after) in both treatment groups did not differ significantly ($p > 0.05$). and the mean changes are 45.5 in the rosuvastatin group and 42.62 in the atorvastatin group. The percentage changes in VLDL levels

(before to after) in both treatment groups did not differ significantly ($p>0.05$). and the mean changes are 32.45 for the rosuvastatin group and 27.69 for the atorvastatin group.

There is a significant difference in the mean serum cholesterol/HDLC level between the two treatment groups ($p<0.05$) based on the distribution of serum cholesterol/HDLC prior to treatment. The atorvastatin group's mean serum cholesterol/HDLC level is 6.16, whereas the rosuvastatin group's is 5.66. Additionally, prior to treatment, the rosuvastatin 20 mg group had lower serum cholesterol and HDLC than the atorvastatin group.

SERUM CHOLESTEROL/HDLC Distribution Following therapy, both treatment groups' mean blood cholesterol and HDLC levels differ significantly ($p<0.05$). The atorvastatin group's mean serum cholesterol/HDLC level is 4.40, while the rosuvastatin group's is 3.81. Additionally, after treatment, the rosuvastatin 20 mg group had lower serum cholesterol and HDLC than the atorvastatin group.

The distribution of serum cholesterol and HDLC levels before and after treatment with 20 mg of atorvastatin reveals a significant difference in mean serum cholesterol and HDLC levels ($p<0.05$). and the atorvastatin group's mean serum cholesterol/HDLC level is lower following treatment. There is a significant difference in the mean serum cholesterol/HDLC level ($p<0.05$) between the distribution of serum cholesterol/HDLC before and after rosuvastatin treatment. and the Rosuvastatin group's mean serum cholesterol/HDLC level is lower following treatment.

The percentage changes in LDL/HDL levels (before to after) in both treatment groups did not differ significantly ($p>0.05$). and the mean changes are 27.98 for the atorvastatin group and 35.26 for the rosuvastatin group. The distribution of LDL/HDL by group before and after treatment reveals a significant difference in the mean serum LDL/HDL levels in both treatment groups ($p<0.05$). The rosuvastatin group's mean LDL/HDLC level is 3.36, while the atorvastatin group's is 3.79. Additionally, compared to the atorvastatin group prior to treatment, the mean rosuvastatin 20 mg group had lower serum LDL/HDLC. The mean blood LDL/HDL levels in the two therapy groups varied significantly ($p<0.05$). The rosuvastatin group's mean LDL/HDLC level is 2.15, while the atorvastatin group's is 2.65. And according to mean rosuvastatin 20 mg have less serum LDL/HDLC in respect to atorvastatin group after treatment.

The mean LDL/HDLC level before and after treatment change significantly ($p<0.05$). and the Atorvastatin 20 mg group's mean LDL/HDLC level is lower following treatment.

The mean LDL/HDLC level before and after treatment change significantly ($p<0.05$). and the Rosuvastatin group's mean LDL/HDLC level is lower following medication. Total cholesterol, triglycerides, LDL-C, and VLDL-C were all considerably reduced by atorvastatin and rosuvastatin. The mean HDL cholesterol levels in the two treatment groups varied significantly ($p<0.05$). And according to mean rosuvastatin 20 mg have less LDL cholesterol in respect to atorvastatin group after treatment.

The percentage changes in HDL cholesterol levels before and after were observed to differ significantly between the two treatment groups ($p<0.05$). and the mean changes are -6.11 for the rosuvastatin group and -4.09 for the atorvastatin group. Additionally, the atorvastatin group's mean HDL cholesterol level is greater.

The mean serum cholesterol/HDLC levels in the two therapy groups varied significantly ($p<0.05$). The atorvastatin group's mean serum cholesterol/HDLC level is 4.40, while the rosuvastatin group's is 3.81. Additionally, after treatment, the rosuvastatin 20 mg group had lower serum cholesterol and HDLC than the atorvastatin group.

