Proceedings of ICMMGH 2026 Symposium: Cell and Molecular Biology: Understanding Immune Cell Functions
DOI: 10.54254/2753-8818/2026.PJ30430

The Clinical Efficacy of Aspirin Combined with Statins

Yanduan Shan

Nantong University, Nantong, China
2323110196@stmail.ntu.edu.cn

Abstract. Cerebral thrombosis is one of the most common types of ischemic stroke in cerebrovascular diseases. Atherosclerotic plaque rupture and thrombus formation cause cerebral blood flow cessation and cerebral tissue necrosis. It is characterized by high incidence, disability rate and mortality rate. Currently, the main methods to treat cerebral thrombosis include antiplatelet, thrombolysis, anticoagulation, surgery, etc. The use of a single agent regimen, such as aspirin, is limited by suboptimal therapeutic outcomes and a tendency towards recurrence, although it has antiplatelet effect. The benefits of statins go beyond lipid lowering to involve multiple mechanisms, including systemic anti-inflammation, stabilization of atherosclerotic plaques and improvement in endothelial function. Thus, the combination therapy strategy becomes an important means to optimize treatment. This study aims to compare the therapeutic outcomes and safety of aspirin-statin combination regimen versus monotherapy in cerebral thrombosis patients. The results indicate that due to its combined platelet inhibition, lipid modulation, plaque stabilization and inflammation reduction effects, concomitant administration of aspirin and statins yields a synergistic therapeutic effect. It shows better efficacy in not only inhibiting thrombotic progression but also inducing arterial plaque regression. This optimized strategy has great promise for the clinical management of cerebral thrombosis, and its high safety profile lays the foundation for extensive clinical translation.

Keywords: Aspirin, cerebral thrombosis, statins

1. Introduction

Cerebral thrombosis is a common disease in neurology. It is caused by the disorder of cerebral circulation, leading to vascular occlusion and causing ischemia. Hypoxia further induces a series of neuronal injury and death, and finally results in focal or global neurological dysfunction. Patients under routine conditions are usually seriously ill with a high possibility of disability or fatality. With the progress of society and the aging of population, the incidence of cerebral thrombosis keeps increasing [1], and the age of onset of the disease tends to be younger [2]. Data statistics show that about 1000 people out of every million are patients, and males are usually more than females, the elderly are very common [3,4]. In addition, the mortality of cerebral thrombosis is about 10%, about 45% survivors will be disabled [5] and the recurrence rate of one year is 4.69% [6]. Cerebral thrombosis is often accompanied by various complications, which brings a heavy burden to the family and the society.

DOI: 10.54254/2753-8818/2026.PJ30430

Although the treatment options for cerebral thrombosis include thrombolysis, antithrombotics, surgery, and rehabilitation, thrombolytic therapy itself has some critical shortcomings: the short window for use and the ineffectiveness against dyslipidemia [7]. A large number of patients have missed the best treatment opportunity because of late medical visits. Antiplatelet treatment with aspirin alone has a high recurrence rate. The therapeutic effect is unsatisfactory [8]. In addition, it can lead to gastrointestinal reactions such as abdominal pain, nausea, vomiting, diarrhea, etc., and some patients even develop allergic reactions. Anticoagulation therapy focuses on strict monitoring for internal bleeding, which requires constant vigilance for clinical symptoms, and the drug should be stopped immediately once any sign of bleeding appears [9].

Aspirin plus statins has relatively obvious effect on the treatment of cerebral thrombosis. Aspirin reduces the formation of TXA2 by inhibiting COX-1, thus preventing thrombosis. The level of low density lipoprotein cholesterol is reduced by statins. In addition, Statins prevent the synthesis of vascular plaque by inhibiting the activity of the rate limiting enzyme in cholesterol synthesis in hepatocytes. The combination of the two can improve prognosis effectively. This article is aimed at exploring the clinical efficacy and mechanism of this combined treatment plan deeply. It will provide evidence to optimize the clinical strategies for cerebral thrombosis.

