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Original Article

Comparison of efficacy and safety between loading-dose atorvastatin and rosuvastatin in cerebral infarction

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Abstract: Objective: To analyze the efficacy and safety of loading-dose atorvastatin and rosuvastatin in the treatment of cerebral infarction (CI). Methods: A total of 151 CI patients treated at the Third Affiliated Hospital of Qiqihar Medical University from January 2015 to February 2020 retrospectively were selected and divided into four groups: conventional atorvastatin, loading-dose atorvastatin, conventional rosuvastatin, and loading-dose rosuvastatin. Primary outcomes assessed included changes in National Institutes of Health Stroke Scale (NIHSS) scores, clinical efficacy, alterations in serum lipid indices, liver function, inflammation markers, CI indices, and the incidence of adverse reactions. Results: After treatment, all groups showed a significant decrease in NIHSS scores (all $P<0.0001$). The loading-dose groups exhibited greater reductions in NIHSS scores compared to the conventional groups (both $P<0.0001$). No significant difference was found in NIHSS scores between the two loading-dose groups ($P>0.05$). The loading-dose groups demonstrated higher efficacy than the conventional groups (both $P<0.05$), with no significant difference between the two loading-dose groups (both $P>0.05$). Loading-dose rosuvastatin showed superior improvement in blood lipid control compared to loading-dose atorvastatin ($P<0.05$). There were no significant differences in liver function indices among the groups (all $P>0.05$). Inflammation and myocardial indices intensified 24 hours after treatment, with milder intensification in the loading-dose rosuvastatin group compared to the loading-dose atorvastatin group ($P<0.05$). The incidences of adverse reactions did not significantly differ among the groups (all $P>0.05$). Conclusion: Both loading-dose atorvastatin and rosuvastatin demonstrated increased clinical efficacy in the treatment of CI patients, ensuring safety and effectiveness. However, rosuvastatin exhibited superior efficacy in blood lipid control. These findings provide valuable guidance for the clinical management of CI.

Keywords: Loading dose, atorvastatin, rosuvastatin, cerebral infarction

Introduction

Dyslipidemia plays a critical role in the development of arteriosclerosis. The rupture of atherosclerotic plaques and subsequent blockage of cerebral vessels are the main causes of cerebral infarction (CI). Effective management of blood lipid levels is essential in reducing the occurrence or recurrence of CI [1, 2]. In addition to their lipid-regulating effects, statins possess other beneficial functions, such as anti-inflammatory and antioxidant properties, improvement of endothelial cell function, and enhancement of microcirculation. Among the various statins used in clinical practice, atorvastatin and rosuvastatin are the most widely prescribed [3].

Atorvastatin, a potent lipid-regulating drug, is commonly utilized for its ability to regulate blood lipid levels and improve microcirculation, ischemia, and hypoxia. It achieves these effects by directly acting on both cell membranes and within the nucleus [4]. Rosuvastatin, classified as a third-generation statin, is known for its potent efficacy. Statins significantly improve vascular endothelial function and exert strong inhibitory effects on the proliferation of vascu-

lar smooth muscle cells and platelet aggregation. These effects contribute to the prevention of thrombosis, effective stabilization of atherosclerotic plaques, and reduction of inflammation [5].

Recent research indicates that a treatment regimen involving a loading dose of statins can slow the progression of coronary atherosclerosis and, in some cases, even reverse the disease [6, 7].

Statin administration prior to and during hospitalization has been associated with the survival of ischemic stroke patients, suggesting a "dose-effect" relationship in statin usage. Specifically, the high-dose group (statin ≥60 mg/day) demonstrated greater benefits compared to the low-dose group (<60 mg/day) [8]. As first-line lipid-lowering drugs, statins are associated with adverse reactions, including elevated transaminase levels and the risk of rhabdomyolysis, which tend to escalate with higher dosages. Currently, there is no consensus on the optimal dosage of statins, highlighting the need for continued exploration.

This study provides new insights into the efficacy and safety of high-dose atorvastatin and rosuvastatin in treating CI. It particularly highlights the potential advantages of loading-dose rosuvastatin in terms of lipid control and safety. These findings offer new treatment options and reference guidelines for patients with CI.

Materials and methods

Sample information

A total of 151 CI patients treated at the Third Affiliated Hospital of Qiqihar Medical University from January 2022 to January 2023 were retrospectively analyzed. Based on the dosage, patients were grouped into the conventional atorvastatin group (n=35), loading-dose atorvastatin group (n=37), conventional rosuvastatin group (n=39), and loading-dose rosuvastatin group (n=40). The study was approved by the Medical Ethics Committee of the Third Affiliated Hospital of Qiqihar Medical University (2021-158).

