INFLUENCES OF DUAL ANTIPLATELET THERAPY ON NEUROLOGICAL FUNCTION, HEMORHEOLOGY AND OUTCOMES IN PATIENTS WITH POSTERIOR CIRCULATION ISCHEMIC STROKE

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ABSTRACT

Objective*: To investigate the influences of dual antiplatelet therapy on neurological function, hemorheology and outcomes in patients with posterior circulation ischemic stroke (PCIS).*

Methods*: A total of 150 patients with PCIS in our hospital from May 2014 to May 2016 were selected and were divided randomly into a control group and an observation group, with 75 cases in each group. The patients in the two groups were given conventional therapy. The control group was given an aspirin anticoagulant therapy, and the observation group was given a dual antiplatelet therapy (aspirin and clopidogrel). National Institutes of Health Stroke Scale (NIHSS) scores, Alzheimer Disease Assessment Scale (ADAS-Cog) scores and changes in the hemorheology index in the two groups were compared. The differences in outcomes and adverse were observed.*

Results*: After treatment, the NIHSS scores (6.33±2.28) and the ADAS-Cog scores (34.71±5.49) in the observation group were significantly lower than the NIHSS scores (10.04±2.51) and the ADAS-Cog scores (42.83±5.52) in the control group, and the differences were statistically significant ($P < 0.05$). After treatment, the levels of platelet (PLT) and fibrinogen (FIB), whole blood high-shear relative viscosity, whole blood low-shear relative viscosity, and plasma viscosity in the observation group were significantly lower than those in the control group ($P < 0.05$). After a 1-year follow-up, the recurrence rate in the observation group was significantly lower than that in the control group ($P < 0.05$). The disability and mortality in the observation group were slightly lower than those in the control group, but the difference was not statistically significant ($P > 0.05$); the total incidence of adverse reactions in the observation group was slightly higher than that in the control group (14.67% vs 12.00%), but there was no statistically significant difference ($P > 0.05$).*

Conclusion*: Dual antiplatelet therapy can improve the neurological function of the patients with PCIS, has favorable outcomes and safety profiles, and is worthy of clinical application.*

Keywords*: Dual antiplatelet, Posterior circulation ischemic stroke, Neurological function, Outcomes.*

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Introduction

Cerebral ischemic stroke is a common and major adverse cardiovascular and cerebrovascular event. Therefore, ischemia, hypoxia and even necrosis of brain tissue caused by sudden decrease or cessation of blood supply in regional brain tissue results in clinical features of the corresponding site[(1)].

Currently, according to different clinical features and invasive locations, cerebral ischemic stroke is clinically divided into anterior circulation ischemic stroke (ACCI) and posterior circulation ischemic stroke (PCIS). Disability and mortality rates are relatively high due to unfavorable outcomes of PCIS. PCIS refers to vertebral-basilar transient ischemic stroke, which is a clinical syndrome of posterior circulation arterial ischemia

caused by primary thrombus, foraminal stenosis and thrombotic obliteration and is associated with factors such as abnormal adhesion aggregation of platelets, blood hypercoagulability, hemodynamics and hemorheology[2-3]).

Clinical data showed that approximately 15%~25% of stroke patients are diagnosed with PCIS[4-5]. Patients with PCIS have poor outcomes and high mortality due to complex anatomic structure and blood supply of PCIS; therefore, it is of great clinical significance to find measures to improve the neurological function and outcomes of patients with PCIS. Clinically, many patients with PCIS are mostly treated with neurosurgical intervention, thrombolysis treatment or acute endovascular treatment[6-7], but the clinical outcomes are not satisfactory. In recent years, studies showed that strengthening antiplatelet therapy can effectively improve outcomes in patients with PCIS, but currently, there is still some controversy, and dual antiplatelet therapy needs further clinical verification[8]. In this study, 150 cases of patients with PCIS in our hospital were recruited to investigate the neurological function, hemorheology and safety of a dual antiplatelet scheme for PCIS.

Materials and methods

General data

A total of 150 patients with PCIS in our hospital from May 2014 to May 2016 were selected and were divided randomly into a control group and an observation group, with 75 cases in each group. The study met the standard procedure for the Institutional Ethics Committee, and all the patients were informed and signed the informed consents.

