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Effect of Tirofiban Combined with Neurointerventional Therapy on Neurological Function of Patients with Acute Cerebral Infarction

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Abstract: *Objective:* To analyze the effect of tirofiban combined with neurointerventional therapy on neurological function of patients with acute cerebral infarction. *Methods:* 70 patients with acute cerebral infarction admitted between January 2022 and January 2023 were selected as research objects, and patients were divided into control group (neurological interventional therapy) and experimental group (tirofiban combined with neurointerventional therapy) according to the computerized grouping method. The treatment outcomes of both groups were compared. *Results:* The efficacy of the treatment in the experimental group was 94.9%, which was significantly higher that of the control group control group at 77.14% ($P < 0.05$). Before treatment, there was no difference in the National Institutes of Health Stroke Scale and Barthel Index between the control group and the experimental group ($P < 0.05$); after treatment, the NIHSS score of the experimental group was lower than that of the control group, and the BI score was higher than that of the control group ($P < 0.05$). Before treatment, there was no difference in the levels of high-sensitivity C-reactive protein (hs-CRP) and neuron-specific enolase (NSE) between the control group and the experimental group ($P < 0.05$); after treatment, the levels of hs-CRP and NSE in the experimental group were lower than those in the control group ($P < 0.05$). Before treatment, there was no difference in the levels of carbon monoxide (CO), endothelin (ET), nerve growth factor (NGF), and myelin basic protein (MBP) between the control group and the experimental group ($P < 0.05$); after treatment, the levels of CO and NGF in the experimental group were higher than those in the control group, and the levels of ET and MBP were lower than those in the control group, with statistical significance ($P < 0.05$). *Conclusion:* Tirofiban combined with neurointervention is effective in treating acute cerebral infarction. It can not only control the development of the disease, but also improve the patients' neurological function and the quality of life.

Keywords: Tirofiban; Neurological intervention; Acute cerebral infarction; Neurological function

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1. Introduction

Acute cerebral infarction has the characteristics of acute onset, rapid disease progression, and high disability rate. It occurs when external factors lead to a disruption in the brain's blood supply, resulting in local tissue hypoxia and ischemia. This substantially elevates the risk of neurological deficits [1]. At present, the etiology

of acute cerebral infarction is not very clear. It is generally believed that it is closely related to cerebral arteriosclerosis and genetics [2]. Neurointervention is a common method for the treatment of acute cerebral infarction. It involves unblocking large blood vessels in brain. Although this procedure can control the progression of the disease, it damages the vascular endothelium and cause thrombus fragmentation, making overall therapeutic effect rather unsatisfactory [3]. In this study, we analyzed the effect of tirofiban combined with neurological intervention on the neurological function of patients with acute cerebral infarction.

2. General information and methods

2.1. General information

A total of 70 patients with acute cerebral infarction admitted between January 2022 and January 2023 were selected as the research objects, and the patients were divided into a control group and an experimental group through the computer grouping method. Inclusion criteria: (i) patients who meet the diagnostic criteria for acute cerebral infarction established by the Academic Conference on Cerebrovascular Diseases [4], (ii) patients who were diagnosed with acute cerebral infarction by CT and other examinations, (iii) patients who were not treated with thrombolysis beyond the time window, (iv) patients who were informed of the study. Exclusion criteria: (i) patients with recent history of trauma or surgery, (ii) patients with coagulation disorders, (iii) patients who had taken anticoagulant drugs near the duration of the study, (iv) patients with contraindications to the treatment plan. In the control group, there were 20 male patients and 15 female patients, aged 51–78 years, with an average of 64.50 ± 4.68 years old, and the time from onset to admission was 1–7 hours, with an average of 4.00 ± 0.87 hours; in the experimental group, there were 19 male patients and 16 female patients, aged 51–76 years, with an average of 63.50 ± 4.65 years old, and the time from onset to admission was 2–8h, mean 5.00 ± 0.89 hours. The above data information was entered into statistical software for comparison, and the results showed that there was no difference ($P > 0.05$).