Table : Distribution of age according to Groups

Group Statistics						
	Group	N	Mean	Std. Deviation	T value	P value
Age	Atorvastatin	50	60.4200	13.09633	0.287	0.775

	20mg					
	Rosuvastatin	50	59.7600	9.64166		

Table : Distribution of Gender according to Groups

		Group		Total	Chi sq	P value
		Atorvastatin 20mg	Rosuvastatin			
Gender	Female	22	19	41		
		44.0%	38.0%	41.0%		
	Male	28	31	59	0.372	0.542
		56.0%	62.0%	59.0%		
Total		50	50	100		
		100.0%	100.0%	100.0%		

Table : Distribution of BLOOD GLUCOSE according to Groups

Group Statistics						
	Group	N	Mean	Std. Deviation	T value	P value
BLOOD GLUCOSE	Atorvastatin 20mg	50	258.8400	40.19613		
	Rosuvastatin	50	275.3200	52.88510	1.754	0.083

Table : Distribution of Serum lipid profile before treatment according to Groups

Group Statistics						
	Group	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL BEFORE	Atorvastatin 20mg	50	258.8400	36.61569		
	Rosuvastatin	50	247.6800	30.58974	1.654	0.101
HDL CHOLESTEROL	Atorvastatin 20mg	50	42.7000	5.70803		

BEFORE						
	Rosuvastatin	50	44.1200	4.56535	1.374	0.173
LDL CHOLESTEROL BEFORE	Atorvastatin 20mg	50	158.7400	34.65643		
	Rosuvastatin	50	146.5720	26.79735	1.964	0.052
TRIGLYCERIDES BEFORE	Atorvastatin 20mg	50	287.0000	21.84267		
	Rosuvastatin	50	284.9400	36.78643	0.340	0.734
VLDL BEFORE	Atorvastatin 20mg	50	57.4000	4.36853		
	Rosuvastatin	50	56.9880	7.35729	0.340	0.734

Table : Distribution of Serum lipid profile After treatment according to Groups

	Group	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL AFTER	Atorvastatin 20mg	50	193.0800	27.53094		
	Rosuvastatin	50	176.5000	30.40089	2.858	0.005*
HDL CHOLESTEROL AFTER	Atorvastatin 20mg	50	44.4200	5.76439		
	Rosuvastatin	50	46.7400	4.26093	2.289	0.024*
LDL CHOLESTEROL AFTER	Atorvastatin 20mg	50	115.7720	23.33745		
	Rosuvastatin	50	98.9760	30.73748	3.077	0.003*
TRIGLYCERIDES AFTER	Atorvastatin 20mg	50	164.4400	25.54081		
	Rosuvastatin	50	153.9200	20.18975	2.285	0.024
VLDL AFTER	Atorvastatin 20mg	50	32.8880	5.10816		
	Rosuvastatin	50	30.7840	4.03795	2.285	0.024

Atorvastatin 20mg

Table : Distribution of Serum lipid profile before treatment according to Gender in Atorvastatin 20mg

	Gender	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL BEFORE	Male	28	255.8571	37.75036		
	Female	22	262.6364	35.62442	0.646	0.521
HDL CHOLESTEROL BEFORE	Male	28	41.1786	5.57145		
	Female	22	44.6364	5.39440	2.209	0.032*
LDL CHOLESTEROL BEFORE	Male	28	156.8857	35.18116		
	Female	22	161.1000	34.65150	0.423	0.674
TRIGLYCERIDES BEFORE	Male	28	288.9643	23.96832		
	Female	22	284.5000	19.05068	0.714	0.479
VLDL BEFORE	Male	28	57.7929	4.79366		
	Female	22	56.9000	3.81014	0.714	0.479

Table : Distribution of Serum lipid profile After treatment according to Gender in Atorvastatin 20mg

