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Original Research Article

Comparative Study of Atorvastatin versus Rosuvastatin in Reducing LDL Cholesterol in Dyslipidemia Patients

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Abstract

Background: Dyslipidemia is a major risk factor for cardiovascular diseases and is characterized by elevated levels of low-density lipoprotein cholesterol (LDL-C), triglycerides, or reduced high-density lipoprotein cholesterol (HDL-C). Statins remain the cornerstone of pharmacological therapy for dyslipidemia due to their ability to inhibit HMG-CoA reductase and reduce cholesterol synthesis in the liver. Among commonly prescribed statins, atorvastatin and rosuvastatin are widely used due to their potent lipid-lowering effects. Several studies suggest that rosuvastatin may provide greater reductions in LDL-C compared with atorvastatin at equivalent doses.

Aim: To compare the efficacy of atorvastatin and rosuvastatin in reducing LDL cholesterol levels in patients with dyslipidemia.

Materials and Methods: A prospective comparative study was conducted among 200 patients with dyslipidemia attending the medicine outpatient department of a tertiary care hospital. Patients were randomly allocated into two groups: Group A (Atorvastatin 20 mg, n=100) and Group B (Rosuvastatin 10 mg, n=100). Baseline lipid profile parameters were recorded and repeated after 12 weeks of therapy. Statistical analysis was performed using Student's t-test and chi-square test. A p-value <0.05 was considered statistically significant.

Results: Both atorvastatin and rosuvastatin significantly reduced LDL cholesterol levels. However, rosuvastatin demonstrated a greater percentage reduction in LDL-C compared with atorvastatin (48.6% vs 38.2%, p<0.001). A higher proportion of patients receiving rosuvastatin achieved target LDL levels (<100 mg/dl) compared with those receiving atorvastatin (74% vs 56%).

Conclusion: Both statins were effective in lowering LDL cholesterol levels, but rosuvastatin showed significantly greater LDL reduction and better achievement of target lipid levels. Rosuvastatin may therefore be considered a more potent statin for managing dyslipidemia.

Keywords: Dyslipidemia, Atorvastatin, Rosuvastatin, LDL cholesterol, Statins.

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Introduction

Dyslipidemia is a major modifiable risk factor for cardiovascular diseases such as coronary artery disease, stroke, and peripheral vascular disease. It is characterized by elevated levels of low-density lipoprotein cholesterol (LDL-C), increased triglycerides, or decreased levels of high-density lipoprotein cholesterol (HDL-C).

Elevated LDL-C plays a crucial role in the development of atherosclerosis by promoting lipid deposition within arterial walls, leading to plaque formation and vascular obstruction. [1,2] Cardiovascular diseases remain the leading cause of mortality worldwide, accounting for millions of deaths annually. Reduction of LDL cholesterol has been shown to significantly decrease the risk of cardiovascular events. Consequently, pharmacological agents that effectively lower LDL-C are essential in the management of dyslipidemia. [3]

Statins are the most widely prescribed lipid-lowering drugs and function by inhibiting the enzyme 3-hydroxy-3-methylglutaryl coenzyme A

(HMG-CoA) reductase, which plays a critical role in cholesterol biosynthesis in the liver. Inhibition of this enzyme leads to decreased hepatic cholesterol synthesis and increased expression of LDL receptors on hepatocytes, resulting in enhanced clearance of LDL cholesterol from circulation. [4]

Among available statins, atorvastatin and rosuvastatin are commonly used due to their potent lipid-lowering properties and favorable safety profiles. Atorvastatin is a lipophilic statin with strong LDL-lowering ability, while rosuvastatin is a hydrophilic statin with higher potency and longer half-life. [5]

Clinical studies have shown that rosuvastatin may produce greater reductions in LDL cholesterol compared with atorvastatin. A meta-analysis of randomized trials demonstrated that rosuvastatin significantly reduced LDL-C levels more effectively than atorvastatin, with a weighted mean difference of approximately −7 mg/dl ($p<0.0001$). [6] Similarly, comparative clinical studies have reported approximately 50% LDL reduction with rosuvastatin compared with about 40% reduction with atorvastatin after treatment. [7]

Another observational study also reported that rosuvastatin produced greater reductions in LDL cholesterol and improved attainment of LDL targets compared with atorvastatin therapy. [8,9] Despite these findings, both statins remain widely used in clinical practice, and comparative evidence from real-world settings remains valuable for guiding treatment decisions.

