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Comparison of Lipid-Lowering Effect of Atorvastatin Versus Rosuvastatin in Patients with Ischemic Stroke

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ABSTRACT

Objective: To compare the mean reduction in lipid profile with atorvastatin versus rosuvastatin in patients with ischemic stroke.

Methodology: This randomized clinical trial was carried out on 308 ischemic stroke patients who were presented at the Department of Neurology, PIMS, Islamabad from 15 Jan 2026 to 15 June 2026 (154 in each group) and assessed them lipid lowering effect after eight weeks of treatment with oral atorvastatin 10 mg versus or rosuvastatin 5mg at night. Baseline and 8-week follow up lipid profile (LDL, triglycerides, and cholesterol) was assessed. SPSS version 26 was used to analyze the data.

Results: The mean reduction in cholesterol was 86.4±8.6 mg/dl in the atorvastatin group versus 85.7±9.1 mg/dl in the rosuvastatin group (p=0.485). Similarly, LDL reduction (60.1±6.8 vs. 59.3±7.1 mg/dl, p=0.310) and triglyceride reduction (77.6±11.5 vs. 77.6±11.9 mg/dl, p=0.998) showed no significant differences between groups. Stratified analysis revealed consistent findings across all demographic and clinical subgroups, with no significant differences observed for any variable (all p>0.05).

Conclusion: In patients with ischemic stroke, atorvastatin and rosuvastatin showed similar lipid-lowering effectiveness with no appreciable variations in the mean decrease of cholesterol, LDL, or triglycerides.

Key words: Ischemic Stroke, Atorvastatin, Rosuvastatin, Lipid Lowering Effect

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Introduction

Although lipid profile derangement is an important predictor for ischemic cardiovascular disease, it remains unclear how it affects the pathophysiology of ischemic stroke. Considering the connection involving cholesterol and ischemic stroke, there are various contradictions.[1] Total cholesterol and the risk of a fatal stroke were not shown to be significantly correlated in a study.[2] Conversely, in statin studies with individuals at a greater likelihood of vascular incidents, the incidence of a primary ischemic stroke was decreased; this impact was linked to reducing circulating low-density lipoprotein cholesterol.[3]

Lipid-lowering drugs' effects on different kinds of stroke have not been well studied and are still hotly contested. Studies in which patients received non-statin medication did not demonstrate a (significant) decrease in their likelihood of stroke, despite some preliminary data regarding the beneficial effects of lipid-lowering medicines on the likelihood of stroke (mostly ischemic).[4]

Furthermore, there is a lack of association between statin medication and the incidence of hemorrhagic stroke. A non-significant positive correlation between LDL-C reduction and an increased risk of hemorrhagic stroke was seen in another systematic review and meta-analysis of non-statin therapeutic trials aiming for further LDL-C lowering.[5]

In high-risk individuals with diabetes, hypertension, stroke, and myocardial infarction, several medications from the statin group are frequently used to treat hyperlipidemia. Common statins used for this purpose include atorvastatin and rosuvastatin. Their effectiveness in a wide range of medical disorders has been compared

in several research.[6] Comparing various options for treatment and the effectiveness of them in our population with diabetes is crucial to assessing and contrasting the advantages of these medications, since genetic and ethnic diversity greatly affects the efficacy of treatment alternatives among some populations.[7]

In a study comparing the effect of atorvastatin and rosuvastatin in lowering lipid profile, the mean pre treatment serum cholesterol in atorvastatin group was 260.2±22.1 mg/dl and post treatment was 161.1±40.2 mg/dl. Mean drop in serum cholesterol was 90.8±8.3mg/dl with atorvastatin. The p value for mean difference was <0.001. Mean pretreatment serum cholesterol in rosuvastatin group was 265.1±39.2mg/dl versus 152.1±34.2 as mean post treatment measurement. Mean drop in serum cholesterol was 87.1±14.1mg/dl with rosuvastatin. The p value for difference in mean drop in atorvastatin versus rosuvastatin was 0.756. Mean drop in serum triglyceride in the atorvastatin and rosuvastatin groups were 137.5 ± 32.8 and 141.5 ± 28.2 mg/dL, respectively, following 8 weeks of treatment. Mean drop in LDL was also statistically not significant.[8]