Group Statistics						
	Gender	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL AFTER	Male	28	190.9286	25.74077		
	Female	22	195.8182	30.04384	0.619	0.539
HDL CHOLESTEROL AFTER	Male	28	42.9643	5.82130		
	Female	22	46.2727	5.24796	2.082	0.043*
LDL CHOLESTEROL AFTER	Male	28	115.1214	22.65610		
	Female	22	116.6000	24.68969	0.220	0.827
TRIGLYCERIDES AFTER	Male	28	164.2143	24.26300		
	Female	22	164.7273	27.65981	0.070	0.945

VLDL AFTER	Male	28	32.8429	4.85260		
	Female	22	32.9455	5.53196	0.070	0.945

Table : Distribution of Serum lipid profile before and after treatment in Atorvastatin 20mg

	Mean	N	Std. Deviation	T value	P value
SERUM CHOLESTEROL BEFORE	258.8400	50	36.61569		
SERUM CHOLESTEROL AFTER	193.0800	50	27.53094	15.054	<0.0001*
HDL CHOLESTEROL BEFORE	42.7000	50	5.70803		
HDL CHOLESTEROL AFTER	44.4200	50	5.76439	15.031	<0.0001*
LDL CHOLESTEROL BEFORE	158.7400	50	34.65643		
LDL CHOLESTEROL AFTER	115.7720	50	23.33745	9.604	<0.0001*
TRIGLYCERIDES BEFORE	287.0000	50	21.84267		
TRIGLYCERIDES AFTER	164.4400	50	25.54081	31.612	<0.0001*
VLDL BEFORE	57.4000	50	4.36853		
VLDL AFTER	32.8880	50	5.10816	31.612	<0.0001*

Rosuvastatin

Table : Distribution of Serum lipid profile before treatment according to Gender in Rosuvastatin

Group Statistics						
	Gender	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL BEFORE	Male	31	244.8387	30.43145		
	Female	19	252.3158	31.10029	0.836	0.407
HDL CHOLESTEROL	Male	31	44.3226	4.98924		

BEFORE	Female	19	43.7895	3.88128	0.397	0.693
LDL CHOLESTEROL BEFORE	Male	31	143.5484	27.31563		
	Female	19	151.5053	25.88078	1.020	0.313
TRIGLYCERIDES BEFORE	Male	31	284.8387	35.28937		
	Female	19	285.1053	40.10250	0.025	0.980
VLDL BEFORE	Male	31	56.9677	7.05787		
	Female	19	57.0211	8.02050	0.025	0.980

Table : Distribution of Serum lipid profile After treatment according to Gender in Rosuvastatin

Group Statistics						
	Gender	N	Mean	Std. Deviation	T value	P value
SERUM CHOLESTEROL AFTER	Male	31	177.9677	33.15568		
	Female	19	174.1053	25.96128	0.432	0.667
HDL CHOLESTEROL AFTER	Male	31	46.9355	4.57483		
	Female	19	46.4211	3.79057	0.411	0.683
LDL CHOLESTEROL AFTER	Male	31	99.5032	33.58916		
	Female	19	98.1158	26.27338	0.153	0.879
TRIGLYCERIDES AFTER	Male	31	157.6452	17.09103		
	Female	19	147.8421	23.66259	1.698	0.096
VLDL AFTER	Male	31	31.5290	3.41821		
	Female	19	29.5684	4.73252	1.698	0.096

Table : Distribution of Serum lipid profile before and after treatment in Rosuvastatin

	Mean	N	Std. Deviation	T value	P value
SERUM CHOLESTEROL BEFORE	247.6800	50	30.58974		

SERUM CHOLESTEROL AFTER	176.5000	50	30.40089	16.096	<0.0001*
HDL CHOLESTEROL BEFORE	44.1200	50	4.56535		
HDL CHOLESTEROL AFTER	46.7400	50	4.26093	13.687	<0.0001*
LDL CHOLESTEROL BEFORE	146.5720	50	26.79735		
LDL CHOLESTEROL AFTER	98.9760	50	30.73748	11.290	<0.0001*
TRIGLYCERIDES BEFORE	284.9400	50	36.78643		
TRIGLYCERIDES AFTER	153.9200	50	20.18975	26.746	<0.0001*
VLDL BEFORE	56.9880	50	7.35729		
VLDL AFTER	30.7840	50	4.03795	26.746	<0.0001*