Therefore, the present study was conducted to compare the efficacy of atorvastatin and rosuvastatin in reducing LDL cholesterol levels in patients with dyslipidemia.

Materials and Methodology

Study Design: The present study was conducted as a prospective comparative observational study to evaluate and compare the efficacy of atorvastatin and rosuvastatin in reducing low-density lipoprotein cholesterol (LDL-C) levels in patients diagnosed with dyslipidemia.

Study Setting: The study was carried out in the Department of General Medicine at a tertiary care teaching hospital. Patients attending the outpatient department (OPD) for routine evaluation and management of dyslipidemia were screened for eligibility.

Study Duration: The study was conducted over a period of 12 months, including patient recruitment, treatment, follow-up, and data analysis.

Study Population: A total of 200 patients diagnosed with dyslipidemia were included in the study after fulfilling the inclusion and exclusion criteria.

Sample Size: The sample size for the study was 200 patients, divided equally into two groups of 100 patients each. This sample size provided adequate statistical power to detect clinically significant differences in LDL cholesterol reduction between the two treatment groups.

Sampling Technique: Patients were selected using a simple random sampling technique from those attending the outpatient department and meeting the eligibility criteria.

Inclusion Criteria

Patients fulfilling the following criteria were included in the study:

- Patients aged 30–70 years
- Diagnosed cases of dyslipidemia based on lipid profile
- LDL cholesterol level greater than 130 mg/dL
- Patients who were not receiving statin therapy previously
- Patients willing to provide written informed consent

Exclusion Criteria

Patients meeting any of the following criteria were excluded from the study:

- Patients with severe hepatic disease or abnormal liver function tests
- Patients with renal impairment
- Pregnant or lactating women
- Patients with hypersensitivity to statins
- Patients already receiving lipid-lowering medications
- Patients with uncontrolled hypothyroidism or other metabolic disorders affecting lipid levels

Study Groups

The selected patients were randomly divided into two equal groups:

Group	Treatment	Sample Size
Group A	Atorvastatin 20 mg once daily	100
Group B	Rosuvastatin 10 mg once daily	100

Both medications were administered orally once daily at night for a period of 12 weeks.

Baseline Assessment

At the time of enrollment, a detailed clinical history and physical examination were performed for each patient. The following information was recorded:

- Age
- Gender
- Body mass index (BMI)
- Medical history including hypertension, diabetes mellitus, and smoking status
- Family history of cardiovascular disease

Laboratory Investigations

Baseline laboratory investigations included:

- Total cholesterol (TC)
- Low-density lipoprotein cholesterol (LDL-C)
- High-density lipoprotein cholesterol (HDL-C)
- Triglycerides (TG)
- Liver function tests (LFTs)
- Fasting blood glucose

Blood samples were collected after 12 hours of overnight fasting.

Follow-Up Assessment: Patients were followed up for 12 weeks after initiation of therapy. At the end of the study period, lipid profile parameters were reassessed, including:

- Total cholesterol
- LDL cholesterol
- HDL cholesterol
- Triglycerides

Compliance with medication and any adverse effects were also recorded during follow-up visits.

Outcome Measures

The primary outcome measure of the study was:

- Reduction in LDL cholesterol levels after 12 weeks of therapy

Secondary outcome measures included:

- Percentage reduction in LDL cholesterol
- Achievement of target LDL levels (<100 mg/dL)
- Changes in other lipid parameters (total cholesterol, HDL, triglycerides)

Data Collection Procedure: All relevant clinical and laboratory data were recorded in a structured case record form prepared specifically for the study. Each patient was assigned a unique identification number to maintain confidentiality.

Statistical Analysis:

The collected data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 26.

- Comparison between groups was performed using the Student's t-test for continuous variables
- The Chi-square test was used for categorical variables
- A p-value less than 0.05 was considered statistically significant

Results

This was a prospective comparative study conducted in the Department of Medicine, tertiary care hospital in a duration of 12 months. A total of 200 patients diagnosed with dyslipidemia were included.