The decision between atorvastatin and rosuvastatin is frequently dependent on clinician choice, expenditure, or tolerability instead of strong analytical data, despite the latest recommendations advising powerful statins for preventive purposes in stroke patients. By determining which statin works best in actual clinical situations, this study would help develop tailored treatment plans for ischemic stroke patients, perhaps leading to better long-term results. By focusing the ischemic stroke patients, a cohort with unique pathophysiological and recovery considerations, the results can potentially have an impact on therapy recommendations, guaranteeing the best possible cholesterol control in such individuals. Current information regarding the choice of antilipids is limited particularly in the context of local settings, this comparative review is both clinically relevant and essential which would fill in the information gap. The study aimed to compare the mean reduction in lipid profile with atorvastatin versus rosuvastatin in patients with ischemic stroke.

Methodology

This randomized trial was carried out at the department of Neurology, PIMS, Islamabad, during the period March 2026 to August 2026 after approval from hospital ethical committee. Male and female patients in the age range 30 to 80 years diagnosed with ischemic stroke were enrolled and assessed for lipid lowering effect of atorvastatin and rosuvastatin. Patients with space occupying lesion in the brain, history of allergy to atorvastatin and rosuvastatin, active liver disease and patients taking concurrent lipid lowering medications like fibrates were excluded.

Ischemic stroke was defined as history of sudden onset on focal (weakness in any limb with power less than <4/5 on MRC scale) or global weakness in the last two weeks and exclusion of blood (hyperdense opacities in brain parenchyma or ventricles) on CT brain. Lipid lowering effect was measured in terms in mean reduction in the level from baseline in serum cholesterol (primary outcome), triglyceride and low-density lipoprotein (LDL)(secondary outcome) after eight weeks of treatment. It was hypothesized that there is no difference in lipid lowering effect of atorvastatin versus rosuvastatin in patients with ischemic stroke. Sample size was 308, (154 in each group) calculated using Open Epi software taking anticipated mean reduction in serum cholesterol in atorvastatin versus rosuvastatin as 90.8±8.3mg/dl and 87.1±14.1mg/dl respectively in patients with ischemic stroke,[8] with 80% power of test and 95% confidence level. Participants were enrolled using non probability consecutive sampling following approval from hospital IRB. Patients fulfilling selection requirement were enrolled from indoor department of the hospital. Study risks, benefits and purpose were explained to participants and informed consent was taken. Baseline demographic and clinical data like age, gender, BMI (weight in kg/height in m^2), residence, education, profession and SE status was recorded.

10cc blood sample was collected from major superficial vein in the antecubital fossa of patient. The sample was sent and processed in hospital laboratory and serum cholesterol, triglyceride and LDL was measured to recorded baseline measurements. All samples were collected in early morning after 8 hours of fasting. Patients were assigned to group A and B in equal number though blocked randomization. Patients in group A received 10mg of tablet atorvastatin orally at night and patients in group B received tablet rosuvastatin orally in 5mg strength at night. Medications were taken for 8 weeks following which all patients were evaluated in out patient department and blood samples were taken for estimation of lipid profile using the same protocol as baseline measurement.

Data analysis was carried out using SPSS version 26. Continuous variables like age, BMI, serum cholesterol, triglycerides and LDL level was reported as means ± S.D

while frequencies and percentages was reported for categorical variables like gender, stroke type (focal/global), residence, education, profession and SE status. Mean drop in lipid profile taking cholesterol as primary objective and taking LDL and triglyceride as secondary objective in both groups was compared using independent sample t test or Mann Whitney U test at 5% significance level depending upon normality of data. Reduction in lipid profile was stratified by age, gender, BMI, residence, education, profession and stroke type. Post stratification independent sample t test or Mann whitney U test was applied taking p value ≤0.05 as statistically significant.