Inclusion and exclusion criteria

Inclusion criteria: Patients who met the diagnostic criteria of ischemic stroke from the *Chinese Guidelines for Diagnosis and Treatment of Acute Ischemic Stroke (2018)* [9]; patients older larger than 18 years old; patients who had not taken statins before.

Exclusion criteria: Patients with liver, kidney, or organ diseases affecting gastrointestinal function; patients with myopathy, myositis, or other peripheral nerve diseases; patients with malignant tumors, mental illness, or disturbances of consciousness; patients allergic to statins; patients who had received anti-inflammatory drugs or immunosuppressive treatment before admission; patients with a history of prior stroke; patients who underwent interventional surgery.

Treatment regimen

Before treatment, all groups received basic treatment measures such as neuroprotection and blood pressure control. All patients in the study received standard treatment with essential medications, including a dose of 75 mg of clopidogrel (Actavis Group PTC ehf, H20140966) orally, in accordance with guidelines and individual patient conditions. Additionally, other medications were administered based on established guidelines and the specific needs of each patient.

Patients in the loading-dose atorvastatin group were treated with atorvastatin (Pfizer Pharmaceutical Co., Ltd., State Food and Drug Administration approval no.: H20051407) at 80 mg per dose. Patients in the conventional atorvastatin group were treated with atorvastatin at 20 mg per dose. Patients in the loading-dose rosuvastatin group were given rosuvastatin (Lunan BETTER Pharmaceutical Co., Ltd., SFDA approval number: H20080236) at 40 mg per dose. Patients in the conventional rosuvastatin group were given rosuvastatin at 5 mg per dose. All patients received oral administration of the respective drugs once daily before bedtime, with the treatment duration lasting for 6 months [10]. Atorvastatin and rosuvastatin were administered to each patient on the first day after admission.

Outcome measures

Primary outcome measures: 1. Changes in National Institutes of Health Stroke Scale (NIHSS) scores were analyzed between the two groups. 2. The clinical efficacy of the four

Table 1. Comparison of baseline data

Factors	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)	F/x^2	P
Age (years)	60.2±9.7	60.3±10.4	56.7±12.8	58.2±10.2	0.917	0.433
Gender (Male/Female)	20/15	22/15	20/17	27/13	1.598	0.659
Body mass index (kg/m^2)	24.44±2.51	24.24±4.51	23.96±5.51	24.88±1.51	1.376	0.871
Time of onset (h)	5.5±1.8	5.0±2.5	5.4±2.1	5.0±2.0	0.678	0.566
History of smoking (Yes/No)	25/10	27/10	27/12	25/15	1.157	0.763
History of hypertension (Yes/No)	17/18	15/22	16/23	15/25	0.997	0.801
History of diabetes mellitus (Yes/No)	5/30	7/30	8/31	7/33	0.523	0.913
History of hyperlipidemia (Yes/No)	6/29	6/31	5/34	8/32	0.7492, 3	0.8616
History of cardiovascular disease (Yes/No)	3/32	7/30	6/33	7/33	1.752, 3	0.6254

groups was evaluated. The criteria for efficacy were as follows: a decrease of ≥90% in NIHSS score was considered markedly effective, a decrease of 45%-89% was considered effective, and a decrease of less than 45% was classified as ineffective. 3. After 6 months, the levels of high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), alanine aminotransferase (ALT), total cholesterol (TC), and aspartate aminotransferase (AST) were tested in the two groups. High-sensitivity C-reactive protein (hs-CRP) and creatine kinase myocardial band (CK-MB) levels were compared between patients before treatment and 24 hours after treatment.

Secondary outcome measures: 1. The clinical baseline data of the four groups were compared. 2. Adverse reactions (nausea and vomiting, rash, coma, and allergic reactions) were compared between the two groups.

Statistical analyses

GraphPad 9 was used for data analysis and figure drawing. All categorical data were analyzed using the chi-square test and expressed as percentages. The chi-square partition method was adopted for chi-square comparisons among groups. All measurement data were described as mean ± standard deviation (SD). Inter-group comparisons were performed using the independent sample t-test; multi-group comparisons were performed using one-way Analysis of Variance (ANOVA) and presented as F; post hoc pairwise comparisons were performed using the Least Significant Difference test; comparisons at multiple time points were conducted using repeated measures ANOVA and expressed as F. Bonferroni correction was used for post hoc tests. P<0.05 was considered statistically significant.