Table 1: Age Distribution of Study Participants

Age Group (years)	Atorvastatin (n=100)	Rosuvastatin (n=100)	p-value
30–40	18 (18%)	16 (16%)	
41–50	34 (34%)	36 (36%)	
51–60	30 (30%)	32 (32%)	
>60	18 (18%)	16 (16%)	0.91

Table 1 shows the age distribution of patients in both groups. The majority of patients were in the 41–50 year age group, accounting for 34% in the atorvastatin group and 36% in the rosuvastatin group. Patients aged 51–60 years constituted 30% and 32% in the atorvastatin and rosuvastatin groups respectively. The difference in age distribution between the two groups was not statistically significant (p=0.91), indicating that both groups were comparable at baseline.

Table 2: Changes in LDL Cholesterol Levels

Parameter	Atorvastatin	Rosuvastatin	p-value
Baseline LDL (mg/dl)	165 ± 20	167 ± 22	0.56
LDL after treatment	102 ± 18	86 ± 16	<0.001
% LDL reduction	38.2%	48.6%	<0.001

Table 2 demonstrates the changes in LDL cholesterol levels after 12 weeks of treatment. Baseline LDL levels were similar in both groups (165 mg/dl vs 167 mg/dl). After treatment, LDL levels decreased to 102 mg/dl in the atorvastatin group and 86 mg/dl in the rosuvastatin group. The percentage reduction in LDL cholesterol was significantly higher in the rosuvastatin group

(48.6%) compared with the atorvastatin group (38.2%). This difference was statistically significant ($p<0.001$), indicating that rosuvastatin is more effective in lowering LDL cholesterol.

Table 3: Achievement of Target LDL Levels (<100 mg/dl)

LDL Target	Atorvastatin (n=100)	Rosuvastatin (n=100)	p-value
<100 mg/dl	56 (56%)	74 (74%)	0.008
≥100 mg/dl	44 (44%)	26 (26%)	

Table 3 shows the proportion of patients who achieved the recommended LDL cholesterol target of <100 mg/dl after treatment.

In the atorvastatin group, 56% of patients achieved the target LDL level, whereas 74% of patients in the rosuvastatin group achieved the target. The difference was statistically significant ($p=0.008$), suggesting that rosuvastatin therapy results in better attainment of LDL cholesterol targets.

Discussion

Dyslipidemia is one of the most important modifiable risk factors for cardiovascular disease and is strongly associated with the development of atherosclerosis. Elevated low-density lipoprotein cholesterol (LDL-C) plays a central role in plaque formation and progression of coronary artery disease. Statins are widely used as first-line pharmacological agents for the management of dyslipidemia due to their ability to inhibit HMG-CoA reductase, thereby reducing hepatic cholesterol synthesis and increasing LDL receptor activity.

The present study compared the efficacy of atorvastatin and rosuvastatin in reducing LDL cholesterol levels in patients with dyslipidemia. Our findings demonstrated that both statins significantly reduced LDL cholesterol levels after treatment; however, rosuvastatin showed a greater percentage reduction in LDL cholesterol compared with atorvastatin.

In the present study, rosuvastatin produced a higher reduction in LDL cholesterol levels compared with atorvastatin. These findings are consistent with the results of several previous clinical studies that reported superior lipid-lowering efficacy of rosuvastatin. A systematic review and meta-analysis comparing the lipid-lowering effects of these two statins demonstrated that rosuvastatin resulted in greater reductions in LDL cholesterol and triglyceride levels and greater increases in HDL cholesterol compared with atorvastatin. [10]

Similarly, another meta-analysis involving multiple randomized controlled trials found that rosuvastatin therapy resulted in significantly greater LDL cholesterol reduction compared with atorvastatin. The weighted mean difference in LDL reduction between the two drugs was approximately −7 mg/dL, indicating a superior effect of rosuvastatin. [11] The greater lipid-lowering efficacy of rosuvastatin may be attributed to its higher potency and stronger binding affinity for HMG-CoA reductase. Rosuvastatin is also relatively hydrophilic and demonstrates greater hepatic selectivity, which enhances its cholesterol-lowering effect. Studies have shown that rosuvastatin can produce substantial LDL reductions even at lower doses compared with atorvastatin. [12]