2. The pathogenesis and current treatment status of cerebral thrombosis

Cerebral thrombosis is caused by some unstable dangerous factors. For instance, hypertension, diabetes, dyslipidemia, hemodynamic factors, and lifestyle are five critical reasons. Three stages are intersected in the illness timeline: the increase of blood thickness, the changes of transport pattern of blood and lesion of the internal cortex of the body. Chronic insults such as hypertension and smoking cause chronic damage to the vascular endothelium. This damage allows for lipid deposition and the formation of an atherosclerotic plaque. Platelets will be motivated by the breakdown of plaque. It will lead to the adsorption of plaque in the damaged areas. Besides, coagulation factors will be sensitive and animated. Fibrinogen undergoes a molecular transition to form fibrin polymers. Red blood cells are captured by the net of fibrin. With the growth of thrombus, the cerebral vasculature is clogged. It will induce oxygen deficiency, cerebral ischemia, neuronal death. Meanwhile, after the emergence of disease, it has a poor control of the patient's disease progress [10].

Many pharmaceuticals comprise thrombolytic, anticoagulant , antihypertensive drugs, and other drugs for the underlying disease are being used in clinical practise. Among them, aspirin has special antiplatelet effects. The main mechanism of aspirin is nonreversible acetylation of cyclooxygenase-1 (COX-1). The synthesis of thromboxane A2 (TXA2) suffer sustained suppression. It makes a further step to inhibit the activation and aggregation of platelet. At the same time, it improves perfusion dynamics at the capillary level. The inhibitory effect cover platelets' life from inception to obsolescence which is long term effectiveness. Therefore, the antithrombotic effect lasts until the next generation when the new platelets is formed. Aspirin has a lasting and moderate antiplatelet effect [11]. It is used for anticoagulation treatment of ischemic stroke. In spite of this, using aspirin alone as an antiplatelet has various response in clinical manifestation according to the clinical data. Alongside this, it is often ineffective. It is also worth nothing that another approach is to switch anticoagulants such as warfarin. It works through a mechanism which is different from aspirin. However, warfarin has a serious disadvantage. It needs close and regular monitoring to the coagulation indicators. In order to ensure the pharmaceuticals are safe and effective. These limitations restrict its wider use in the word.

DOI: 10.54254/2753-8818/2026.PJ30430

The most frequently and widely application and deeply researched of lipid-lowering pharmaceuticals are statins. HMG-CoA reductase inhibitors are the name which people known. An enzyme is inhibited by statins. It is critical for cholesterol production in the liver. The number of the production of hepatic cholesterol was lessened by the inhibition of this enzyme. This further precipitating the hepar autonomously absorb more cholesterol from the blood. Eventually, it leads to the decrease of LDL-C. A large number of statins are commonly used such as atorvastatin, simvastatin, and rosuvastatin.

3. The synergistic mechanism of aspirin and statins

To improve prognosis in cerebral thrombosis, it is suggested that aspirin be used in combination with statins. This strategy supplements the antiplatelet effect of aspirin with the pleiotropic effects of statins (lipid regulation, anti-inflammation, antithrombotic effects, etc.), resulting in significantly improved therapeutic efficacy, which is reflected in several specific aspects. Firstly, statins have a basic lipid-lowering effect, which can significantly reduce the total cholesterol and LDL levels. In addition to this basic effect, researchers attribute important pleiotropic benefits, such as anti-inflammatory and antioxidant effects. These mechanisms act synergistically to stabilize vulnerable atherosclerotic plaques by thickening the fibrous cap and reducing the lipid core volume [12]. This stabilization of previously rupture-prone plaques eliminates one of the main triggers for thrombosis, while also reducing vascular endothelial inflammation and the overall risk of plaque formation. Secondly, the antiplatelet effect of aspirin, which fundamentally hinders thrombus formation by preventing platelet clumping, is significantly enhanced by combination therapy. This enhanced disruption of the aggregation process leads to stronger inhibition and better overall efficacy. Thirdly, statins such as simvastatin can improve endothelial function.They promote endothelial cell migration. Statins promote blood circulation and reduce the risk of formation of cerebral thrombosis [9]. From the stable period of cerebral thrombosis, aspirin makes a significant contribution through its anti-inflammatory effects, which help to alleviate vascular inflammation and reduce plasma hyperviscosity. These actions collectively reduce the risk of further plaque formation and slow disease progression. Statins, on the other hand, operate via complementary mechanisms, inhibiting endothelial inflammatory responses, stabilizing existing atherosclerotic plaques, and providing direct neuroprotective benefits [13].

