Journal of Cardiovascular Disease Research

ISSN:0975-3583,0976-2833 VOL12,ISSUE06,2021

Comparative Study of Efficacy and Safety of Rosuvatatin 5mg Daily versus Rosuvastatin 10 mg Alternate Day in Patients with Dyslipidemia

Dr. Arikeri Vasu Deva Rao[1], Dr. Anand M. Ingale[2], Dr. Shrinivas.R.Raikar [3,*], Dr. Akram A. Naikawdi[4]

[1]Assistant Professor, Department of Pharmacology, BLDE (DU) Shri B.M.Patil Medical College Hospital and Research Centre Vijayapura Karnataka

[2]Associate Professor, Department of Pharmacology, BLDE (DU) Shri B. M.Patil Medical College Hospital and Research Center, Vijayapura Karnataka.

[3]Associate Professor, Department of Pharmacology, BLDE (DU) Shri.B.M.Patil Medical College, Hospital and Research Centre , Vijayapura Karnataka

[4]Professor and Head, Department of Pharmacology, BLDE (DU) Shri B.M.Patil Medical College Hospital and Research Centre Vijayapura Karnataka

Abstract

Background: To compare the efficacy of Rosuvastatin (5 mg) daily versus Rosuvastatin (10mg) in patients with dyslipidemia.

Methods: 52-week open-label, parallel-group, comparator, prospective, and crossover trial follows a randomized process with open-label treatments. The research included 60 patients with newly diagnosed dyslipidemia who were 18–70 years old and had Serum triglycerides levels > 150 mg/dl, VLDL-C > 30 mg/dl, LDL-C>100 mg/dl, Total Cholesterol > 200 mg/dl, and HDL-C of 40 mg/dl for both men and women, according to NCEP ATP III criteria. A group of patients was divided into two. Rosuvastatin 5 mg was given to group A daily, while Rosuvastatin 10 mg was given to group B every other day for 25 weeks. The second washout period comprised 25 weeks using Rosuvastatin at 10 mg every other day, while 5 mg was administered every day to group B. Following baseline measurements, TG, LDL-C, VLDL-C, HDL, and TC were measured, as well as at the end of weeks 25 and 52.

Results: As the 25th and 52nd weeks progressed, LDL-C, TG, VLDL-C, TC, and LDL/HDL were reduced more significantly in group B (13.44%, 27.27%, 37.74%, 25.86%, 25.43%, 40.74%) than in group A (33.04%, 17.79%, 15.31%, -1.79%, 37.03%, 22.22%) respectively. HDL-C concentrations in group A increased significantly (-8.68%) compared to group B (-4.9%).

Conclusions: Patients with dyslipidemia treated with Rosuvastatin 10mg alternate day had a better lipid profile and reduced patient costs and pill burden than those treated with Rosuvastatin 5mg daily.

Keywords: Dyslipidemia, Rosuvastatin

[*] Corresponding Author
Dr.Shrinivas.R.Raikar
Associate Professor
Department of Pharmacology
BLDE (DU) Shri B.M.Patil Medical College Hospital and Research Centre Vijayapura Karnataka
Email id shrinivasraikar@gmail.com

Introduction

Nearly 200 million people suffer from cardiovascular disease (CVD). (3,4) While CVD causes mortality rates to rise, it affects public health in a major way. The risk of cardiovascular disease can be modified by modifying dyslipidemia.(3). TC, LDL-C, TG and HDL-C levels are all major components of atherosclerosis and thus significant contributors to its progression; plaques develop in the endothelial cells of the arterial walls, increasing risks of heart disease.(4). An abnormal amount of lipids circulates in the blood as a result of dyslipidemia, a disorder of lipid metabolism. The progression of atherosclerosis includes high levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), as well as low levels of high-density lipoprotein cholesterol (HDL-C) in arteries.(5) Dyslipidemia can be classified as primary and secondary. There is a genetic component to primary dyslipidemia. Dyslipidemia secondary to secondary medical conditions such as hyperthyroidism is primarily caused by lifestyle choices or secondary medical conditions. Dyslipidemia may also be idiopathic (without a known cause). (6) A risk of mixed kinds of dyslipidemia is emerging due to the increasing prevalence of metabolic conditions such as type 2 diabetes, metabolic syndrome, and hypertension. (7)