Inclusion criteria[9]: clinical manifestations included visual and visual field disturbances, consciousness disorders and unilateral sensory disturbance, etc.; 140 mm Hg ≤ systolic pressure ≤ 220 mm Hg, diastolic pressure ≥ 80 mm Hg; and CT or MRI examination confirmed cerebral ischemic strokes in the sites of brainstem, cerebellum and occipital lobe and so on. There were 75 cases in the control group, including 42 males and 33 females, age 50 to 78 years, and the mean age was 59.33 ± 7.18 years; there were 31 cases of dizziness and headache, 10 cases of dysarthria, 5 cases of hemiplegia, and 29 cases of other symptoms. There was no significant difference in age, sex and disease type between the two groups (P>0.05). There were 75 cases in the observation group, including 46 males and 29 females, age 15 to 76 years, and the mean age was 61.27±7.02 years; there were 29 cases of dizziness and headache, 13 cases of dysarthria, 7 cases of hemiplegia, and 26 cases of other symptoms. There were no significant differences in age, gender and type of illness between the two groups (P > 0.05). Exclusion criteria: patients with blood-borne diseases; patients with critical organ lesions, such as in the liver, kidney, and heart; patients who cannot cooperate with the treatment and follow-up observation; patients with incomplete information; and patients who refuse the follow-up or are lost to follow-up.

Methods

The patients in the two groups received routine treatments, including improvement of blood pressure, reduction of intracranial pressure, application of cerebral protective agents and calcium antagonists for prevention of complications. The systolic pressure was kept at 90 to 140 mm Hg, and the diastolic pressure was kept at 60 to 90 mm Hg. The control group was treated only with aspirin (Germany Bayer Health Care Co., Ltd., J20130078), 100 mg /d, PO. The patients in the observation group were treated with aspirin combined with clopidogrel (Chinese Hangzhou Sanofi Pharmaceutical Co., Ltd., J20130083), 75 mg/d, PO. After 2 weeks of continuous treatment, the National Institutes of Health Stroke Scale (NIHSS) and Alzheimer Disease Assessment Scale (ADAS-Cog) scores and whole blood viscosity indexes in the two groups were observed and compared. In addition, the levels of platelet (PLT) and fibrinogen (FIB) and plasma viscosity indexes were detected, the adverse reactions of the patients were counted, and the patients were followed up after 1 year for outcomes observation.

Observational Target

Before and after the treatment, the ADAS-Cog scale (10) was used to score the patients. The scoring criteria included 12 items, and the scores ranged from 0 to 75. The higher the score was, the more serious the cognitive impairment. Meanwhile, the NIHSS scale (11) was used to score disease severity. Scores from 0 to 6 were considered mild grade, scores from 7 to 15 were considered as moderate grade and scores that were 16 or above were considered severe grade. Before treatment and 2 weeks after treatment, the levels of PLT and

FIB in the patients were detected by turbidimetry, and the blood viscosity indexes including whole blood high-shear relative viscosity, whole blood low-shear relative viscosity and plasma viscosity were detected by a LB-2A automatic blood viscosity tester (China Jinan Hanfang Medical Devices Co., Ltd.). Adverse reactions of patients during drug therapy, such as skin rash, nausea, vomiting and excessive low platelet, were observed. Patients were followed up by telephone regularly for 1 year after discharge, at least once every three months, and the disability, recurrence rate and mortality of patients were recorded.

Statistical analysis

The data were analyzed with the SPSS 20.0 software. The numerical data were expressed as a percentage (%) and analyzed by the χ^2 test. The measurement data were expressed as the mean ± standard deviation (mean±SD) and analyzed by the t test; a P value of <0.05 was considered a significant difference.

Results

Comparison of the general data in the two groups

There was no significant difference in general data of gender, age and type of illness between the two groups (P > 0.05). They are shown in Table 1.

Group	Control group (75)	Observation group (75)	T/χ^2	P
Gender				
Male	42(56.00)	46(61.33)	0.440	0.507
Female	33(44.00)	29(38.67)		
Age	59.33±7.18	61.27±7.02	1.673	0.096
Types of illness			0.955	0.812
Headache and dizziness	31(41.33)	29(38.67)		
Dysarthria	10(13.33)	13(17.33)		
Hemiplegia	5(6.67)	7(9.33)		
Others	29(38.67)	26(34.67)		

Table 1: Comparison of the general data in the two groups [n (%), (mean±SD)].

Comparison of therapeutic indexes in the two groups

Before treatment, the NIHSS scores in the observation group were slightly lower than those in the control group, the ADAS-Cog scores in the observation group were slightly higher than those in the control group, and there was no significant difference (P>0.05). After 2 weeks of treatment, the NIHSS and ADAS-Cog scores in the two groups were decreased, but the NIHSS and ADAS-Cog scores in the observation group were lower than those in the control group, and the difference was statistically significant (P<0.05). The detailed information is shown in Table 2.