Results

Comparison of clinical baseline data

Comparison of clinical baseline data between the four groups revealed no significant differences in terms of age, gender, body mass index, time of onset, history of smoking, history of hypertension, history of diabetes mellitus, history of hyperlipidemia, and history of cardiovascular disease (all P>0.05) (**Table 1**).

Comparison of NIHSS scores

The analysis of NIHSS scores for the four groups revealed no significant differences in NIHSS scores before therapy (Conventional atorvastatin group: 28.92±2.62 vs. Loading-dose atorvastatin group: 28.45±2.39 vs. Conventional rosuvastatin group: 29.11±2.11 vs. Loading-dose rosuvastatin group: 29.10±1.95, all P>0.05). After treatment, the NIHSS scores of the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 8.36±1.20, 6.34±1.02, 8.48±1.36, and 6.36±2.13, respectively (Supplementary Table 1). Significant decreases in NIHSS scores were found in all four groups after therapy (P<0.05). Additionally, no significant difference was found in NIHSS scores between the two high-dose groups after therapy (P>0.05), as shown in **Figure 1**.

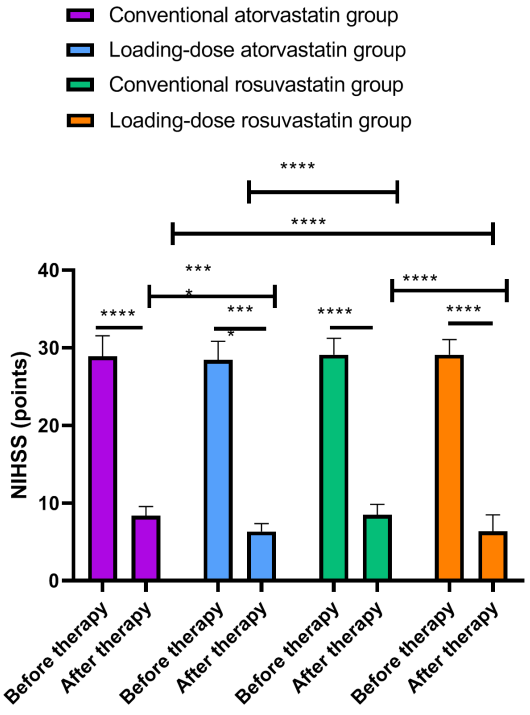

Figure 1. Changes in NIHSS scores of patients. Note: NIHSS: National Institutes of Health Stroke Scale. ***P<0.001; ****P<0.0001.

Comparison of clinical efficacy

The efficacy was significantly lower in the conventional groups compared to the corresponding loading-dose groups (P<0.05) (**Table 2**). However, no significant difference was found between the two loading-dose groups (χ^2= 2.084, P=0.149).

Comparison of changes in blood lipid indices

Before treatment, there were no significant differences in HDL-C, LDL-C, TG, and TC levels among the four groups (all P>0.05) (**Figure 2**). After treatment, the HDL-C levels in the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 1.39±0.21, 1.53±0.19, 1.45±0.23, and 1.68±0.16 mmol/L, respectively. The LDL-C levels were 2.26±0.33, 1.94±0.37, 1.78±0.34, and 1.52±0.27 mmol/L, respectively. The TG levels were 1.73±0.45, 1.74±0.52, 1.68±0.3, and 1.52±0.25 mmol/L, respectively. The TC levels were 4.33±0.41, 4.04±0.3, 3.81±0.3, and 3.55±0.25 mmol/L, respectively (Supplementary Table 2). After treatment, decreased LDL-C, TG, and TC levels were observed in all four groups (all P<0.05), and elevated HDL-C levels were observed in three groups (all P<0.05), except the conventional atorvastatin group (P>0.05) (**Figure 2**).

Comparison of changes in liver function indices

Before therapy, the ALT levels in the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 26.04±8.35, 28.71±11.12, 28.39±9.9, and 26.49±10.52 U/L, respectively. After therapy, the ALT levels were 28.55±12.09, 28.95±10.8, 28.86±10.39, and 27.39±9.59 U/L, respectively. Before therapy, the AST levels in the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 26.45±10.06, 25.58±11.88, 26.39±9.61, and 26.79±8.04 U/L, respectively. After therapy, the AST levels were 26.97±6.25, 26.21±6.67, 27.15±9.83, and 27.63±9.23 U/L, respectively (Supplementary Table 3). There were no significant differences in ALT and AST levels among the four groups before and after therapy (all P>0.05) (**Figure 3**).