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In addition to lifestyle changes, changes in diet and exercise, stress, and sedentary work, CVD incidence is increasing. In summary, lifestyle modification remains the first step in the treatment of dyslipidemia.(8) However, elderly patients can have difficulty keeping up with their treatment regimens. Patients with dyslipidemia can achieve the best results by changing their lifestyles and taking medication(9). Statins (HMG-CoA reductase inhibitors) are commonly prescribed medications in the world for the syndrome(10). Various statins are manufactured chemically and can be derived from fungi (Lovastatin, Simvastatin, and Pravastatin) or synthetically produced (Cerivastatin, Fluvastatin, Atorvastatin, Pitavastatin, Pravastatin, and Rosuvastatin).(11) By binding to the enzyme's active site, 3-hydroxy-3-methylglutaryl coenzyme A (HMGCoA), inhibits HMGCR's binding to HMGCOA, causing mevalonate to be produced..(12) In summary, lowering intracellular cholesterol levels in hepatocytes results in a reduction in LDL cholesterol levels, resulting in a drop in circulating total cholesterol, total LDL cholesterol (LDL-C), and triglycerides (TG).(13) Rosuvastatin should be prescribed to patients who have primary hypercholesterolemia, mixed dyslipidemia, hypertriglyceridemic disease, or homozygous familial hypercholesterolemia as well as children (aged 10-17) who have heterozygous familial hypercholesterolemia, for coronary revascularization and myocardial infarction prevention in patients with multiple risk factors and no evidence of coronary heart disease.(14)

Methods

Study design

We conducted a cross-over, crossover, and comparative open label, randomized, parallel group study.

Study population

The results of 60 patients with new dyslipidemia, according to NCEP ATP III guidelines (15) of both sexes, having total cholesterol greater than 200 mg/dl,LDL-C greater than 100 mg/dl, serum triglyceride levels over 150 mg/dl , and HDL-C levels of <40 mg/dl for men and 50% for women were enrolled in the study. Active liver disease, gallbladder disease, kidney disease, thyroid disease, pregnant and lactating women, or anyone on OCPs, corticosteroids, or hypolipidemic agents since the prior month were excluded from testing.

Treatment groups

Rosuvastatin 5 mg was administered daily in group A while Rosuvastatin 10 mg alternate days in group B, for 25 weeks, was given to patients meeting the eligibility criteria in a 1:1 ratio. A washout period of 2 weeks followed by a crossover of group A receiving Rosuvastatin 10 mg alternate day and group B receiving Rosuvastatin 5 mg daily for another 25 weeks was performed. Each patient was seen four times: at screening, at randomization, at follow-up (seeking to determine the end of 25th week, seeking to determine the end of 27th week) and at the end (at the end of 52nd week). Randomization and post-study visits provided study medication to enrolled patients. In addition to hematology and fasting lipid profile, renal function testing, thyroid test, liver function testing, blood sugar, creatine phosphokinase, and 12-lead electrocardiogram, the screening included numerous other tests. Tests on liver function, creatine phosphokinase, and fasting lipid profiles were repeated at the 25th and 52nd week as well as at the end of the study.

Primary and secondary endpoints

A primary endpoint was the percentage change in total cholesterol, LDL-cholesterol, triglycerides, and serum HDL-cholesterol at the end of the study as compared to the baseline. As part of the trial, we evaluated the following safety features: adverse events reported, observed, or enquired about during the course of the trial; and deviations from baseline in laboratory results, vital signs, and physical examination.