Reference

1. Wilson PW, Anderson KM, Castelli WP. Twelve-year incidence of coronary heart disease in middle-aged adults during the era of hypertensive therapy: the Framingham offspring study. *Am J Med.* 1991 Jan;90(1):11-6. doi: 10.1016/0002-9343(91)90500-w. Erratum in: *Am J Med* 1991 Apr;90(4):537. PMID: 1986576.
2. Kontopoulou AG, Athyros VG, Papageorgiou AA, Hatzikonstandinou HA, Mayrouti MC, Boudoulas H. Effects of simvastatin and ciprofibrate alone and in combination on lipid profile, plasma fibrinogen and low density lipoprotein particle structure and distribution in patients with familial combined hyperlipidaemia and coronary artery disease. *Coron Artery Dis.* 1996 Nov;7(11):843-50. doi: 10.1097/00019501-199611000-00009. PMID: 8993943.
3. Bakker-Arkema RG, Davidson MH, Goldstein RJ, Davignon J, Isaacsohn JL, Weiss SR et al. Efficacy and safety of a new HMG-CoA reductase inhibitor, atorvastatin, in patients with hypertriglyceridemia. *JAMA.* 1996;275(2):128-33.
4. Bhopal R, Fischbacher C, Vartiainen E, Unwin N, White M, Alberti G. Predicted and observed cardiovascular disease in South Asians: application of FINRISK, Framingham and SCORE models to Newcastle Heart Project data. *J Public Health (Oxf).* 2005 Mar;27(1):93-100. doi: 10.1093/pubmed/fdh202. PMID: 15749725.
5. McKenney JM. Update on the National Cholesterol Education Program Adult Treatment Panel III guidelines: getting to goal. *Pharmacotherapy.* 2003 Sep;23(9 Pt 2):26S-33S. doi: 10.1592/phco.23.11.26s.32710. PMID: 14524636.