Changes in myocardial index

Before therapy, the CK-MB levels in the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 41.43±17.44, 44.78±16.42, 40.96±18.55, and 45.27±19.63 U/L, respectively. After therapy, the CK-MB levels were 205.00±60, 177.57±52, 151.37±81.18, and 125.43±48.54 U/L, respectively (Supplementary Table 4). Before treatment, the CK-MB levels were not significantly different among the four groups (all P>0.05). After treatment, all four groups showed significantly increased CK-MB levels (all P<0.05). The increase was more significant in the loading-dose atorvastatin group compared to the loading-dose rosuvastatin group (both P<0.05) (**Figure 4**).

Changes in inflammatory indices

Before therapy, the hs-CRP levels in the conventional atorvastatin group, loading-dose atorvastatin group, conventional rosuvastatin group, and loading-dose rosuvastatin group were 3.1±2.13, 3.66±2.54, 3.67±2.12, and

Table 2. Comparison of efficacy evaluation

Efficacy evaluation	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)	x^2	P
Markedly effective	0	18	0	13		
Effective	35	19	39	27	41.018	<0.001
Ineffective	0	0	0	0		

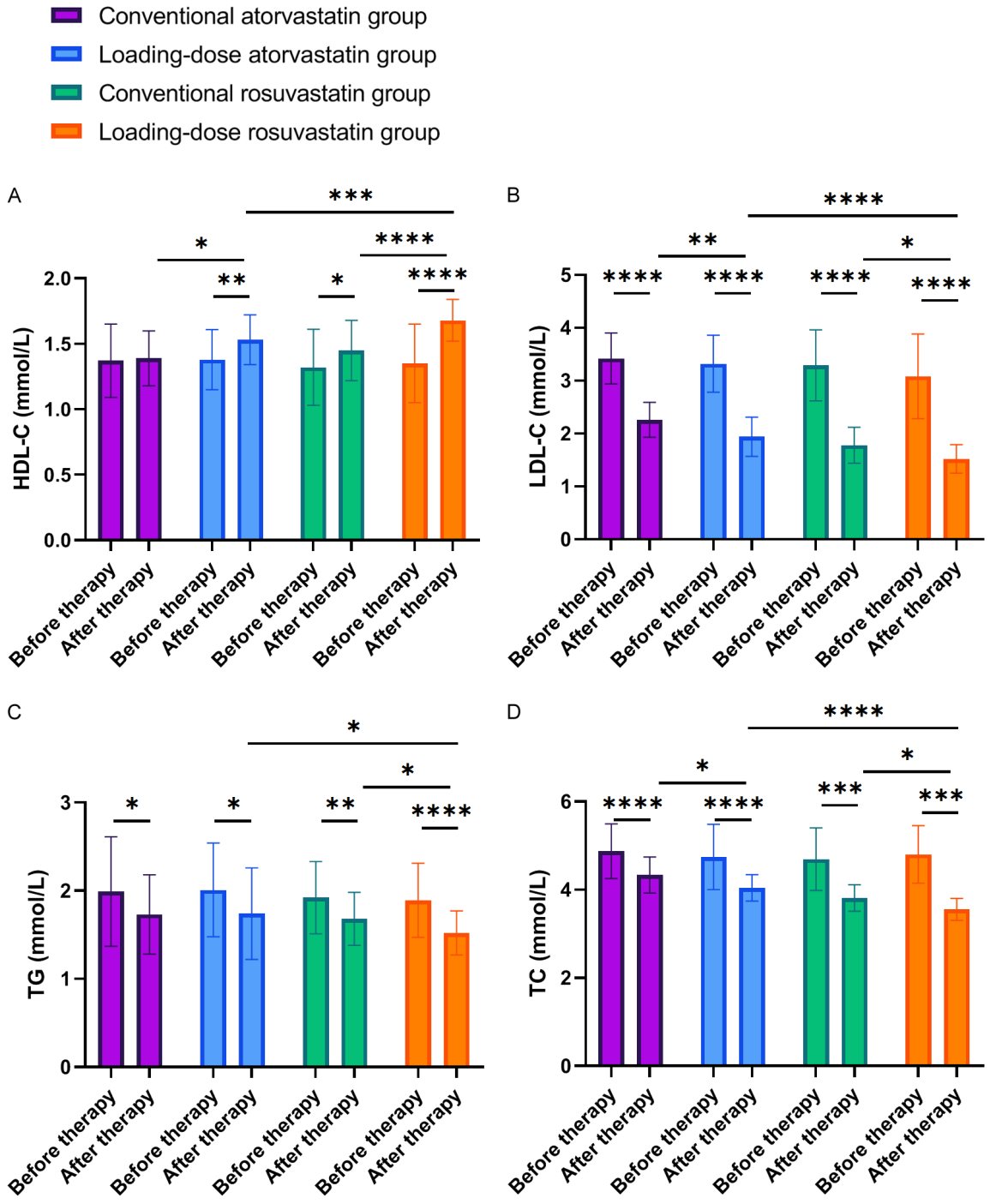

Figure 2. Changes of blood lipid indices in patients before and after therapy. A. Changes of HDL-C in the four groups before and after therapy; B. Changes of LDL-C in the four groups before and after therapy; C. Changes of TG in the four groups before and after therapy; D. Changes of TC in the four groups before and after therapy. Notes: HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; TG: Triglyceride; TC: Total cholesterol. *P<0.05; **P<0.01; ***P<0.001.

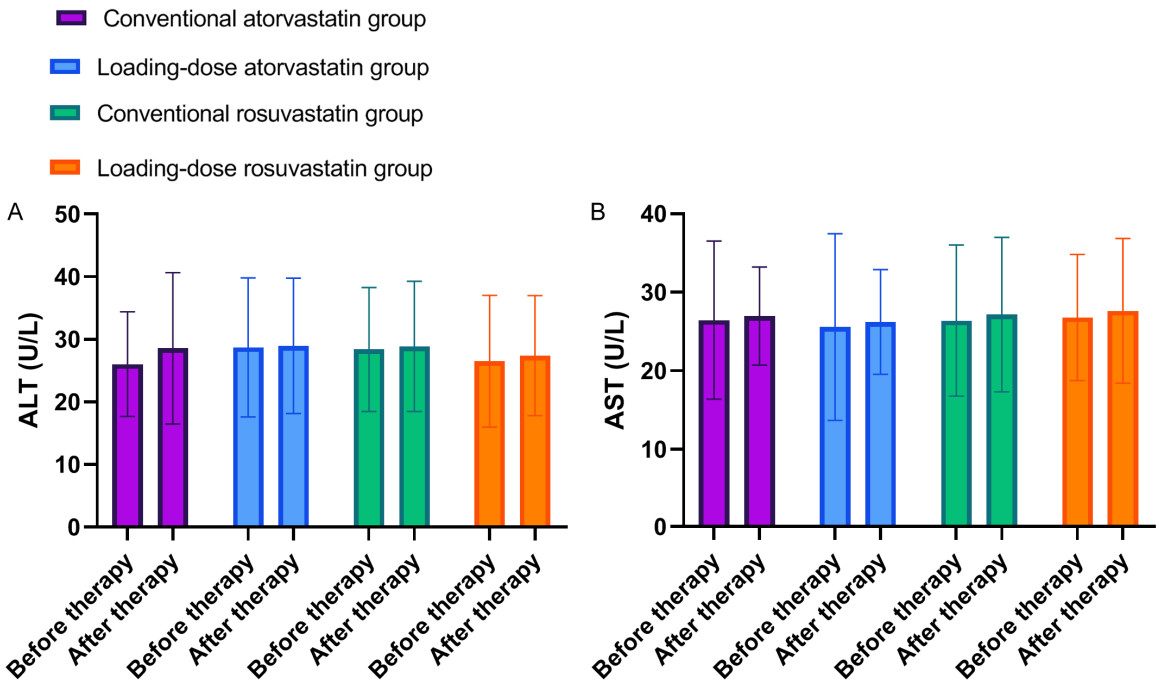

Figure 3. Changes of liver function indices in patients before and after therapy. A: Changes of ALT in the four groups before and after therapy; B: Changes of AST in the four groups before and after therapy. Notes: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase.