4. Clinical study on the combination therapy of aspirin and statins

In order to verify the effect of aspirin work together with statins for cerebral thrombosis, two majority types of statins, pravastatin and simvastatin, were selected for analysis in combination with aspirin.

4.1. The data analysis of aspirin work in company with pravastatin sodium to fight against cerebral thrombosis

A total of 80 patients with cerebral thrombosis admitted to Jiuquan Peoples Hospital of Gansu Province for this study. The patients were stochastically divided into a control group and a study group. There were 40 patients in each group, which included 22 males and 18 female. They were between 45-75 years old, with an average age of (52.15±2.57) years old, and the time from onset to treatment ranged from 1 to 7 h, with an average of (3.47±0.53) h; there were 23 males and 17 females in the control group who were between 46-75 years old, with an average age of

DOI: 10.54254/2753-8818/2026.PJ30430

(52.17±2.59) years old, and the time from onset ranged from 2 to 7 h, with an average of (3.45±0.51) h. There was no statistically significant difference in gender, age, and treatment time between these two groups of patients. (P>0.05). The people who divided in the control group were given aspirin orally at a dose of 75-100 mg bid; Pravastatin sodium tablets (10 mg qd) were given to the people who in the study group, which was on the base of the control group. Both groups were treated for 15 days as one course of treatment, and the therapeutic effect was observed after two courses of treatment. The evaluation indexes included clinical efficacy (cured, markedly effective, effective, ineffective according to the degree of reduction in neurological deficit score, and the total effective rate was the sum of the cured, markedly effective and effective rates), carotid plaque area, carotid intima-media thickness (IMT) and the incidence of adverse reactions (anaphylactic shock, dyspnea, conjunctival hemorrhage). All data were processed by SPSS 22.0. The count data was expressed as a percentage (%), and the chi-square test was used for comparison between groups. Measurement data was expressed as meanstandard deviation, and the t test was used to compare the difference between two groups.A P-value less than 0.5 was regarded as statistical standards [14].

The results suggested that the study group had a significantly better overall response rate than the control group (P<0.05), see Table 1. For carotid artery parameters, both groups had similar plaque area and intima-media thickness at baseline (P>0.05). After treatment, the study group had a significantly better plaque area regression and a larger intima-media thickness increase than the control group (P<0.05), see Table 2. As for safety, the frequency of study group of adverse drug reaction was 7.50% and the date of control group was 5.00% , which had nothing statistical differences between the two group (P>0.05), see Table 3.

Table 1. Comparison of clinical efficacy between two groups [14]

Group	Recover	Tangible Results	Effective	Invalid	Total Effective
Study group	21(52.50)	11(27.50)	5(12.50)	3(7.50)	37(92.50)
Control group	7(17.50)	10(25.00)	12(30.00)	11(27.50)	29(72.50)
χ^2					5.541
P					0.019

Table 2. Comparison of carotid plaque area and carotid intima-media thickness between two groups [14]

Group	Carotid artery plaque area(cm^2)		Carotid intima-media thickness	
	Before treatment	After treatment	Before treatment	After treatment
Study group	1.82±0.29	1.43±0.21	0.83±0.14	0.68±0.12
Control group	1.81±0.26	1.58±0.23	0.82±0.13	0.51±0.11
t	0.162	3.046	0.331	6.605
P	0.817	0.003	0.742	0.000

DOI: 10.54254/2753-8818/2026.PJ30430

Table 3. Comparison of adverse reactions between two groups of drugs [14]

Group	Anaphylactic shock	Breathing difficulties	Conjunctival hemorrhage	Adverse reactions
Study group	1(2.50)	1(2.50)	1(2.50)	3(7.50)
Control group	1(2.50)	0(0.00)	1(2.50)	2(5.00)
χ^2				0.000
P				1.000