6. Bersot TP, Pépin GM, Mahley RW. Risk determination of dyslipidemia in populations characterized by low levels of high-density lipoprotein cholesterol. *Am Heart J.* 2003 Dec;146(6):1052-9. doi: 10.1016/S0002-8703(03)00516-7. PMID: 14660998
7. Topol EJ. Intensive statin therapy--a sea change in cardiovascular prevention. *N Engl J Med.* 2004 Apr 8;350(15):1562-4. doi: 10.1056/NEJMe048061. Epub 2004 Mar 8. PMID: 15007111.
8. Wang TD, Chen WJ, Lin JW, Cheng CC, Chen MF, Lee YT. Efficacy of fenofibrate and simvastatin on endothelial function and inflammatory markers in patients with combined hyperlipidemia: relations with baseline lipid profiles. *Atherosclerosis.* 2003 Oct;170(2):315-23. doi: 10.1016/s0021-9150(03)00296-x. PMID: 14612213.
9. Farnier M, Picard S. Diabetes: Statins, fibrates, or both. *Curr Atheroscler Rep.* 2001;3(1):19-28.
10. Cziraky MJ, Watson KE, Talbert RL. Targeting low HDL-cholesterol to decrease residual cardiovascular risk in the managed care setting. *J Manag Care Pharm.* 2008 Oct;14(8 Suppl):S3-28; quiz S30-1. doi: 10.18553/jmcp.2008.14.S8-A.1. PMID: 19891279; PMCID: PMC10438150.
11. Jackevicius CA, Tu JV, Ross JS, Ko DT, Krumholz HM. Use of ezetimibe in the United States and Canada. *N Engl J Med.* 2008;358:1819-28. doi:10.1056/NEJMs0801461. [DOI] [PubMed] [Google Scholar]
12. Guerin M, Egger P, Soudant C, Le Goff W, van Tol A, Dupuis R, Chapman MJ. Dose-dependent action of atorvastatin in type IIB hyperlipidemia: preferential and progressive reduction of atherogenic apoB-containing lipoprotein subclasses (VLDL-2, IDL, small dense LDL) and stimulation of cellular cholesterol efflux. *Atherosclerosis.* 2002 Aug;163(2):287-96. doi: 10.1016/s0021-9150(02)00037-0. PMID: 12052475.
13. Kreisberg RA. Diabetic dyslipidemia. *Am J Cardiol.* 1998 Dec 17;82(12A):67U-73U; discussion 85U-86U. doi: 10.1016/s0002-9149(98)00848-0. PMID: 9915665.
14. Cholesterol Treatment Trialists' (CTT) Collaboration; Baigent C, Blackwell L, Emberson J, Holland LE, Reith C, Bhala N, Peto R, Barnes EH, Keech A, Simes J, Collins R. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. *Lancet.* 2010 Nov 13;376(9753):1670-81. doi: 10.1016/S0140-6736(10)61350-5. Epub 2010 Nov 8. PMID: 21067804; PMCID: PMC2988224.
15. Kinlay S, Schwartz GG, Olsson AG, Rifai N, Leslie SJ, Sasiela WJ, et al. High dose atorvastatin enhances the decline in inflammatory markers in patients with acute coronary syndromes. *Circulation* 2003;26:571-81
16. Bairaktari ET, Tzallas CS, Tsimihodimos VK, Liberopoulos EN, Miltiados GA, Elisaf MS. Comparison of the efficacy of atorvastatin and micronized fenofibrate in the treatment of mixed hyperlipidemia. *J Cardiovasc Risk.* 1999 Apr;6(2):113-6. doi: 10.1177/204748739900600208. PMID: 10353071.
17. Goel PK, Bharti BB, Pandey CM, Singh U, Tewari S, Kapoor A, Garg N, Sinha N. A tertiary care hospital-based study of conventional risk factors including lipid profile in proven coronary artery disease. *Indian Heart J.* 2003 May-Jun;55(3):234-40. PMID: 14560932.
18. Jones PH, Hunninghake DB, Ferdinand KC, Stein EA, Gold A, Caplan RJ, Blasetto JW; Statin Therapies for Elevated Lipid Levels Compared Across Doses to Rosuvastatin Study Group. Effects of rosuvastatin versus atorvastatin, simvastatin, and pravastatin on non-high-density lipoprotein cholesterol, apolipoproteins, and lipid ratios in patients with hypercholesterolemia: additional results from the STELLAR trial. *Clin Ther.* 2004 Sep;26(9):1388-99. doi: 10.1016/j.clinthera.2004.09.006. Erratum in: *Clin Ther.* 2005 Jan;27(1):142. PMID: 15531001.
19. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In

- Adults (Adult Treatment Panel III). JAMA. 2001 May 16;285(19):2486-97. doi: 10.1001/jama.285.19.2486. PMID: 11368702.
20. Focus on hypertriglyceridemia: improving patient outcomes. J Am Acad Nurse Pract. 2007;19(12 Suppl 2):3. PMID: 18510280.
21. Talbert RL. Dyslipidemia. In: Diprio JT, editor. Pharmacotherapy-A Pathophysiological Approach. 8th ed. New York: McGraw-Hill; 2008. p. 365-88.
22. Tribble DL, Farnier M, Macdonell G, Perevozskaya I, Davies MJ, Gumbiner B, Musliner TA. Effects of fenofibrate and ezetimibe, both as monotherapy and in coadministration, on cholesterol mass within lipoprotein subfractions and low-density lipoprotein peak particle size in patients with mixed hyperlipidemia. Metabolism. 2008 Jun;57(6):796-801. doi: 10.1016/j.metabol.2008.01.026. PMID: 18502262.
23. Wierzbicki AS, Lumb PJ, Semra YK, Crook MA. High-dose atorvastatin therapy in severe heterozygous familial hypercholesterolaemia. QJM. 1998 Apr;91(4):291-4. doi: 10.1093/qjmed/91.4.291. PMID: 9666952.