Group	NIHSS		ADAS-Cog	
	Before treatment	After treatment	Before treatment	After treatment
Control group (75)	13.68±3.27	10.04±2.51	59.03±8.84	42.83±5.52
Observation group (75)	12.92±3.09	6.33±2.28	61.27±8.59	34.71±5.49
T	1.463	9.475	1.574	9.033
P	0146	0.000	0.118	0.000

Table 2: Comparison of therapeutic indexes in the two groups (mean±SD, score).

The levels of PLT and FIB and markers of blood viscosity before and after treatment in the two groups

The levels of PLT and FIB before treatment in patients in the two groups were compared, and there was no significant difference (P>0.05). After treatment, the levels of PLT and FIB in the two groups were decreased. The comparison showed the levels of PLT and FIB in the observation group were significantly lower than those in the control group (P < 0.05).

There were no significant differences in the whole blood high-shear relative viscosity, whole blood low-shear relative viscosity and plasma viscosity before treatment in patients in the two groups. After treatment, the whole blood high-shear relative viscosity, whole blood low-shear relative viscosity and plasma viscosities in the two groups were decreased. The comparison showed the whole blood high-shear relative viscosity, whole blood low-shear relative viscosity and plasma viscosity in the observation group were significantly lower than those in the control group (P < 0.05). The detailed information is shown in Table 3.

Comparison of outcomes in the two groups

After the 1-year follow-up, the recurrence rate in the observation group was significantly lower than that in the control group, and the difference was statistically significant (P < 0.05). There were no statistically significant differences in disability and mortality in patients in the two groups (P > 0.05). The detailed information is shown in Table 4.

Indexes	Control group (75)	Observation group (75)	T	P
FIB(g/L)				
Before treatment	4.90±0.36	4.79±0.41	1.746	0.083
After treatment	4.06±0.42	3.24±0.37	12.687	0.000
PLT (10^9/L)				
Before treatment	199.46±32.27	205.35±29.38	1.169	0.244
After treatment	183.39±27.38	173.54±29.46	0.121	0.036
Blood viscosity markers				
Platelet aggregation rate (%)				
Before treatment	65.68±8.75	66.76±7.80	0.798	0.426
After treatment	56.24±7.84	49.76±6.83	5.397	0.000
Whole blood high-shear relative viscosity (mPa.s)				
Before treatment	5.85±0.68	6.04±0.71	1.674	0.096
After treatment	4.92±0.49	4.22±0.61	7.748	0.000
Whole blood low-shear relative viscosity (mPa.s)				
Before treatment	8.31±1.73	8.28±1.64	0.109	0.913
After treatment	7.21±1.22	6.38±1.28	4.065	0.000
Plasma viscosity (mPa.s)				
Before treatment	1.97±0.36	2.08±0.45	1.653	0.100
After treatment	1.21±0.30	1.07±0.38	2.504	0.041

Table 3: The levels of PLT and FIB and markers of blood viscosity before and after treatment in the two groups (mean±SD).

Group	Recurrence rate	Disability	Mortality
Control group (75)	8 (10.67)	2 (2.67)	3 (4.00)
Observation group (75)	1 (1.33)	1 (1.33)	1 (1.33)
χ^2	5.792	0.340	1.027
P	0.016	0.560	0.311

Table 4: Comparison of outcomes in the two groups (n, %).

Comparison of adverse reactions in patients in the two groups

The percentage of patients with skin rash, nausea and vomiting in the observation group was slightly lower than that in the control group; the percentage of patients with upper gastrointestinal hemorrhage, low platelet counts and other adverse reactions (i.e., common slight adverse reactions such as headache, fever and itching) in the observation group was slightly higher than that in the control group, but the differences were not statistically significant ($P > 0.05$). The total incidence of adverse reactions in the observation group was slightly higher, and there were no statistically significant differences in total incidence rate between the two groups ($P > 0.05$). There were no serious hemorrhages or other serious adverse reactions in the two groups. The detailed information is shown in Table 5.

Group	Skin Rash	Nausea and Vomiting	Upper Gastrointestinal Hemorrhage	Low Platelet Counts	Other complications	Total incidence rate
Control group (75)	2(2.67)	2(2.67)	2(2.67)	2(2.67)	1(1.33)	9(12.00)
Observation group (75)	2(2.67)	1(1.33)	3(4.00)	3(4.00)	2(2.67)	11(14.67)
χ^2						0.231
P						0.631

Table 5: Comparison of adverse reactions in patients in the two groups (n, %).