4.2. The data analysis of aspirin work in company with simvastatin to fight against cerebral thrombosis

A total of 82 patients with cerebral thrombosis admitted to our hospital from March 2022 to February 2023 were selected as research objects. According to the random number table method, they were divided into a control group and an observation group, with 41 cases in each group. The control group had 27 men and 14 women, aged 58-76 years old, with an average age of 64.25±4.12years old, and the time from onset to the end of the disease was between 7 months and 4 years, with an average illness timeline of 2.33±0.71years. The observation group had 25 men and 16 women, aged 54-77 years old, with an average age of 63.05±5.03years old, and the illness timeline was between 6 months and 4 years, with an average illness timeline of 2.24±0.69 years. There was no statistical difference in gender, age, and course of disease between the two groups (P>0.05), which can be compared. The control group was treated with enteric-coated aspirin, 70 mg/d. The observation group was treated with a combination of simvastatin (20 mg/d) and aspirin (same dose as the control group). Both groups were treated for 6 months continuously. The neurological impairment was evaluated by the National Institutes of Health Stroke Scale (NIHSS) score, with a score of 0 to 24 points, the lower the score, the better the function. The Barthel index was used to evaluate the ability of daily life, with a score of 0100 points, the higher the score, the better the independence. The therapeutic effect was divided into markedly effective, effective and ineffective. Markedly effective refers to a reduction in the NIHSS score of 40%-90%, effective refers to a reduction in the NIHSS score of 18%-39%, and ineffective means that the above standards were not met. The carotid intima-media thickness and plaque area were evaluated by ultrasonography before and after treatment [15].

Comparative analysis of the post-treatment data showed better results in the observation group. Post-treatment NIHSS and Barthel Index scores were significantly improved in the observation group compared with the controls (P<0.05, Table 4). The overall treatment response rate was significantly higher in the observation group (P<0.05, Table 5). Carotid ultrasound measurements also showed that the observation group had significantly lower intima-media thickness and plaque area (P<0.05, Table 6).

Table 4. Comparison of NIHSS and barthel index scores

Group	Number of cases	NIHSS		Barthel	
		Before treatment	After treatment	Before treatment	After treatment
Study group	41	25.12±3.63	12.25±3.02	36.87±16.05	81.05±3.36
Control group	41	25.31±2.93	18.14±3.45	37.05±15.57	73.63±4.12
t		0.441	5.052	0.636	9.121
P		>0.05	<0.05	>0.05	<0.05

DOI: 10.54254/2753-8818/2026.PJ30430

Table 5. Comparison of total effective rates of treatment

Group	Number of cases	Tangible Results	Effective	Invalid	Total Effective
Study group	41	27	12	2	95.12%
Control group	41	24	10	7	82.92%
χ^2					13.041
P					<0.05

Table 6. Comparison of intima-media thickness and plaque area of carotid artery

Group	Number of cases	Carotid intima-media thickness(mm)		plaque area(cm^2)	
		Before treatment	After treatment	Before treatment	After treatment
Study group	41	0.77±0.23	0.49±0.17	1.72±0.35	1.31±0.32
Control group	41	0.76±0.63	0.62±0.22	1.74±0.38	1.59±0.35
t		0.241	2.052	0.633	4.021
P		>0.05	<0.05	>0.05	<0.05

5. Discussion and further outlook

Based on multidimensional data analysis and assessment, aspirin plus statin therapy is more effective and has better therapeutic effects on cerebral thrombosis than aspirin monotherapy, with better performance in improving neurological deficits. Post-treatment observation revealed a marked decrease in carotid intima-media thickness (IMT) and plaque area. Interestingly, among the statins, pravastatin sodium combination therapy had higher post-treatment carotid IMT values than aspirin alone, while simvastatin combination therapy had lower values. The incidence of adverse reactions related to combination therapy was low, with a minor effect on patients. In terms of the future development trend of aspirin-statin combination therapy, it is moving from a general use strategy to a precise targeting strategy. Although the combination still plays a fundamental role in the treatment of atherosclerotic cardiovascular disease, efforts will be made in the future to identify specific populations that can benefit significantly with acceptable risks through the application of biomarkers and multidimensional risk scores. At the same time, research will explore the possibility of using new and safer antiplatelet drugs to replace aspirin in some cases, while actively promoting the development of fixed-dose combination formulations containing both drugs. This aims to strategically improve long-term treatment compliance and the efficiency of chronic disease management, thus realizing comprehensive reductions in the disability rate, mortality rate, and recurrence rate of cerebral thrombosis.

