

Original Research Article

Comparison of the efficacy and safety of rosuvastatin versus atorvastatin in reduction of low density lipoprotein cholesterol in patients of type 2 diabetes mellitus with dyslipidemia

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ABSTRACT

Background: Approximately 80% of deaths in diabetic patients are attributable to cardiovascular disease (CVD), which in turn is highly correlated with diabetic dyslipidemia. Statins are drug of choice for raised LDL-C in treating dyslipidemia. The present study compares the efficacy and safety of rosuvastatin against commonly used atorvastatin in patients of type 2 diabetes mellitus with dyslipidemia, so as to guide the present treatment strategies in the management of the same in Indian population.

Methods: The study was a single blinded study conducted in a district level tertiary care hospital attached to a medical teaching institute. Patients fulfilling the inclusion criteria were randomized in two groups. Group I received atorvastatin (10mg) and group II received rosuvastatin (5mg) at bedtime orally daily. Serum TC, serum LDL-C, serum HDL-C and serum TG were assessed on week 0, week 6 and week 12.

Results: At the end of 12 weeks, the percentage reduction of LDL-C levels in atorvastatin group was 33.58% whereas in rosuvastatin group, it was 43.12%. The percentage reduction in total cholesterol (TC) in atorvastatin group was 24.85% while in rosuvastatin group, it was 30.8%. Rise in HDL-C levels in atorvastatin group was 7.1% whereas in rosuvastatin group, it was 11.16%. All these differences were statistically significant. There was no significant difference in reduction of TG levels between the two groups.

Conclusions: Rosuvastatin 5mg causes greater reduction in LDL-C and TC, comparable reduction of TG and greater rise in HDL-C when compared with atorvastatin 10mg therapy.

Keywords: Atorvastatin, Cholesterol, Cardiovascular disease, Diabetes, Dyslipidemia, Rosuvastatin

INTRODUCTION

Diabetes mellitus is a common metabolic disorder characterized by absolute or relative deficiencies in insulin secretion and/or insulin action associated with chronic hyperglycemia and disturbances of carbohydrate, lipid and protein metabolism.¹ India has earned the distinction of being called as diabetic capital of the World.² It is estimated that 69.2 million people aged 20-79 years live with diabetes in India. This number is

expected to increase to 123.5 million by 2040. About 1 million people died from diabetes in India in 2015.³

Diabetes mellitus is associated with increased oxidative stress due to hyperglycemia, which plays a role in development of micro and macro vascular complications involving almost all vital organs such as heart, eyes, kidney, blood vessels, and nervous system. These complications lead to the development of obesity, hypertension, dyslipidemia and insulin resistance.⁴

Approximately 80% of deaths in diabetic patients are attributable to cardiovascular disease (CVD), which in turn is highly correlated with diabetic dyslipidemia.⁵ Abnormalities in the lipid profile leads to a release of free fatty acids from adipose tissue, increased delivery of these acids to the liver and increased hepatic synthesis of lipoproteins, resulting in a pro-atherogenic lipid profile consisting of small dense Low Density Lipoprotein (LDL) particles, low High Density Lipoprotein (HDL) concentrations and high levels of triglycerides (TG).^{6,7} Besides the co-existence of cardiovascular risk factors and co-morbidity, patients with T2DM have an early development of abnormal endothelial dysfunction, platelet hyperactivity and impaired fibrinolysis with a tendency for thrombosis and inflammation. This leads to early development of adverse arterial remodeling and aggressive atherosclerosis.⁸

Atherosclerosis of coronary vessels is the main pathogenic mechanism responsible for CVD and efforts to reduce this provide an important therapeutic strategy to reduce mortality related to acute cardiovascular events.⁹ Therefore lowering LDL-C levels is the first priority in treating diabetic dyslipidemia.

Statins are competitive inhibitors of the enzyme 3-hydroxy 3-methyl glutaryl coenzyme A (HMG-CoA) reductase, which catalyze an early, rate limiting step in cholesterol biosynthesis.¹⁰ Because of their safety, efficacy and tolerability these cholesterol lowering agents have become drug of choice for raised LDL-C in treating dyslipidemia.¹¹ In addition to the numeric reduction in lipid levels, statins significantly reduce vascular events and all-cause mortality through their pleiotropic effects. It has already been proved that statins have antioxidant, anti-inflammatory effects and antithrombotic properties that add to their clinical utility.¹² They improve endothelial dysfunction and reduce the growth of atherosclerotic plaque.¹³

Different statins require different dosing to reach the same LDL-C level.¹⁴ Multiple clinical trials have documented the efficacy and safety of atorvastatin and rosuvastatin in reducing fatal and non-fatal CVD events.¹⁵ Atorvastatin is the most commonly prescribed statin.¹¹ Evidence from the Western countries suggests that rosuvastatin achieves greater reductions in LDL-C.¹⁶

However, such data from our country in the clinical subset of diabetic patients is limited and it is well known that Asians may respond differently from whites because of genetic differences in drug metabolism at the hepatic enzyme and drug transporter level.¹⁷

The present study was thus planned to evaluate the efficacy and safety of rosuvastatin against commonly used atorvastatin in patients of type 2 diabetes mellitus with dyslipidemia, so as to guide the present treatment strategies in the management of the same in Indian population.