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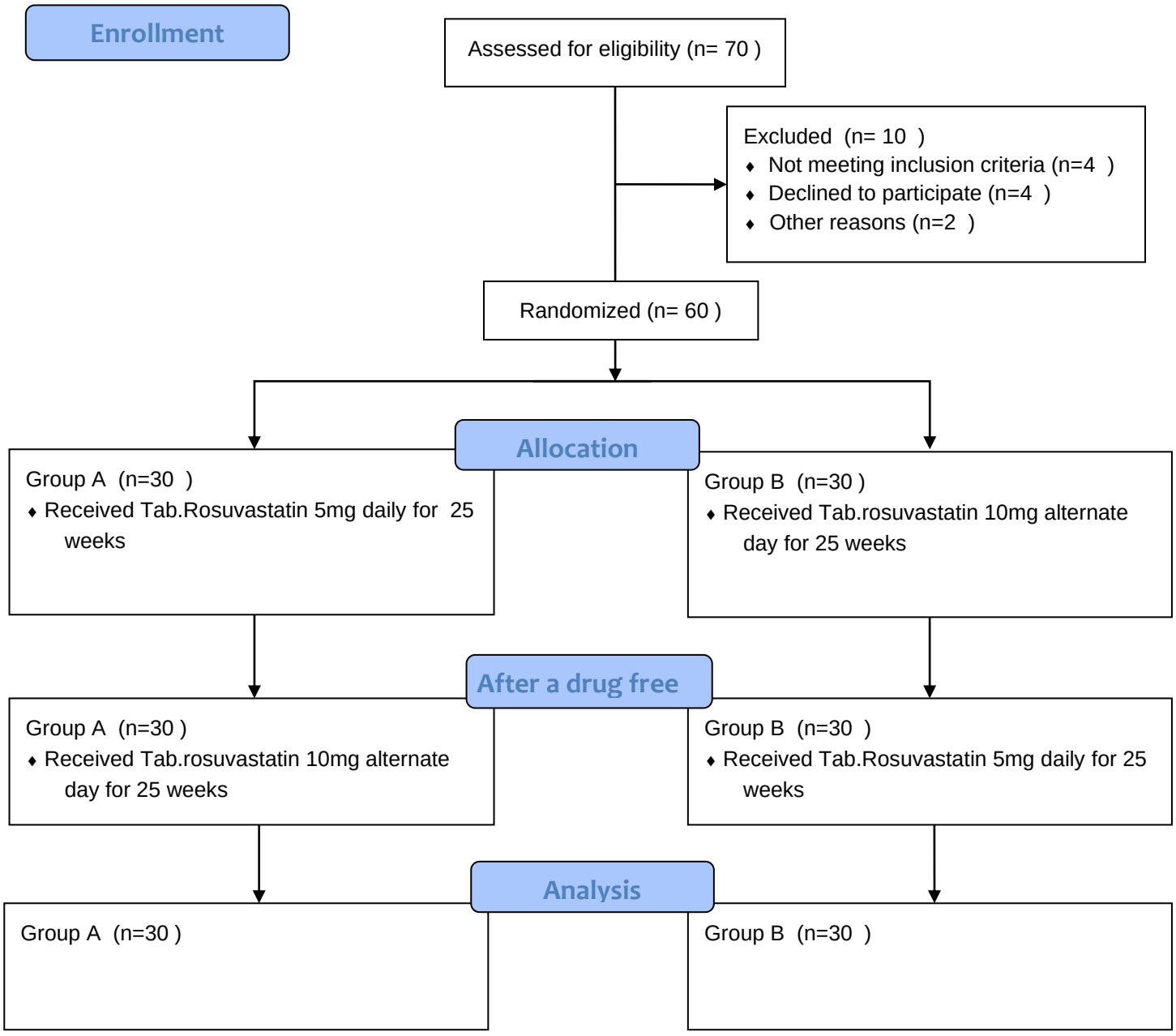
CONSORT Flow Diagram



In this study, approximately 70 patients were expected to enroll, assuming a 10% dropout rate (16). All information was collected by electronic case reports. Descriptive statistics were used to describe all characteristics. Summary statistics were calculated using the standard deviation (SD) for continuous variables. Data summaries and diagrammatic presentations use numbers and percentages for categorical data. In order to test the association

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between two categorical variables, the Chi-square test was used (*2). Unpaired t-tests were used to test whether two independent groups differed in their means. In a paired t test, the mean difference of variable analysis variables was compared between two time points within the same group. Statistical significance was considered to exist at a p-value of 0.05. The data were entered into the SPSS software v.23.0 and analyzed with Microsoft Office 2007.

Fig:1 Demographic data of study groups (A & B)

Parameters	Group A	Group B	p value
Age(yrs)	56.9±7.1	56.9±6.2	1
HEIGHT	149.7±6	151.6±9.4	0.364
WEIGHT	72.3±5	73.7±6.5	0.363
SYSTOLIC BP	147.7±12.3	143.7±14.2	0.249
DIASTOLIC BP	88.8±6.4	87±7	0.301
PULSE	89.9±6	86.2±5.2	0.013*
FBS	115.5±20.2	116.4±16.2	0.845
PPBS	213.2±33.4	191.4±56.8	0.075

Note: * significant at 5% level of significance (p<0.05)

Sex	Group A		Group B		p value
	N	%	N	%	
Male	21	70.0%	22	73.3%	0.774
Female	9	30.0%	8	26.7%	
Total	30	100.0%	30	100.0%	

Results

The study enrolled 60 patients (30 patients in each of the study arms- were included in the safety analysis) out of 70 screened patients. The protocol analysis included 60 patients (30 patients per group) who completed the study.