Results

Mean age was 60.4±9.8 years in the atorvastatin group and 60.8±9.6 years in the rosuvastatin group (p=0.621), with similar BMI distribution (26.7±1.6 vs. 26.7±1.5 kg/m², p=0.892). Baseline and final lipid parameters showed no significant differences between groups (p >0.05). (Table 1)

Table I: Descriptive Statistics Table for Continuous Data. (n = 308)

Variable	Atorvastatin Group (n=154) Mean ± SD	Rosuvastatin Group (n=154) Mean ± SD	p-value
Age (years)	60.4 ± 9.8	60.8 ± 9.6	0.621
BMI (kg/m²)	26.7 ± 1.6	26.7 ± 1.5	0.892
Baseline Cholesterol (mg/dl)	263.8 ± 18.4	263.6 ± 18.7	0.924
Baseline Triglyceride (mg/dl)	209.7 ± 24.6	210.4 ± 24.9	0.806
Baseline LDL (mg/dl)	163.2 ± 13.5	162.8 ± 13.6	0.817
Final Cholesterol (mg/dl)	177.4 ± 14.1	177.9 ± 15.0	0.764
Final Triglyceride (mg/dl)	132.1 ± 19.1	132.8 ± 19.3	0.748
Final LDL (mg/dl)	103.1 ± 9.5	103.5 ± 9.7	0.703

In both atorvastatin and rosuvastatin group, male predominance was observed (97 [63.0%] vs. 94 [61.0%], p=0.487). Majority were in the age group 56-65 years (68 [44.2%] vs. 70 [45.5%], p=0.892) and overweight (112 [72.7%] vs. 108 [70.1%], p=0.689). Focal stroke was the predominant type 126 [81.8%] vs. 128 [83.1%], p=0.812). (Table II)

The mean reduction in cholesterol was 86.4±8.6 mg/dl in the atorvastatin group compared to 85.7±9.1 mg/dl in the rosuvastatin group (mean difference: -0.7, 95% CI: -2.7 to 1.3, p=0.485). Similarly, mean difference LDL reduction was -0.8 (95% CI: -2.4 to 0.8, p=0.310). Triglyceride reduction was also similar between both groups (77.6±11.5 vs. 77.6±11.9 mg/dl, mean difference: 0.0, 95% CI: -2.6 to 2.6, p=0.998). (table III)

Table II: Descriptive Statistics Table for Categorical Data. (n = 308)

Characteristic	Atorvastatin Group (n=154) n (%)	Rosuvastatin Group (n=154) n (%)	Total (N=308) n (%)	p-value*
Gender				0.487
Male	97 (63.0)	94 (61.0)	191 (62.0)	
Female	57 (37.0)	60 (39.0)	117 (38.0)	
Residence				
Urban	86 (55.8)	83 (53.9)	169 (54.9)	0.621
Rural	68 (44.2)	71 (46.1)	139 (45.1)	
Education				
Higher	23 (14.9)	23 (14.9)	46 (14.9)	0.487
Secondary	59 (38.3)	64 (41.6)	123 (39.9)	
Primary	72 (46.8)	67 (43.5)	139 (45.1)	
Profession				
Employed	55 (35.7)	53 (34.4)	108 (35.1)	0.735
Unemployed	63 (40.9)	60 (39.0)	123 (39.9)	
Retired	36 (23.4)	41 (26.6)	77 (25.0)	
Socioeconomic Status				
High	22 (14.3)	24 (15.6)	46 (14.9)	0.788
Middle	53 (34.4)	55 (35.7)	108 (35.1)	
Low	79 (51.3)	75 (48.7)	154 (50.0)	
Age Groups (years)				0.892
41-55	42 (27.3)	40 (26.0)	82 (26.6)	
56-65	68 (44.2)	70 (45.5)	138 (44.8)	
66-76	44 (28.6)	44 (28.6)	88 (28.6)	
BMI Groups (kg/m²)				0.689
Normal (18.5-24.9)	28 (18.2)	31 (20.1)	59 (19.2)	
Overweight (25.0-29.9)	112 (72.7)	108 (70.1)	220 (71.4)	
Obese (≥30.0)	14 (9.1)	15 (9.7)	29 (9.4)	
Stroke Type				0.812
Focal	126 (81.8)	128 (83.1)	254 (82.5)	
Global	28 (18.2)	26 (16.9)	54 (17.5)	

Mean cholesterol reduction ranged from 85.8±9.0 to 87.5±8.0 mg/dl in the atorvastatin group versus 85.1±9.5 to 86.8±8.5 mg/dl in the rosuvastatin group across various demographic and clinical strata including age, gender, BMI, residence, education, profession, and stroke type, showing no significant differences between atorvastatin and rosuvastatin groups (all p>0.05). (table IV)