METHODS

This study was conducted at medicine department of a tertiary care hospital attached to medical college. The study was approved by Institutional Ethics Committee on 05.12.2014.

The patients were recruited from cardiovascular OPD and diabetes OPD. They were screened for participating in the study. Patients were diagnosed on the basis of history and biochemical investigations. Patients who were found fit to be included into the study were explained the aims and objectives of the study in detail. They were informed about the benefits of the study along with possible risks. After explaining the entire scope of the study, a written informed consent was obtained from them. The written informed consent was based on the specimen informed consent document. The patients were randomly allocated to either group I or group II of the treatment group based on chit method. Patients were blinded and were not informed about the drug they were to receive.

Baseline investigations including serum TC, serum LDL-C, serum HDL-C, serum TG levels, SGOT, SGPT and serum creatine phosphokinase (CPK) levels were done at the time of enrolment of patients (0 week).

Patients from Group I received atorvastatin (10mg) at bedtime orally daily and patients from group II received rosuvastatin (5mg) at bedtime orally daily. All patients also received the other concurrently required medications such as antidiabetic, antihypertensive or antianginal drugs etc as advised by treating physician. No patient used any other lipid lowering agents like bile acid sequestrants, fibrates or niacin. For patients who were already on statin therapy, a drug wash-out period of six weeks was allowed.

Study treatment was started on the day of randomization and continued for 12 weeks. After randomization, follow up visits were scheduled at 6 and 12 weeks. At each follow up, investigations like serum TC, serum LDL-C, serum HDL-C and serum TG were estimated, and patients were interviewed and examined for occurrence of myalgia, jaundice or any other adverse effect. Also, CPK, SGOT, and SGPT estimations were done at 6 and 12 weeks in all patients from both the groups to check for hepatotoxicity or myopathy.

Statistical Analysis was done using 'Z' test, paired t-test and unpaired t-test at appropriate places. A 'p' value <0.05 was considered statistically significant.¹⁸

RESULTS

A total 100 patients were included in the study, of which 50 patients were allocated to daily atorvastatin group and 50 patients to daily rosuvastatin group. During the study period two patient from daily atorvastatin group and one patient from daily rosuvastatin group were lost to follow

up and hence excluded from the analysis. Thus 48 patients from daily atorvastatin group and 49 patients from daily rosuvastatin group were considered for the analysis of data.

The baseline characteristics of the patients of both the groups were comparable with respect to age, sex and clinical profile.

Table 1: Baseline lipid profile of patients.

Baseline lipid values (mg/dL)	Daily atorvastatin (10mg)	Daily rosuvastatin (5mg)	p value
LDL-C	154.4±13.65	153.1±15.45	>0.05
TC	225.68±15.58	224.66±16.04	>0.05
TG	155.04±22.25	154.82±26.17	>0.05
HDL-C	40.27±3.58	40.59±3.18	>0.05

Unpaired t-test, Figures are Mean ± Standard Deviation

As Table 1 shows, the baseline mean lipid values of both the groups were comparable and there was no statistically significant difference between the two groups (p>0.05).

Table 2: LDL-C (mg/dL) in both treatment groups.

Group	Daily Atorvastatin (10mg)	Daily Rosuvastatin (5mg)	p value
6 weeks	121.83±19	106.39±13.04	<0.0001
12 weeks	102.56±16.11	87.1±11.68	<0.0001

Unpaired t test, Figures are Mean ± Standard Deviation

As Table 2 shows, there was significantly greater reduction in levels of LDL-C in patients treated with rosuvastatin therapy as compared to those treated with atorvastatin (p<0.0001). The percentage reduction of LDL-C levels in atorvastatin group at 6 and 12 weeks was 21.1% and 33.58% respectively (Figure 1). The percentage reduction of LDL-C levels in rosuvastatin group at 6 and 12 weeks was 30.51% and 43.12% respectively.