Baseline characteristics

Both groups had similar baseline characteristics, including age, gender, weight, height, concurrent illnesses, and baseline lipid levels.

Primary endpoints

All time points during the study period, the TC,LDL-C,VLDL-C,TG,TC/HDL,LDL/HDL serum triglyceride, total cholesterol, LDL-C levels significantly decreased. The mean percentage difference between groups A (17.79%,33.04%,15.32%), B (25.4%,37.74%,25.86%,27.27%,40.07%) was statistically significant (P 0.001). At the end of the 25th week after initiation of therapy, TC, LDL-C, and TG had significantly decreased with both doses of Rosuvastatin. Throughout the study, both treatment groups showed a decreasing trend of reduction.

Secondary endpoints

Both groups had a substantial rise in blood HDL-C levels compared to their baseline values at all time points during the trial (P 0.001). At the conclusion of the trial, group A's serum HDL-C level had climbed by 8.68 percent, whereas group B's had increased by 4.9 percent. In both groups, a rise in serum HDL-C was found, with an incremental impact reported throughout the research period. In group B, there was a substantial drop (13.44 percent) in VLDL levels (P 0.001). When compared to their baseline values, the TC/HDL ratio and LDL/HDL ratio were lowered more considerably in group B (27.27 percent and 40.7 percent) than in group A (22.22 percent and 37.03 percent) (P 0.001). However, following crossover, there was no statistically significant difference between the groups. During the trial period, no patient had an AST or ALT level that was more than 3 times the baseline level, or a CK level that was more than 10 times the baseline level.

Safety

During the course of the trial, a total of 50 adverse events were observed, 41 of which were minor in character. Nausea, vomiting, constipation, diarrhea, myalgia, sleeplessness, and headache were the most often reported side effects throughout the trial (17). During the research, nine severe adverse events (SAEs) necessitating hospitalization were recorded. All nine SAEs, on the other hand, were resolved without any squealae.

Fig:2 Comparison of mean lipid profile between group A & group B.

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Lipid Profile	Group A	Group B	p value
TC			
BASELINE	196.1±26.2	188.3±19.1	0.189
25TH WEEK	168.8±23	145.4±18	<0.001*
52ND WEEK	161.2±17.1	140.4±14.5	<0.001*
LDL			
BASELINE	149.8±16.8	151.8±10.7	0.588
25TH WEEK	114.3±20.8	103.7±14.3	0.025*
52ND WEEK	100.3±10.6	94.5±7.9	0.021*
VLDL			
BASELINE	50.2±4.2	59.5±5.2	<0.001*
25TH WEEK	44.5±4.9	51.9±10	0.001*
52ND WEEK	51.1±6.5	51.5±6	0.779
TG			
BASELINE	164.5±17.2	178.2±13.2	0.001*
25TH WEEK	144.1±14.8	134.4±13	0.009*
52ND WEEK	139.3±13.3	132.1±13.9	0.046*
HDL			
BASELINE	54.1±2.8	56.6±2.6	0.001*
25TH WEEK	57.3±2.4	61±3.1	<0.001*
52ND WEEK	58.8±4	59.4±3.4	0.521
TC/HDL			
BASELINE	3.6±0.5	3.3±0.4	0.009*
25TH WEEK	3±0.4	2.4±0.3	<0.001*
52ND WEEK	2.8±0.4	2.4±0.4	<0.001*
LDL/HDL			
BASELINE	2.7±0.3	2.7±0.2	0.395
25TH WEEK	2±0.4	1.7±0.2	0.001*
52ND WEEK	1.7±0.2	1.6±0.1	0.037*

Note: * significant at 5% level of significance (p<0.05)

Fig:3 Comparison of LFT & CK between group A & group B.