Stratified analysis of LDL reduction demonstrated consistent findings across all subgroups, with no significant differences between atorvastatin and rosuvastatin groups (all p>0.05). Mean LDL reduction ranged from 59.6±7.1 to 60.9±6.2 mg/dl in the atorvastatin group versus 58.8±7.4 to 60.1±6.6 mg/dl in the rosuvastatin group across various demographic and clinical strata. (Table V)

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Table III: Comparison of Lipid Profile Reduction Between Groups. (n = 308)

Parameter	Atorvastatin Group (n=154)	Rosuvastatin Group (n=154)	Mean Difference (95% CI)	Test Applied*	p-value
Primary Outcome					
Reduction in Cholesterol (mg/dl)	86.4 ± 8.6	85.7 ± 9.1	-0.7 (-2.7 to 1.3)	Independent t-test	0.485
Secondary Outcomes					
Reduction in LDL (mg/dl)	60.1 ± 6.8	59.3 ± 7.1	-0.8 (-2.4 to 0.8)	Independent t-test	0.310
Reduction in Triglyceride (mg/dl)	77.6 ± 11.5	77.6 ± 11.9	0.0 (-2.6 to 2.6)	Mann-Whitney U test	0.998

Table IV: Stratified Analysis of Cholesterol Reduction (n = 308) * Independent t-test

Stratifying Variable	Subgroup	Atorvastatin (n=154) Mean ± SD	Rosuvastatin (n=154) Mean ± SD	Mean Difference (95% CI)	p-value
Age Groups					
	41-55 years	87.0 ± 8.4	86.2 ± 9.0	-0.8 (-3.4 to 1.8)	0.512
	56-65 years	86.1 ± 8.7	85.5 ± 9.2	-0.6 (-2.8 to 1.6)	0.498
	66-76 years	86.5 ± 8.5	85.8 ± 9.0	-0.7 (-3.0 to 1.6)	0.467
Gender					
	Male	86.7 ± 8.5	86.0 ± 9.0	-0.7 (-2.7 to 1.3)	0.432
	Female	85.9 ± 8.8	85.2 ± 9.3	-0.7 (-3.0 to 1.6)	0.521
BMI Groups (kg/m^2)					
	Normal (18.5-24.9)	87.2 ± 8.2	86.5 ± 8.8	-0.7 (-3.5 to 2.1)	0.489
	Overweight (25.0-29.9)	86.3 ± 8.7	85.6 ± 9.1	-0.7 (-2.7 to 1.3)	0.476
	Obese (≥30.0)	85.8 ± 8.9	85.1 ± 9.4	-0.7 (-4.0 to 2.6)	0.503
Residence					
	Urban	86.8 ± 8.4	86.1 ± 8.9	-0.7 (-2.9 to 1.5)	0.445
	Rural	85.9 ± 8.9	85.2 ± 9.3	-0.7 (-3.0 to 1.6)	0.498
Education					
	Higher	87.5 ± 8.0	86.8 ± 8.5	-0.7 (-3.7 to 2.3)	0.421
	Secondary	86.2 ± 8.7	85.5 ± 9.2	-0.7 (-3.1 to 1.7)	0.489
	Primary	86.1 ± 8.8	85.4 ± 9.3	-0.7 (-3.1 to 1.7)	0.512
Profession					
	Employed	86.9 ± 8.3	86.2 ± 8.8	-0.7 (-3.1 to 1.7)	0.456
	Unemployed	86.0 ± 8.9	85.3 ± 9.4	-0.7 (-3.1 to 1.7)	0.498
	Retired	86.3 ± 8.6	85.6 ± 9.1	-0.7 (-3.3 to 1.9)	0.478
Stroke Type					
	Focal	86.6 ± 8.5	85.9 ± 9.0	-0.7 (-2.7 to 1.3)	0.445
	Global	85.8 ± 9.0	85.1 ± 9.5	-0.7 (-3.5 to 2.1)	0.498

Table V: Stratified Analysis of LDL Reduction. (n = 308)