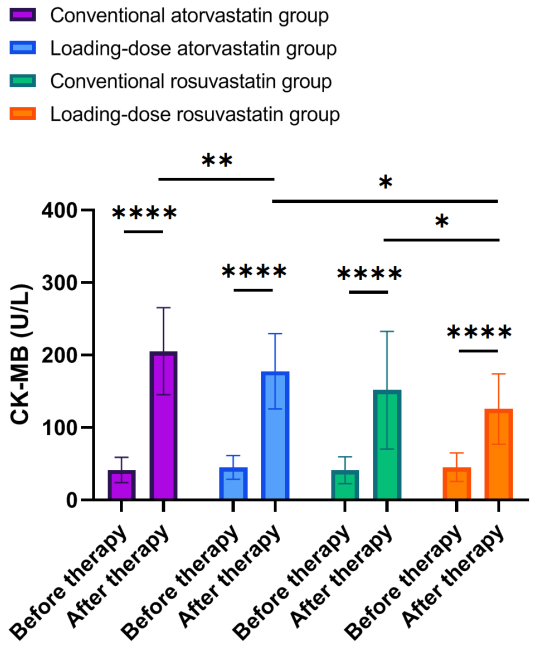

Figure 4. Changes of CK-MB in the four groups before and after therapy. Note: CK-MB: Creatine kinase myocardial band. *P<0.05; **P<0.01; ****P<0.0001.

3.72±2.09 mg/L, respectively. After therapy, these levels were 15.51±3.5, 13.5±4.2, 11.28±4.73, and 9.64±3.52 mg/L, respectively (Supplementary Table 5). Before treatment, the hs-CRP levels were not significantly different among the four groups (P>0.05). However, after 24 hours of treatment, all groups showed significantly increased hs-CRP levels (P<0.05), with the loading-dose atorvastatin group exhibiting a more significant increase than the loading-dose rosuvastatin group (P<0.05) (**Figure 5**).

Comparison of adverse reactions

The occurrence of adverse reactions, including nausea and vomiting, rash, coma, and allergic reactions, was recorded in all four groups. No significant differences were found among the groups (all P>0.05) (**Table 3**).

Discussion

Ischemic stroke is a severe cerebrovascular disease characterized by clinical ischemia, with

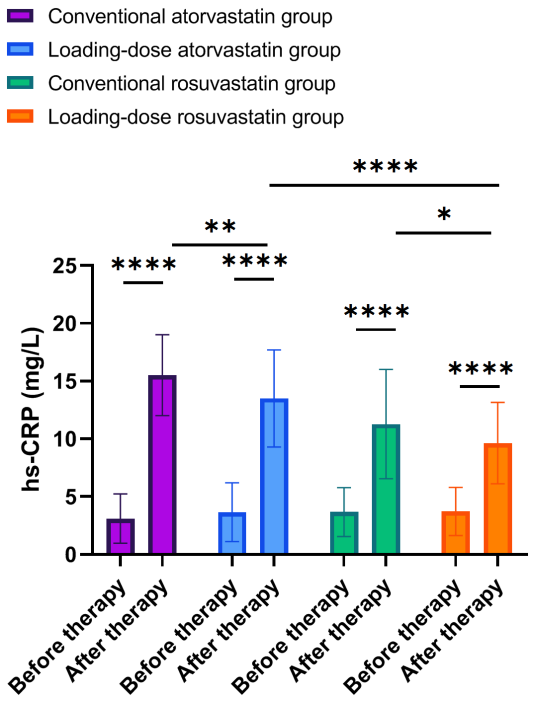

Figure 5. Changes of hs-CRP in the four groups before and after therapy. Note: hs-CRP: High-sensitivity C-reactive protein. *P<0.05; **P<0.01; ****P<0.0001.

a high recurrence rate and elevated disability and mortality rates [11, 12]. Statins, such as atorvastatin and rosuvastatin, are commonly used in treating cerebrovascular diseases [13]. They regulate blood lipid levels, inhibit inflammation and smooth muscle cell proliferation, improve vascular endothelial function, and reduce lipid deposition [14, 15]. Atorvastatin has pleiotropic effects, including anti-inflammatory, antioxidant, anti-platelet, and plaque stabilization properties [16]. Rosuvastatin also has unique efficacy and pleiotropic effects, contributing to the control and prevention of cerebrovascular diseases by lowering cholesterol and improving blood lipid levels [17]. Both statins provide strong support for reducing cerebrovascular risk and improving arteriosclerosis conditions [18].

The NIHSS score is a widely used tool for assessing the degree of neurological impairment in stroke patients, encompassing evaluations of consciousness, movement, sensation, language, and vision [19]. In this study, the use of a loading dose regimen demonstrated a more significant improvement in NIHSS scores compared to the conventional dosing regimen after treatment. The advantages of a loading dose regimen in treatment may be attributed to its faster drug action and higher drug concentration [20]. By using a larger initial dose, the therapeutic dose can be reached more quickly, expediting the medication's efficacy [21]. This can help alleviate neurological damage caused by CI and improve clinical outcomes for patients.