LFT & CK	Group A	Group B	p value
SGOT			
BASELINE	35.8±7.1	36.3±4.5	<0.001*
52ND WEEK	55±9.9	76.2±11.2	<0.001*
SGPT			
BASELINE	32.5±6.3	27.8±5	0.002*
52ND WEEK	53.6±9.9	59.3±12.8	0.049*
ALP			
BASELINE	58.4±13	62.1±12	0.259
52ND WEEK	94.9±17.4	113.5±13.3	<0.001*
CK			
BASELINE	47±14.3	45.1±11.6	0.572
52ND WEEK	81.1±14.9	108.1±19.4	<0.001*

Note: * significant at 5% level of significance (p<0.05)

Fig:4 Comparison lipid profile from baseline to 25th week and 52nd week in group A

Paramete	Baseline	25th week	p	Baseline	52nd	p	25th week	52nd	p

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rs			value		week	value		week	value
TC	196.1±26.2	168.8±23	<0.001*	196.1±26.2	161.2±17.1	<0.001*	168.8±23	161.2±17.1	<0.001*
LDL	149.8±16.8	114.3±20.8	<0.001*	149.8±16.8	100.3±10.6	<0.001*	114.3±20.8	100.3±10.6	<0.001*
VLDL	50.2±4.2	44.5±4.9	<0.001*	50.2±4.2	51.1±6.5	0.47	44.5±4.9	51.1±6.5	<0.001*
TG	164.5±17.2	144.1±14.8	<0.001*	164.5±17.2	139.3±13.3	<0.001*	144.1±14.8	139.3±13.3	0.184
HDL	54.1±2.8	57.3±2.4	<0.001*	54.1±2.8	58.8±4	<0.001*	57.3±2.4	58.8±4	0.095
TC/HDL	3.6±0.5	3±0.4	<0.001*	3.6±0.5	2.8±0.4	<0.001*	3±0.4	2.8±0.4	0.001*
LDL/HDL	2.7±0.3	2±0.4	<0.001*	2.7±0.3	1.7±0.2	<0.001*	2±0.4	1.7±0.2	0.001*

Note: * significant at 5% level of significance (p<0.05)

Fig:5 Comparison lipid profile from baseline to 25th week and 52nd week in group B

Parameters	Baseline	25th week	p value	Baseline	52nd week	p value	25th week	52nd week	p value
TC	188.3±19.1	145.4±18	<0.001*	188.3±19.1	140.4±14.5	<0.001*	145.4±18	140.4±14.5	0.075
LDL	151.8±10.7	103.7±14.3	<0.001*	151.8±10.7	94.5±7.9	<0.001*	103.7±14.3	94.5±7.9	0.011*
VLDL	59.5±5.2	51.9±10	<0.001*	59.5±5.2	51.5±6	<0.001*	51.9±10	51.5±6	0.849
TG	178.2±13.2	134.4±13	<0.001*	178.2±13.2	132.1±13.9	<0.001*	134.4±13	132.1±13.9	0.544
HDL	56.6±2.6	61±3.1	<0.001*	56.6±2.6	59.4±3.4	<0.001*	61±3.1	59.4±3.4	0.034*
TC/HDL	3.3±0.4	2.4±0.3	<0.001*	3.3±0.4	2.4±0.4	<0.001*	2.4±0.3	2.4±0.4	0.832
LDL/HDL	2.7±0.2	1.7±0.2	<0.001*	2.7±0.2	1.6±0.1	<0.001*	1.7±0.2	1.6±0.1	0.137

Note: * significant at 5% level of significance (p<0.05)

Fig:6 Comparison of LFT & CK baseline to 52nd week in group A

Parameters	BASELINE	52ND WEEK	t value	p value
SGOT	35.8±7.1	55±9.9	-14.426	<0.001*
SGPT	32.5±6.3	53.6±9.9	-13.269	<0.001*
ALP	58.4±13	94.9±17.4	-14.215	<0.001*
CK	47±14.3	81.1±14.9	-16.895	<0.001*

Note: * significant at 5% level of significance (p<0.05)

Fig:7 Comparison of LFT & CK baseline to 52nd week in group B

Parameters	BASELINE	52ND WEEK	t value	p value
SGOT	36.3±4.5	76.2±11.2	-14.42	<0.001*

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SGPT	27.8±5	59.3±12.8	-11.618	<0.001*
ALP	62.1±12	113.5±13.3	-15.385	<0.001*
CK	45.1±11.6	108.1±19.4	-21.552	<0.001*

Note: * significant at 5% level of significance ($p<0.05$)