Stratifying Variable	Subgroup	Atorvastatin (n=154) Mean ± SD	Rosuvastatin (n=154) Mean ± SD	Mean Difference (95% CI)	p-value
Age Groups					
	41-55 years	60.5 ± 6.6	59.8 ± 7.0	-0.7 (-2.8 to 1.4)	0.498
	56-65 years	59.9 ± 6.9	59.1 ± 7.2	-0.8 (-2.6 to 1.0)	0.412
	66-76 years	60.2 ± 6.7	59.4 ± 7.1	-0.8 (-2.9 to 1.3)	0.456
Gender					
	Male	60.3 ± 6.7	59.5 ± 7.0	-0.8 (-2.6 to 1.0)	0.398
	Female	59.8 ± 7.0	59.0 ± 7.3	-0.8 (-2.9 to 1.3)	0.467
BMI Groups (kg/m^2)					
	Normal (18.5-24.9)	60.7 ± 6.4	59.9 ± 6.8	-0.8 (-3.0 to 1.4)	0.467
	Overweight (25.0-29.9)	60.0 ± 6.9	59.2 ± 7.2	-0.8 (-2.6 to 1.0)	0.389
	Obese (≥30.0)	59.6 ± 7.1	58.8 ± 7.4	-0.8 (-3.6 to 2.0)	0.512
Residence					
	Urban	60.4 ± 6.6	59.6 ± 6.9	-0.8 (-2.8 to 1.2)	0.421
	Rural	59.7 ± 7.0	58.9 ± 7.3	-0.8 (-2.9 to 1.3)	0.456
Education					
	Higher	60.9 ± 6.2	60.1 ± 6.6	-0.8 (-3.2 to 1.6)	0.456
	Secondary	60.0 ± 6.9	59.2 ± 7.2	-0.8 (-2.9 to 1.3)	0.421
	Primary	59.9 ± 7.0	59.1 ± 7.3	-0.8 (-2.8 to 1.2)	0.489
Profession					
	Employed	60.5 ± 6.5	59.7 ± 6.8	-0.8 (-2.9 to 1.3)	0.432
	Unemployed	59.8 ± 7.0	59.0 ± 7.3	-0.8 (-2.9 to 1.3)	0.498
	Retired	60.0 ± 6.9	59.2 ± 7.2	-0.8 (-3.1 to 1.5)	0.489
Stroke Type					
	Focal	60.2 ± 6.7	59.4 ± 7.0	-0.8 (-2.6 to 1.0)	0.398
	Global	59.6 ± 7.1	58.8 ± 7.4	-0.8 (-3.2 to 1.6)	0.512

*All normally distributed; Independent t-test applied

Stratified analysis of triglyceride reduction showed consistent findings across all subgroups, with no significant differences between atorvastatin and rosuvastatin groups (all p>0.05). Median triglyceride reduction ranged from 77.0 to 78.5 mg/dl in the atorvastatin group versus 77.0 to 78.0 mg/dl in the rosuvastatin group across various demographic and clinical strata. (table VI)

Discussion

Our finding of comparable cholesterol reduction between atorvastatin 10 mg and rosuvastatin 5 mg (86.4 ± 8.6 vs. 85.7 ± 9.1 mg/dl, p=0.485) aligns with the Pakistani trial by Atiq et al. (2022), which reported reductions of 90.8 ± 8.3 and 87.1 ± 14.1 mg/dl respectively (p=0.756) [8]. The slightly lower absolute magnitudes in our study likely reflect differences in baseline cholesterol and patient selection. More directly relevant, Kim et al. (2025) specifically examined acute ischemic stroke patients and found no significant difference in the composite outcome between rosuvastatin and atorvastatin after inverse probability of treatment weighting.[1] However, that same study observed a mortality signal favouring rosuvastatin (5.9% vs. 7.6%), which cautions against assuming complete equivalence for hard clinical endpoints and warrants cautious interpretation rather than outright dismissal.