However, the absence of statistical differences between the two loading dose regimens may suggest similar efficacy in terms of NIHSS scores. This similarity in efficacy can be attributed to the fact that both medications have similar pharmacological mechanisms of action [22]. They both lower cholesterol synthesis by inhibiting HMG-CoA reductase [23]. This pharmacological action likely provides neuroprotection by reducing cholesterol levels and mitigating damage caused by cerebral ischemic events. Despite similar effects in the treatment and prevention of ischemic stroke, the two may differ in specific outcomes, such as reducing the size of the stroke focus and the severity of the stroke. These differences are possibly influenced by factors such as drug dose, administration time, and individual patient differences.

Previous research revealed that pretreatment with high-dose and low-to-medium-dose statins had no effect on the severity of ischemic stroke [24]. There was also no significant association between treatment with double doses of statins and NIHSS scores when assessing stroke severity. These discrepancies highlight the contrasting conclusions drawn from different research studies on this topic. The discrepancy may be due to patient heterogeneity: Ischemic stroke is a complex condition influenced by various factors, including patient demographics, comorbidities, and stroke etiology [25]. Differences in patient populations across studies, such as age, underlying medical conditions, or stroke subtypes, can introduce heterogeneity and affect observed outcomes.

Higher levels of HDL-C are associated with a reduced risk of cardiovascular disease, and the significant improvement in HDL-C levels in the loading-dose groups indicates a potential reduction in cardiovascular risk [26, 27]. The loading-dose groups demonstrated a significant reduction in LDL-C levels, indicating a more effective lipid-lowering effect for manag-

Table 3. Comparison of adverse reactions

Adverse reactions	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)	x^2	P
Nausea and vomiting	3	5	2	4	1.633	0.652
Rash	1	2	3	2	0.869	0.832
Coma	2	1	3	3	1.082	0.781
Allergic reaction	2	3	2	4	0.870	0.832

ing and preventing cardiovascular disease [27]. Additionally, there was substantial improvement in TG levels, suggesting that the loading-dose regimen may be more effective in reducing triglyceride levels and improving cardiovascular health [28]. Moreover, the loading-dose groups exhibited a significant reduction in TC levels, indicating a stronger lipid-lowering effect compared to the conventional-dose groups, which is beneficial for overall cardiovascular health [29].

Furthermore, the loading-dose rosuvastatin group showed significant reductions in hs-CRP and CK-MB levels after 24 hours, indicating decreased inflammation and myocardial injury compared to the loading-dose atorvastatin and conventional rosuvastatin groups. Rosuvastatin, a potent HMG-CoA reductase inhibitor, effectively inhibits cholesterol synthesis, improving lipid profiles [30, 31]. It also possesses anti-inflammatory and antioxidant effects, further reducing inflammatory responses and oxidative stress damage [32]. These properties contribute to the observed lower levels of hs-CRP and CK-MB in the loading-dose rosuvastatin group. Overall, the loading dose of rosuvastatin, with its stronger inhibitory effects on cholesterol synthesis, anti-inflammatory properties, and antioxidant effects, leads to better lipid improvement and reduced inflammatory and myocardial injury markers compared to atorvastatin [33-35].

Moreover, the results revealed no significant difference in the changes of ALT and AST levels between the four groups before and after therapy. This indicates that both loading dose and conventional dose regimens of atorvastatin and rosuvastatin had minimal impact on liver function. The comparable changes in liver enzyme levels suggest a low risk of hepatotoxicity. Additionally, there were no significant differences in the incidence of adverse reactions. These findings highlight the good safety profile and tolerability of both medications. Healthcare professionals can confidently prescribe either the loading dose or conventional dose regimens of atorvastatin or rosuvastatin, considering the low risk of hepatorenal toxicity and the overall good safety profile observed in this study.

This study has certain limitations that should be considered. Firstly, the sample size may not be sufficient to provide a comprehensive evaluation of the efficacy and safety of the two drugs, particularly when considering different doses and time points. Secondly, the analysis may not have accounted for all potential confounding factors that could influence the outcomes, such as patients' baseline health status, presence of other complications, and concurrent medications. Additionally, as a retrospective study, there is a possibility of selection bias and recall bias, which could affect the validity of the results. Lastly, the relatively short duration of the study may limit the assessment of long-term effects and the occurrence of adverse reactions associated with the drugs.

These results, however, provide valuable insights for clinicians in selecting a suitable and safe treatment approach to improve the prognosis of patients with ischemic stroke. Future research should focus on larger-scale and long-term studies to gain a better understanding of the efficacy and safety of these two drugs. This will help in determining the optimal treatment approach to improve the quality of life and survival rates among patients with ischemic stroke.