Discussion

In this investigation, both Rosuvastatin regimens were shown to have proven the lipid-modulating characteristics of Rosuvastatin in patients with dyslipidemia: decreases in LDL-C, (18) triglycerides, and non-HDL-C when compared to Zhao S study (19). Similarly, a rise in HDL-C serum levels in both groups was consistent with Wang et alfindings. .'s (20) According to Panchavarthi et al., when two regimens were compared to evaluate the superiority of one medication over the other, it was discovered that Rosuvastatin 10mg alternate day dosage was helpful in improving the lipid profile of individuals with dyslipidemia (21). The major goal of this research was to lower LDL-C levels in accordance with NCEP ATP III recommendations in order to lessen the risk of CAD development. For people at high risk of coronary heart disease, the NCEP III recommends an LDL reduction objective of less than 100 mg/dl. According to NCEP ATP III recommendations, 83.33 percent of participants in this research met their LDL-C goal. Rosuvastatin is one of the most powerful statins, capable of reaching therapeutic goals in the great majority of patients. Treatment with statins has been linked to decreased LDL levels and fewer cardiovascular events, according to research. (22) HDL cholesterol, which is an independent measure of cardiovascular risk, responds well to rosuvastatin (low HDL). When Statin therapy for high cholesterol levels were evaluated across dosages of Rosuvastatin [STELLAR] research, HDL-C rose by 8% to 11% and triglyceride reductions varied from 22% to 34%. (23). Furthermore, according to Al Shafi Majumder A et al., the safety profiles of both regimens seem to be comparable (24)

Statin usage has also been shown to alter hepatic function (18). Asymptomatic increases of the liver enzymes ALT and AST, often known as transaminitis (19), are the most common indicator. No participants in the present research had an ALT level more than 3 times the upper limit of normal. As a result, no adverse events related to hepatic function were recorded with any of the statins studied. This is not unexpected, given that clinical studies have shown a 0.5–3.0% frequency of aminotransferase increases in people on statins, as well as extremely rare occurrences of serious liver damage. Hepatic failure seems to be more common in people using statins than in the general population (19).

After two years, the three statins studied (atorvastatin 10, 20, and 40 mg; rosuvastatin 10 and 20 mg; and pravastatin 20 and 40 mg) did not have a significant effect on blood creatinine and GFR. Furthermore, the three statins proved to be generally safe for individuals with microalbuminuria at baseline, with only a small percentage of patients seeing an increase in microalbuminuria. Pravastatin (40 mg) seemed to minimize the frequency of individuals with microalbuminuria at baseline. In individuals without baseline microalbuminuria, however, there seemed to be a rather substantial beginning of microalbuminuria in patients receiving pravastatin (26.6%), rosuvastatin (14.3%), and atorvastatin (13.3%). (10.9 percent). The literature on the effects of statins on microalbuminuria is mixed. While other statin studies have found a decrease in proteinuria (10) or no impact (11), some literature supports the present study's conclusions that statins do have deleterious effects on proteinuria onset (19). The key explanation for atorvastatins' safety in regard to renal function is their relatively unique manner of metabolism, in which it has the least amount of renal excretion (2%) compared to fluvastatin (5%), rosuvastatin (10%), lovastatin (10%), simvastatin (13%), and pravastatin (20%) (22).

Conclusion

The main reasons of atherogenic risk include hypercholesterolemia and low HDL-C. Dyslipidemia is caused by both hereditary and lifestyle factors. With alternate-day Rosuvastatin 10mg, we saw a substantial decrease in TC, LDL-C, and TG in our trial. Even at relatively low dosages of Rosuvastatin, modest side effects have been documented, despite the fact that it is well tolerated. Treatment with low doses of rosuvastatin is safe and well tolerated. Statin side effects are rare and typically minor, temporary, and reversible, highlighting the fact that the benefits of statins much outweigh the dangers. This research found that alternate-day medication may be more effective not only in terms of improving lipid profiles, but also in terms of lowering therapy costs, pill load, and statin-related side effects, as well as enhancing patient compliance, which is critical in the healthcare system. This enhanced efficacy and safety for dyslipidemic individuals may translate into an advantage in reaching and maintaining the treatment objective in clinical practice. Clinicians will be better equipped to choose the right statin for the right regimen if they have a better grasp of the relatively frequent statin-related side effects.

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