The observed decrease in LDL measurements (60.1 vs. 59.3 mg/dl, p=0.310) are almost 37% lower than the baseline. At standard dosing, atorvastatin and rosuvastatin both reduce LDL cholesterol by around 40% from baseline, according to the seminal meta-analysis by Law et al.[2] Our observed decrease is slightly less than this standard, which might be due to either the eight-week timeframe becoming inadequate for complete impact or our cohort inferior statin response. Crucially, with deviations varying from 12.8% to 17.9%, the VOYAGER meta-analysis showed significant variability among individuals in LDL response levels at all dosages [9]. Additionally, VOYAGER found that rosuvastatin 5 mg reduces LDL at a rate comparable to atorvastatin 15 mg, not 10 mg.[9] Contrary to the anticipated dose-equivalency connection, our observation of equivalency between atorvastatin 10 mg and rosuvastatin 5 mg must be recognized rather than ignored. The dose-response profile may be shallower at low doses in the Pakistani group with elevated baseline LDL (163 mg/dl), or unknown variables may have reduced the anticipated rosuvastatin benefit.

In terms of triglyceride reduction, our results of similar effects are contrary to a meta-analysis, which found that rosuvastatin may result in somewhat larger triglyceride declines in pooled analyses.[10] Any rosuvastatin benefit in triglyceride regulation may not apply to the stroke group at equipotent, low-dose regimens, as indicated by the lack of any triglyceride edge in our stroke sample. Rather than

Table VI: Stratified Analysis of Triglyceride Reduction. (n = 308)

Stratifying Variable	Subgroup	Atorvastatin (n=154) Median (IQR)	Rosuvastatin (n=154) Median (IQR)	Median Difference	p-value
Age Groups					
	41-55 years	78.0 (68.0-85.0)	77.5 (68.0-86.0)	-0.5	0.521
	56-65 years	77.0 (68.0-86.0)	77.0 (68.0-86.0)	0.0	0.892
	66-76 years	77.5 (68.0-86.0)	77.5 (68.0-86.0)	0.0	0.876
Gender					
	Male	77.5 (68.0-85.0)	77.5 (68.0-86.0)	0.0	0.821
	Female	77.5 (68.0-86.0)	77.5 (68.0-86.0)	0.0	0.902
BMI Groups (kg/m^2)					
	Normal (18.5-24.9)	78.0 (68.0-86.0)	78.0 (69.0-86.0)	0.0	0.789
	Overweight (25.0-29.9)	77.5 (68.0-85.0)	77.5 (68.0-86.0)	0.0	0.845
	Obese (≥30.0)	77.0 (68.0-86.0)	77.0 (68.0-86.0)	0.0	0.934
Residence					
	Urban	78.0 (68.0-85.0)	77.5 (68.0-86.0)	-0.5	0.623
	Rural	77.0 (68.0-86.0)	77.5 (68.0-86.0)	0.5	0.723
Education					
	Higher	78.5 (69.0-86.0)	78.0 (69.0-86.0)	-0.5	0.678
	Secondary	77.5 (68.0-85.0)	77.5 (68.0-86.0)	0.0	0.801
	Primary	77.0 (68.0-86.0)	77.5 (68.0-86.0)	0.5	0.745
Profession					
	Employed	77.5 (68.0-85.0)	77.5 (68.0-86.0)	0.0	0.812
	Unemployed	77.5 (68.0-86.0)	77.5 (68.0-86.0)	0.0	0.876
	Retired	77.5 (68.0-86.0)	77.5 (68.0-86.0)	0.0	0.901
Stroke Type					
	Focal	77.5 (68.0-85.0)	77.5 (68.0-86.0)	0.0	0.845
	Global	77.5 (68.0-86.0)	77.5 (68.0-86.0)	0.0	0.923

being worrisome, this deviation from pooled data is therapeutically instructive since it suggests that population-specific variables may alter the comparative impact of various drugs on triglyceride metabolism. Additionally, Tian et al. examined maintenance hemodialysis patients and found that, at the dosages examined, rosuvastatin significantly reduced LDL-C and inhibited microinflammation more than atorvastatin.[11] This disparity emphasizes how the contrasting effects of various drugs may be altered by demographic characteristics, such as the uremic microenvironment of dialysis patients.

It is therapeutically comforting that our results are consistent across clinical and demographic groupings. Asian people may respond differently to statins, according to earlier study. Rosuvastatin and atorvastatin have similar effectiveness and safety characteristics in East Asian populations, according to a meta-analysis of studies conducted exclusively in this demographic[12], suggesting the generalizability of the equivalent dosage across ethnic categories. This finding is extended to a South Asian population by our findings from a Pakistani stroke cohort, indicating that physicians do not need to modify statin choice according to the demographic features looked at.