2.2. Methods

The treatment method for patients in the control group was neurointerventional therapy: the patients were instructed to lie in a supine position, and local anesthesia or general anesthesia was given depending on the condition of the patients. The Envoy6F catheter sheath was inserted through the femoral artery using the Seldinger method. The lesion site was viewed through angiography, and the collateral circulation and intracranial blood vessel were observed. A microcatheter was inserted into the distal end of the occlusion segment with the help of a microguide wire, and the SolitaireAB stents placed and released after the distal blood vessel was unobstructed. After 10 minutes, when the stent touched the thrombus, the microcatheter and the stent were withdrawn, and 30 mL of blood was drawn through the arterial sheath to prevent the detached thrombus from entering the cerebral artery.

Patients in the experimental group were treated with tirofiban on the basis of the control group: a microcatheter was indwelled after thrombectomy, and 0.1 g/kg min tirofiban was continuously pumped through the microcatheter for 24–36 hours.

2.3. Evaluation of efficacy and other indexes

Evaluation of efficacy [5]: The efficacy of the treatment was evaluated based on the National Institutes of Health Stroke Scale (NIHSS). The treatment was considered significantly effective if the symptoms disappeared, or that the NIHSS score decreased by more than 90%, or the degree of disability was 0. The treatment was considered effective if the symptoms improved, the NIHSS score decreased by 46–90%, or that the degree of disability was 1–3. The treatment was considered ineffective if there was only a slight improvement in the

symptoms or that the symptoms worsened.

Evaluation indicators: (i) the NIHSS score [6] was used to evaluate the neurological impairment, in which the higher the score, the more serious the neurological impairment.; the Barthel index (BI) [7] was used to evaluate the performance in activities of daily living, in which the higher the score, the better the better the performance. (ii) 3 mL of venous blood was drawn in the morning on an empty stomach, and hypersensitive C-reactive protein (hs-CRP) was detected by immunoscatter turbidimetry, and neuron-specific enolase (NSE) was detected by enzyme-linked immunosorbent assay. (iii) The vascular endothelial function and serological indicators of the patients were compared. 3 mL of venous blood was drawn in a fasting state in the morning, and the serum carbon monoxide (CO) was detected by magnesium nitrate reduction method. Besides, endothelin (ET) was detected by radioimmunoassay. The nerve growth factor (NGF) and myelin basic protein (MBP) were detected by enzyme-linked immunosorbent assay.

2.4. Statistical analysis

Data from the study were analyzed using SPSS 22.0. Count-based indicators were tested with the independent sample chi-square test, and quantitative data were presented as mean ± standard deviation and analyzed with a *t*-test. P <0.05 indicated statistical significance.

3. Results

3.1. Efficacy of treatment

In comparison to the control group's total efficacy of 77.14%, the experimental group achieved a significantly higher efficacy of 94.29% ($P < 0.05$), as shown in **Table 1**.

Table 1. Comparison of the total effective rate of treatment between both groups of patients (*n* [%])

Group	Significantly effective	Effective	Ineffective	Total efficacy
Control group	13 (37.14)	14 (40.00)	8 (22.86)	27 (77.14)
Experimental group	24 (68.57)	9 (25.72)	2 (5.71)	33 (94.29)
χ^2				4.200
P				< 0.05

3.2. NIHSS and BI scores

Before treatment, there was no difference in NIHSS and BI scores between the control group and the experimental group ($P < 0.05$); after treatment, the NIHSS score of the experimental group was lower than that of the control group, and the BI score was higher than that of the control group, with statistical significance ($P < 0.05$). Further details are shown in **Table 2**.

Table 2. Comparison of NIHSS and BI scores between both groups of patients (mean ± standard deviation, points)

Group	NIHSS		BI	
	Before treatment	After treatment	Before treatment	After treatment
Control group	15.35 ± 2.66	10.08 ± 1.96	38.73 ± 4.14	78.66 ± 5.87
Experimental group	15.32 ± 2.63	7.30 ± 1.48	38.76 ± 4.11	86.97 ± 6.35
t	0.047	6.697	0.030	5.685
P	> 0.05	< 0.05	> 0.05	< 0.05

3.3. hs-CRP and NSE levels

Before treatment, there was no difference in the levels of hs-CRP and NSE between the control group and the experimental group ($P < 0.05$); after treatment, the levels of hs-CRP and NSE in the experimental group were lower than those in the control group, and the statistical significance was established ($P < 0.05$). See the table below for detailed data.