In the present study, a higher proportion of patients treated with rosuvastatin achieved the recommended LDL cholesterol target of less than 100 mg/dl compared with those receiving atorvastatin therapy. This finding is consistent with results reported in previous clinical trials. In one comparative clinical trial, rosuvastatin therapy achieved approximately 50% reduction in LDL cholesterol, whereas atorvastatin produced about 40% reduction, and a significantly larger proportion of patients treated with rosuvastatin achieved target LDL levels. [13]

A randomized clinical study conducted among patients with dyslipidemia also demonstrated greater LDL cholesterol reduction with rosuvastatin compared with atorvastatin after 16 weeks of treatment. In that study, rosuvastatin achieved approximately 45.1% LDL reduction compared with 33.9% with atorvastatin, indicating significantly greater efficacy of rosuvastatin in lipid lowering. [8,14]

Long-term clinical studies have also confirmed the superior lipid-lowering effect of rosuvastatin. A large comparative trial evaluating rosuvastatin and atorvastatin over 52 weeks demonstrated that rosuvastatin achieved greater reductions in LDL cholesterol and enabled more patients to reach recommended LDL targets compared with atorvastatin therapy. [15]

Similarly, studies conducted in patients with familial hypercholesterolemia showed that rosuvastatin produced significantly greater reductions in LDL cholesterol compared with atorvastatin, along with improvements in other lipid parameters such as apolipoprotein B and HDL cholesterol. [9] Despite these differences in LDL-lowering potency, both statins have been shown to be highly effective in reducing cardiovascular risk. Atorvastatin remains one of the most commonly prescribed statins worldwide and has demonstrated significant benefits in reducing cardiovascular morbidity and mortality in large clinical trials. Its

lipid-lowering effect is well established, and it is particularly useful in patients requiring moderate-to-high intensity statin therapy.

Another important consideration in statin therapy is the safety profile of the drugs. In general, both atorvastatin and rosuvastatin are well tolerated. The most commonly reported adverse effects include mild gastrointestinal symptoms, headache, and myalgia. Clinical studies have shown that the incidence of adverse events is similar between the two statins, and both drugs are considered safe for long-term use in most patients. [11]

The differences in lipid-lowering efficacy between these statins may also be explained by pharmacokinetic characteristics. Rosuvastatin has a longer half-life and greater potency compared with atorvastatin, which allows effective LDL reduction at lower doses. In contrast, atorvastatin is more lipophilic and undergoes extensive hepatic metabolism through the cytochrome P450 system.

Another advantage of rosuvastatin observed in several studies is its ability to achieve high-intensity statin effects at relatively lower doses compared with atorvastatin. Clinical guidelines classify rosuvastatin as a high-intensity statin at doses of 20–40 mg, whereas atorvastatin requires higher doses (40–80 mg) to achieve similar LDL reductions. [14]

However, some studies have suggested that both statins may have comparable efficacy when used at equivalent doses. In certain clinical settings, atorvastatin has shown similar reductions in LDL cholesterol and triglycerides as rosuvastatin, indicating that the choice of statin may depend on individual patient characteristics and treatment goals. [12]

Overall, the findings of the present study are consistent with the majority of published literature indicating that rosuvastatin is generally more potent than atorvastatin in lowering LDL cholesterol levels. Nevertheless, both drugs remain effective options for the management of dyslipidemia and prevention of cardiovascular disease. The present study had certain limitations. The duration of follow-up was relatively short, and long-term cardiovascular outcomes were not assessed. Additionally, other lipid parameters such as triglycerides and HDL cholesterol were not extensively analyzed in comparison with LDL cholesterol.

Future studies with larger sample sizes and longer follow-up periods are needed to further evaluate the long-term cardiovascular benefits and safety profiles of these statins.

Conclusion

The present study demonstrated that both atorvastatin and rosuvastatin significantly reduce LDL cholesterol levels in patients with dyslipidemia. However, rosuvastatin showed significantly greater LDL reduction and better achievement of target LDL levels compared with atorvastatin.

These findings suggest that rosuvastatin may be considered a more potent and effective statin for managing dyslipidemia, particularly in patients who require aggressive LDL reduction.

Further long-term studies are recommended to evaluate the impact of these statins on cardiovascular outcomes.

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