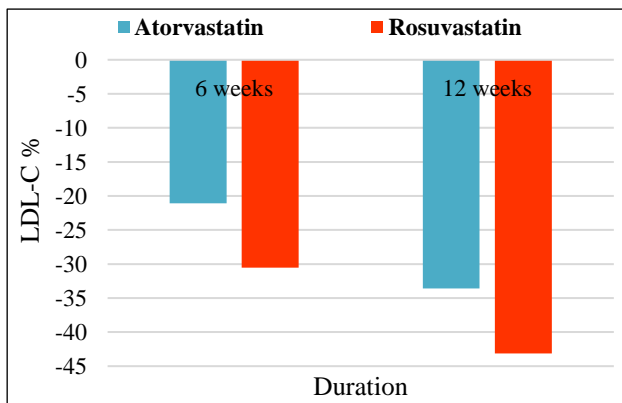


Figure 1: Percentage reduction in the levels of serum LDL-C in both treatment groups.

Table 3: Percentage of patients who achieved levels of LDL-C <100 mg/dL.

Group	Daily Atorvastatin (10 mg)	Daily Rosuvastatin (5 mg)	p value
6 weeks	20.83% (10/48)	44.9% (22/49)	<0.05
12 weeks	60.42% (29/48)	83.67% (41/49)	<0.05

Z test for difference between two proportions

As Table 3 shows, significantly higher number of patients from rosuvastatin group achieved levels of LDL-C <100mg/dL at 6 and 12 weeks (p<0.05).

Table 4: Levels of total cholesterol (TC) mg/dL in two treatment groups.

Group	Daily Atorvastatin (10 mg)	Daily Rosuvastatin (5 mg)	p value
6 weeks	190.22±20.02	174.4±14.16	<0.0001
12 weeks	169.59±18.04	155.47±13.77	<0.0001

Unpaired t-test, Figures are Mean ± Standard Deviation

As Table 4 shows, reduction in levels of total cholesterol in rosuvastatin group was significantly higher than in the atorvastatin group (p<0.0001). The percentage reduction in total cholesterol in atorvastatin group at 6 and 12 weeks was 15.71% and 24.85% respectively. In rosuvastatin group the percentage reduction in total cholesterol at 6 and 12 weeks was 22.37% and 30.8% respectively.

Table 5: Serum triglycerides (mg/dL) in two treatment groups.

Group	Daily Atorvastatin (10 mg)	Daily Rosuvastatin (5 mg)	p value
6 weeks	130.67±18.14	126.27±23.11	>0.05
12 weeks	119.50±18.07	116.24±22.71	>0.05

Unpaired t test, Figures are Mean ± Standard Deviation

Table 6: Changes in mean values of HDL-C (mg/dL) in two treatment groups.

Group	Daily Atorvastatin (10 mg)	Daily Rosuvastatin (5 mg)	p value
6 weeks	42.25±3.46	42.76±3.16	>0.05
12 weeks	43.13±3.67	45.12±3.33	<0.05

As Table 5 shows, though there was greater reduction in levels of triglycerides in patients treated with rosuvastatin therapy as compared to those treated with atorvastatin, the difference was not statistically significant (p>0.05). The percentage reduction in levels of triglyceride in atorvastatin group at 6 and 12 weeks was 15.72% and 22.92% respectively. In rosuvastatin group the percentage

reduction in triglyceride levels at 6 and 12 weeks was 18.44% and 24.92% respectively.

As Table 6 shows, at 6 weeks, the difference between the two therapies was not statistically significant ($p>0.05$) whereas at 12 weeks, there was significantly more increase in HDL-C levels with rosuvastatin therapy as compared to atorvastatin therapy ($p<0.05$).

Total 7 patients of atorvastatin group and 9 patients from rosuvastatin group reported mild and self-limiting adverse effects like nausea, headache, bodyache or abdominal pain. There was no statistically significant difference in the incidence of these adverse effects in the two treatment groups ($p>0.05$). There was no occurrence of any serious adverse event in any patient during this study. During this study no patient from either group showed significant increase in serum CPK, SGOT, SGPT levels at 12 weeks.

DISCUSSION

In the present study, patients received either atorvastatin (10mg) or rosuvastatin (5mg) as daily therapy. Similar doses had been used in several studies comparing the efficacy and safety of atorvastatin therapy with that of rosuvastatin. In studies such as URANUS, ANDROMEDA, Adsule et al and Barakat et al investigators had used 10mg atorvastatin.¹⁹⁻²² In the LISTEN trial and the trial by Arshad et al 10mg atorvastatin was compared against 5mg rosuvastatin.^{23,24} Besides, the FDA recommends a starting dose of rosuvastatin 5mg in Asians while the starting dose of atorvastatin is 10mg.^{25,26}

In the present study, at the end of 12 weeks, it was found that there was statistically significant difference between atorvastatin and rosuvastatin therapy in reduction of LDL-C levels. Also, the percentage reduction of LDL-C levels in rosuvastatin group was significantly higher. These findings are consistent with those of ANDROMEDA, URANUS, CORALL and LISTEN trials, all of which were done on diabetic dyslipidemic patients.^{20,19,27,23}