Table 3. Comparison of serum hs-CRP and NSE levels between both groups of patients (mean ± standard deviation)

Group	hs-CRP (mg/L)		NSE (ng/ml)	
	Before treatment	After treatment	Before treatment	After treatment
Control group	12.75 ± 2.39	9.06 ± 1.75	19.30 ± 3.11	12.27 ± 2.02
Experimental group	12.73 ± 2.36	6.92 ± 1.30	19.33 ± 3.14	8.82 ± 1.60
t	0.035	5.807	0.040	7.921
P	> 0.05	< 0.05	> 0.05	< 0.05

3.4. Vascular endothelial function and serological indicators

Before treatment, there was no difference in the levels of CO, ET, NGF, and MBP between the control group and the experimental group ($P < 0.05$); after treatment, the levels of CO and NGF in the experimental group were higher than those in the control group, and the levels of ET and MBP were lower than those in the control group. Statistics significance was established ($P < 0.05$). Further details are shown in **Table 4**.

Table 4. Comparison of changes in vascular endothelial function and serological indicators between both groups of patients (mean ± standard deviation)

Group	CO (mmol/L)		ET (pg/mL)		NGF (ng/L)		MBP (u/L)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group	46.45 ± 3.81	52.13 ± 4.25	72.23 ± 6.03	51.33 ± 4.13	63.28 ± 5.76	134.24 ± 8.06	11.30 ± 2.03	6.70 ± 1.88
Experimental group	46.43 ± 3.78	65.20 ± 5.89	72.20 ± 6.00	46.21 ± 3.51	63.25 ± 5.73	155.30 ± 8.69	11.31 ± 2.04	4.33 ± 1.03
t	0.022	10.646	0.021	5.589	0.022	0.512	0.021	6.541
P	> 0.05	<0.05	> 0.05	< 0.05	> 0.05	< 0.05	> 0.05	< 0.05

4. Discussion

Acute cerebral infarction is closely related to inflammatory injury and ischemic injury. When the ischemic injury continues to develop, the necrosis in the middle of the brain parenchyma spreads to the periphery, and the area of the ischemic penumbra gradually shrinks, resulting in lipid peroxidation of the cell membrane. As a result, the nerve cells in the brain are affected, causing dysfunction, thus reducing the quality of life of patients [8,9]. The blocked blood vessels should be cleared as soon as possible when cerebral infarction is discovered to improve the prognosis of the patient.

In terms of nutritional factors, NGP plays a role in the growth and development of nerves, inhibits neuron apoptosis and facilitates nerve regeneration by regulating functional protein synthesis, etc [10]. MBP binds lipids within the nerve myelin and stabilizes the myelin structure of the central nervous system. Inflammatory

response plays an important role in the occurrence and development of acute cerebral infarction. Inflammatory factors such as CRP participate in the early reperfusion injury of infarction and the process of secondary inflammatory response [11]. NSE is an enolase distributed in central neurons, which maintains the normal physiological functions of nerve cells. Through the study, it was found that tirofiban increases the efficacy of neurointervention therapy. Through neurointervention, thrombus can be removed and blocked blood vessels can be recanalized. The SolitaireAB stent exhibits excellent flexibility and adaptability, allowing for complete contact with the thrombus within the blood vessel and rapid restoration of blood flow. It achieves a vascular recanalization rate exceeding 80% and significantly reduces the recurrence rate of thrombosis, ultimately enhancing patient prognosis [12,13]. Interventional therapy can expand the time window to 6 hours, and the drug has a higher concentration at the target point. Inserting a microcatheter into the thrombus for thrombolysis has a better effect [14,15]. However, with the flow of blood, the shed emboli can easily block other parts, trigger platelet activation, and re-occlusion of blood vessels is also likely to occur, which affects the treatment results. Tirofiban is a novel reversible non-peptide antithrombotic drug. Tirofiban inhibits fibrinogen-GPII/IIIa binding, enhancing its anti-platelet aggregation effect. It completely prevents platelet activation and aggregation during thrombectomy, reducing thrombus dislodgement and improving vascular recanalization rates. Additionally, tirofiban stimulates endothelial cell migration, aiding in endothelial repair, reducing nerve cell damage, and enhancing nerve function. The combined use of tirofiban and neurointervention provides complementary benefits by effectively inhibiting platelet aggregation, promoting blood vessel recanalization, and enhancing overall quality of life.

5. Conclusion

In conclusion, tirofiban combined with neurointervention is effective in the treatment of acute cerebral infarction. It can not only control the progression of the disease, but also improve neurological function and the quality of life of the patients.

Disclosure statement

The author declares no conflict of interest.

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