Discussion

Posterior circulation ischemic stroke is caused by basilar artery occlusion or, in most cases, caused by arterial occlusion due to dissecting atheromatous plaque from the artery. This disease lacks typical clinical features and is mostly diagnosed in the elderly[12-13]. The posterior circulation blood supply region is an important brain structural part including the thalamus and the brainstem and is closely associated with the respiratory center, the cardiovascular motor center and the ascending activating system to maintain consciousness; thus, posterior circulation blood supply area ischemia can easily lead to clinical syndromes such as posterior cerebral artery infarction and dorsolateral bulbar syndrome[14-16]. Therefore, it is important to stabilize pathogenetic conditions in time, maintain patency of respiratory tract and blood circulation, and improve cerebral blood flow for prevention of clinical symptoms of cerebral infarction[17].

Aspirin is a traditional platelet aggregation inhibitor, which mainly inhibits the production of platelet prostaglandin cyclooxygenase and formation of thromboxane A2 (TXA2) and then exerts anti-platelet aggregation effects[18]. However, aspirin also has deficiencies. For example, it easily interacts with nonsteroidal anti-inflammatory drugs, which then causes aspirin resistance and further leads to alimentary tract hemorrhage and renal function impairment complications[19-21]. Clopidogrel is a common clinical medication, which has inhibitory effects on platelet aggregation; it works by selectively blocking the binding of platelet receptor with adenosine diphosphate (ADP), then inhibits activated ADP-mediated glycoprotein GPIIIb/IIIa compounds, and thereby inhibits platelet aggregation[22-23].

In addition, Clopidogrel may block the increase of activated platelet caused by released ADP and inhibits the induction of other activators in platelet aggregation.

The study showed that Clopidogrel not only can resist platelet aggregation and has an anti-inflammatory effect but also can maintain the stability of vascular endothelium, protect vulnerable plaque, and reduce the possibility of acute progressive cerebral infarction, and thereby enhance the antiplatelet aggregation effect. Previous studies have proven that aspirin combined with clopidogrel has a synergistic effect in the treatment of stroke[24].

The study showed that there was no significant difference in NIHSS score and ADAS-Cog score between patients before treatment. After treatment, the NIHSS and ADAS-Cog scores of patients decreased significantly. However, the NIHSS scores (6.33±2.28) and DAS-Cog scores (34.71±5.49) of the patients with dual antiplatelet therapy were significantly lower than the NIHSS scores (10.04±2.51) and ADAS-Cog scores (42.83±5.52) of patients with aspirin therapy. The results in this study are consistent with the findings in the literature[25]. Before treatment, there was no statistically significant difference in the levels of PLT and FIB and markers of blood viscosity between the patients ($P > 0.05$). After treatment, the levels of PLT and FIB in the patients were decreased significantly, and the blood viscosity indexes were also decreased significantly. After treatment, the levels of PLT and FIB in the patients with dual antiplatelet therapy were significantly lower than those in the patients with aspirin therapy ($P < 0.05$); the whole blood high-shear relatively viscosity (4.22±0.61 mpa.s), whole blood low-shear relatively viscosity (6.38±1.28 mpa.s) and plasma viscosity (1.07±0.38 mPa.s) in the observation group were significantly lower than those in the control group (whole blood high-shear relatively viscosity: 4.92±0.49; whole blood low-shear relatively viscosity: 7.21±1.22; plasma viscosity: 1.21±0.30) ($P<0.05$). This showed that dual antiplatelet therapy in patients with posterior circulation ischemic stroke can effectively improve the levels of PLT and FIB and improve the blood hypercoagulability and blood flow rheological property markers[26-27]. After the 1-year follow-up, the recurrence rate, disability and mortality of patients with dual antiplatelet therapy were slightly lower than those of patients with single aspirin, but there was no statistical significance; this probably was associated with shorter follow-up time. There was no significant difference between the incidence rate of adverse reactions of patients with dual antiplatelet therapy and that of patients with single aspirin (14.67% vs 12.00%). The results suggested that the synergistic effect of the two medications can effectively improve the curative effect of patients by anti-platelet aggregation, reduction of thrombosis, effectively increasing blood flow in the ischemic region, promoting the effective recovery of patients and improving the outcomes in patients with posterior circulation ischemic stroke

In conclusion, the dual antiplatelet therapy for patients with posterior circulation ischemic stroke can improve the neurological function of patients, has favorable outcomes and a high safety profile and is worthy of clinical application.

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