In summary, both loading-dose atorvastatin and rosuvastatin demonstrated increased clinical efficacy in the treatment of patients with CI, while ensuring safety and effectiveness. However, rosuvastatin exhibited superior effi-

cacy in blood lipid control. These findings provide valuable guidance for the clinical management of CI.

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Disclosure of conflict of interest

None.

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Supplementary Table 1. Comparison of changes in NIHSS scores of patients

Group	NIHSS scores before therapy	NIHSS scores after therapy
Conventional atorvastatin group (n=35)	28.92±2.62	8.36±1.20[a,b,c]
Loading-dose atorvastatin group (n=37)	28.45±2.39	6.34±1.02[a]
Conventional rosuvastatin group (n=39)	29.11±2.11	8.48±1.36[a,b,c]
Loading-dose rosuvastatin group (n=40)	29.10±1.95	6.36±2.13[a]

Note: NIHSS: National Institutes of Health Stroke Scale; a indicates P<0.05 vs. Before therapy; b indicates P<0.05 vs. the Loading-dose atorvastatin group; c indicates P<0.05 vs. the loading-dose rosuvastatin group.

Supplementary Table 2. Comparison of changes of blood lipid indices in patients before and after therapy

	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)
HDL-C (mmol/L)				
Before therapy	1.37±0.28	1.38±0.23	1.32±0.29	1.35±0.3
After therapy	1.39±0.21[b]	1.53±0.19[a,c]	1.45±0.23[a,c]	1.68±0.16[a,b]
LDL-C (mmol/L)				
Before therapy	3.42±0.48	3.32±0.54	3.29±0.67	3.08±0.8
After therapy	2.26±0.33[a,b]	1.94±0.37[a,c]	1.78±0.34[a,c]	1.52±0.27[a,b]
TG (mmol/L)				
Before therapy	1.99±0.62	2.01±0.53	1.92±0.41	1.89±0.42
After therapy	1.73±0.45[a]	1.74±0.52[a,c]	1.68±0.3[a]	1.52±0.25[a,b]
TC (mmol/L)				
Before therapy	4.87±0.62	4.74±0.74	4.69±0.71	4.8±0.65
After therapy	4.33±0.41[a,b]	4.04±0.3[a,c]	3.81±0.3[a,c]	3.55±0.25[a,b]

Notes: HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; TG: Triglyceride; TC: Total cholesterol; a indicates P<0.05 vs. Before therapy; b indicates P<0.05 vs. the Loading-dose atorvastatin group; c indicates P<0.05 vs. the loading-dose rosuvastatin group.

Supplementary Table 3. Comparison of changes of liver function indices in patients before and after therapy

	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)
ALT (U/L)				
Before therapy	26.04±8.35	28.71±11.12	28.39±9.9	26.49±10.52
After therapy	28.55±12.09	28.95±10.8	28.86±10.39	27.39±9.59
AST (U/L)				
Before therapy	26.45±10.06	25.58±11.88	26.39±9.61	26.79±8.04
After therapy	26.97±6.25	26.21±6.67	27.15±9.83	27.63±9.23

Notes: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase.

Supplementary Table 4. Comparison of changes of CK-MB in the four groups before and after therapy

	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)
CK-MB (U/L)				
Before therapy	41.43±17.44	44.78±16.42	40.96±18.55	45.27±19.63
After therapy	205.00±60.00[a,b]	177.57±52[a,c]	151.37±81.18[a,c]	125.43±48.54[a,b]

Note: CK-MB: Creatine kinase myocardial band; a indicates P<0.05 vs. Before therapy; b indicates P<0.05 vs. the Loading-dose atorvastatin group; c indicates P<0.05 vs. the loading-dose rosuvastatin group.

Supplementary Table 5. Comparison of changes of hs-CRP in the four groups before and after therapy

	Conventional atorvastatin group (n=35)	Loading-dose atorvastatin group (n=37)	Conventional rosuvastatin group (n=39)	Loading-dose rosuvastatin group (n=40)
hs-CRP (mg/L)				
Before therapy	3.1±2.13	3.66±2.54	3.67±2.12	3.72±2.09
After therapy	15.51±3.5[a,b]	13.5±4.2[a,c]	11.28±4.73[a,c]	9.64±3.52[a,b]

Note: hs-CRP: High-sensitivity C-reactive protein; a indicates P<0.05 vs. Before therapy; b indicates P<0.05 vs. the Loading-dose atorvastatin group; c indicates P<0.05 vs. the loading-dose rosuvastatin group.