6. Conclusion

Cerebral thrombosis refers to pathological occlusion of cerebral vessels by thrombi, which causes interruption of blood supply and necrosis of brain tissue caused by ischemia and hypoxia. The lost function is related to the duties of the necrotic neurons. The severity and specific manifestations of the injury vary with the location and size of the vessel occlusion, often resulting in severe consequences such as hemiplegia, aphasia, intellectual and emotional disorders, and even death. Aspirin combined with statins shows significantly better efficacy for the treatment of cerebral

DOI: 10.54254/2753-8818/2026.PJ30430

thrombosis than monotherapy. It can promote the regression of atherosclerotic plaque area and the decrease of carotid intima-media thickness, and also contribute to obvious neurological recovery and improved performance in activities of daily living. In addition, it is associated with a small number of adverse events and good safety, which supports its wider application in clinical practice.

References

[1] Mu Pengying, Niu Xianghong. (2016). Effect of atorvastatin combined with aspirin on cerebral blood flow and carotid atherosclerotic plaque in patients with acute cerebral infarction. Anhui Medical Journal, 2016, 37(9): 1124-1126, 1127.
[2] Linxin Li, Catherine A. Scott, Peter M. Rothwell. (2022). Changes in Incidence of Stroke and Other Vascular Events at Younger vs Older Ages, 2002-2018. JAMA: the Journal of the American Medical Association, 328(6): 563-574.
[3] Zhang Junna. (2021). Analysis of Clinical Common Cerebrovascular Diseases and Risk Factors. Friend of Health, (10): 45.
[4] Portegies, Marileen L. P., Wolters, Frank J., Hofman, Albert, et al. Prestroke Vascular Pathology and the Risk of Recurrent Stroke and Poststroke Dementia [J]. Stroke: A Journal of Cerebral Circulation, 2016, 47(8): 2119-2122. DOI: 10.1161/STROKEAHA.116.014094.
[5] Peng Bin, Wang Yongjun, Yao Ming. (2015). Chinese Society of Neurology, Chinese Stroke Society. Chinese guidelines for diagnosis and treatment of acute ischemic stroke 2014. Chinese Journal of Neurology, 48(4): 246-257.
[6] Su Runping, Zhang Hongjin. (2015). Analysis of recurrence factors and clinical prognosis of cerebral thrombosis. Psychological Doctor, 21(14): 7-8.
[7] Lu Yang. (2022). Effect of atorvastatin combined with aspirin on neurological impairment and arterial plaque in 92 patients with cerebral thrombosis. Contemporary Medicine, b28(1): 95-97.
[8] Ma Yingli, Hu Xuzhen. (2020). Improvement of arterial plaque and effect on neurological function of aspirin combined with statins in patients with cerebral thrombosis. China Modern Doctor, (36): 48-51.
[9] Liu Xiaoping, Zhang Yaqi. (2023). Efficacy of simvastatin combined with aspirin in the treatment of cerebral thrombosis and its effect on neurological deficit. International Journal of Clinical Research, 7(8).
[10] Wang Xiaowei. (2019). Analysis of the etiology and pathogenesis of cerebral thrombosis. Guide on How to Keep Good Health, (34): 378.
[11] Zheng Ying, Lei Xiaofeng, Cheng Yan, et al. (2018). Predictive value of aspirin resistance combined with platelet-leukocyte aggregate detection for early neurological deterioration in patients with acute cerebral infarction. Chinese Journal of Critical Care Medicine, 38(3): 220-224.
[12] Huang Fei, Li Xia. (2020). Improvement effect of aspirin combined with statins on carotid artery plaque in patients with cerebral thrombosis. Doctor, 5(5): 91-92.
[13] Chen Hua, Wang Xia. (2023). Observation on the effect of aspirin combined with statins in the treatment of cerebral thrombosis. Chinese Community Doctor, 39(18): 10-12.
[14] Cai Ruijun. (2024). Clinical efficacy and adverse drug reactions analysis of statins combined with aspirin in the treatment of cerebral thrombosis. Reproduction and Health, 30(18): 82-84.
[15] Yang Jing. (2023). Effect of simvastatin combined with aspirin on cerebral thrombosis and its influence on neurological deficit. International Journal of Brain Research, 3(1).