In the double blind ANDROMEDA study, the percentage reduction of LDL-C levels from the baseline at 8 weeks in atorvastatin group (10mg) was 39% whereas in rosuvastatin group (10mg) it was 51%.²⁰ In the URANUS study comparing atorvastatin and rosuvastatin, both started at 10mg daily, and the dose titrated up periodically till specific LDL-C goals were achieved, the percentage reduction of LDL-C levels from the baseline in atorvastatin group was 45.5% whereas in rosuvastatin group it was 52.3% at the end of 16 week study.¹⁹ Similar results were obtained in the CORALL study where 45.6% and 50.6% were the percent reductions in LDL-C levels in the atorvastatin 20mg and rosuvastatin 10mg group respectively at the end of 12 weeks.²⁷ In these studies, the difference in the percent reductions of LDL-C levels in

atorvastatin and rosuvastatin groups was statistically significant.^{19,20,27} In the LISTEN trial too, the rosuvastatin group showed greater percent reductions in LDL-C levels as compared to atorvastatin group considering the overall results at the end of 3, 6 and 12 months.²³

STELLAR trial comparing rosuvastatin with atorvastatin, simvastatin, and pravastatin, in which non-diabetics were also included, revealed that rosuvastatin produced a significantly greater reduction in LDL-C levels as compared to its competitors.²⁸ These findings are similar to the one seen in the present study.

However, in the prospective, randomized study by Adsule et al, though the percentage reduction of LDL-C was more in the rosuvastatin group (44.25%) as compared to the atorvastatin group (35.56%), this difference was not statistically significant ($p>0.05$), which may be attributable to the smaller sample size.²¹

In the present study, after 12 weeks, significantly higher number of patients from rosuvastatin group achieved $<100\text{mg/dL}$ LDL-C levels (Table 3). A similar finding was seen in CORALL study where 76.5% patients from atorvastatin group and 83.1% patients from rosuvastatin group achieved LDL-C levels $<100\text{ mg/dL}$ at the end of 12 weeks.²⁷

In the present study, at the end of 12 weeks significantly higher percentage reduction in total cholesterol (TC) was seen in rosuvastatin group. Similar findings had been reported by URANUS trial and CORALL study.^{19,27}

However, the study by Adsule et al, notes that although rosuvastatin caused greater percentage reduction of TC as compared to atorvastatin (30.83% vs 25.75%), there was no statistically significant difference, which may be attributable to the smaller sample size.²¹

The percentage reductions in triglyceride level at the end of the present study in atorvastatin group and rosuvastatin group were 22.92% and 24.92% respectively and this difference between two groups was not statistically significant. Similar finding was found in CORALL, URANUS, ANDROMEDA and LISTEN trials and the study by Adsule et al.^{27,19,20,23,21}

In the present study, after 6 weeks and 12 weeks of therapy, both atorvastatin and rosuvastatin significantly increased HDL-C levels when compared to their respective baseline levels but the difference between both the groups at 6 weeks was not statistically significant. At the end of 12 weeks however, rosuvastatin caused a statistically significant rise in HDL-C as compared to atorvastatin (11.16% vs 7.1%). This finding is similar to the results of studies on Asian diabetic dyslipidemic patients. The LISTEN trial and ASTRO-2 trial on Japanese patients and the study by Adsule et al, on Indian subjects yielded similar results.^{23,29,21} However, European studies such as CORALL, URANUS and

ANDROMEDA on diabetic dyslipidemic patients reported statistically significant increase in HDL-C levels within atorvastatin and rosuvastatin groups but non-significant inter-group differences.^{27,19,20} This may be attributed to the fact that a higher level of plasma exposure to rosuvastatin and its metabolites is found in Asians as compared to Europeans.^{30,31}

The results of our study hence indicate that treatment with rosuvastatin 5mg causes greater reduction in LDL-C and TC and comparable reduction of TG when compared with atorvastatin 10mg therapy. Rosuvastatin therapy also led to greater rise in HDL-C levels at the end of 12 weeks compared to atorvastatin therapy, the inter-group difference being statistically significant. Considering the overall changes to lipid variables, the findings of the present study indicate that a less atherogenic lipid profile was achieved with rosuvastatin. The safety and tolerability elicited by both regimens in present study were consistent with the previous studies.^{19,20-23,27}

CONCLUSION

Rosuvastatin 5mg is more efficacious than atorvastatin 10 mg in reducing LDL-C and TC levels and in increasing HDL-C levels and showed a comparable safety profile with atorvastatin 10mg after 12 weeks of therapy in patients of type 2 diabetes mellitus with dyslipidemia. The greater efficacy of rosuvastatin will enable more patients to achieve recommended treatment goals in clinical practice and may provide further reductions in the risk of CVD. However, long-term economic analyses of rosuvastatin are needed to determine its potential as a more cost-effective therapy compared with atorvastatin.

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