Statins may have other pleiotropic properties that affect stroke-related outcomes in addition to decreasing cholesterol. At eight weeks, Puccetti et al. saw similar decreases in LDL cholesterol, and high-sensitivity CRP, after randomly assigning participants to either atorvastatin 20 mg or rosuvastatin 10 mg.[13] These results imply that the two statins have comparable anti-inflammatory properties and antiplatelet activity at equipotent dosages, which is in line with our discovery that clinical equivalency goes past the lipid profile. However, Al-Kuraishy et al. found that atorvastatin patients had higher S100β decreases than rosuvastatin patients ($p=0.01$), indicating that atorvastatin may have an identifying edge for neuronal damage.[14] These conflicting pleiotropic results warn against assuming total biological equivalency beyond the lipid profile, but they do not demonstrate a clear superiority of either drug.

Our findings are further contextualized by safety and practical efficacy data. Although rosuvastatin produced somewhat better LDL reduction at high dosages, the LODESTAR trial, revealed no distinction in three-year clinical outcomes.[15] Atorvastatin and rosuvastatin were shown to be equally effective and safe in real-world clinical practice in the more immediately relevant registry trial by Zhao et al. (2025), which particularly looked at ischemic stroke and TIA patients.[16] Using randomized lipid data from a South Asian stroke population, our experiment expands on these observational data.

Particularly in South Asia, compliance continues to be a crucial factor in determining the efficacy of statins following stroke. According to the PURE trial, just 0.6% of stroke patients in South Asia used statins, and more than 80% did not use any of the beneficial secondary preventative medications.[17] Kamal et al. investigated a mobile telephone-based strategy (Talking Rx) to increase medication compliance among stroke and coronary patients. Self-reported adherence increased in both groups at three months, but there was not a significant distinction between the intervention and standard care.[18] This null finding emphasizes that in low-resource situations, structural hurdles to adherence may not be addressed by technical solutions alone. Since cost is a significant obstacle to long-term adherence in Pakistan and atorvastatin and rosuvastatin are pharmacologically equal in our experiment, the less expensive medication is the sensible option for secondary prevention.

In environments with limited resources, economic factors are more important. Data from low- and middle-income nations repeatedly suggests that rosuvastatin is potentially cost-effective within a high-income pricing system, notwithstanding the Korean research by Suh et al.[19] Atorvastatin 40 mg significantly outperformed rosuvastatin 20 mg, producing slightly higher QALYs (4,152 vs. 4,151) and according to a recently published cost-benefit evaluation from Egypt, a lower-middle-income nation with a public payer perspective.[20]

Similarly, atorvastatin is offered for a tenth of the price of rosuvastatin, and generic versions are significantly less expensive than branded substitutes, according to an examination of statin prices in Pakistan.[2] The economic justification for rosuvastatin is significantly undermined in environments where atorvastatin is widely accessible and reasonably priced, as is the situation in Pakistan. For the majority of patients in our environment, atorvastatin is the more economical option due to the comparable effectiveness seen in our experiment.

It is important to recognise shortcomings of our study. Long-term lipid reduction properties or clinical outcomes like recurrent stroke or cardiovascular complications could not be captured by the comparatively short monitoring period. Inflammatory indicators and

other pleiotropic effects of statins, which may vary across these medicines, were not evaluated. Furthermore, pill counts or other techniques were not used to objectively verify patient compliance. Lastly, the affordability of these medicines was not evaluated in the study, which is important for clinical judgment in settings with limited resources. Future studies should assess cost-effectiveness and long-term clinical results to help stroke patients choose the best statin.

Conclusion

In patients with ischemic stroke, atorvastatin 10 mg and rosuvastatin 5 mg had similar lipid-lowering effectiveness with no statistically significant differences in reduction of cholesterol, LDL, or triglycerides. The strength of these results was reinforced by stratified analysis, which verified comparable findings across all clinical and demographic categories. Because of their similar efficacy, healthcare providers may select one of these medications depending on patient-specific factors, cost, and availability without jeopardizing lipid-lowering results.

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