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Original Article

Mechanical thrombectomy combined with tirofiban for treatment of acute intracranial atherosclerotic cerebral infarction: clinical observations and effect on serum inflammatory factors

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Abstract: Objective: To evaluate the clinical efficacy of mechanical thrombectomy (MT) combined with tirofiban in treating acute intracranial large artery atheromatous cerebral infarction and its effect on inflammatory factor levels. Methods: This retrospective study included 102 patients with acute intracranial large atherosclerotic cerebral infarction admitted to Jili Hospital, Liuyang City, Hunan Province, between December 2022 and December 2024. Patients were divided into a control group (MT) and an observation group (MT + tirofiban). Pre-treatment and post-treatment assessments included revascularization rate, hypersensitive C-reactive protein (hs-CRP), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), D-dimer (D-D), prothrombin time (PT), activated partial thromboplastin time (APTT), neuron-specific enolase (NSE), central neural specific protein (S100β), and myelin basic protein (MBP). Neurological function was evaluated using the National Institutes of Health Stroke Scale (NIHSS), Mini-Mental State Examination (MMSE), modified Barthel index (MBI), and Fugl-Meyer assessment (FMA). Logistic analysis was used to identify factors influencing vascular re-canalization. Results: After treatment, the revascularization rate was higher in the observation group (94.12% vs. 78.43%, P<0.05). NIHSS, D-D, hs-CRP, TNF-α, IL-6, NSE, S100β, and MBP levels decreased significantly, with lower values observed in the observation group (P<0.05). MMSE, MBI, and FMA scores increased more in the observation group (P<0.05). PT and APTT were prolonged significantly, with higher values in the observation group (P<0.05). Logistic regression identified post-treatment NIHSS score as an independent risk factor for re-occlusion (P<0.05). Conclusion: Tirofiban combined with MT improves revascularization in patients, reduces inflammation and neurobiochemical damage, enhances neurological function, and lowers the risk of re-occlusion in patients with acute intracranial large artery atheromatous cerebral infarction.

Keywords: Acute cerebral atherosclerotic infarction, mechanical thrombectomy, Tirofiban

Introduction

Acute cerebral infarction (ACI) is a cerebrovascular disease with high incidence, disability rate, and fatality rate, showing a trend toward younger ages [1, 2]. Intracranial atherosclerotic cerebral infarction is a significant subtype, and its pathogenesis is complex, with thrombosis resulting from the rupture of intracranial atherosclerotic plaque being the primary cause [3]. The burden of intracranial atherosclerotic plaque is closely linked to the severity of ACI patients, and most ACI patients also have intracranial atherosclerosis, involving multiple arterial segments [4]. At present, mechanical thrombectomy is commonly used in clinical practice and has shown favorable outcomes, including a high vascular recanalization rate. However, the procedure can cause vascular endothelial damage, leading to re-thrombosis or thrombus fragmentation during surgery, which can result in secondary thrombosis when small emboli enter the distal blood vessel, adversely affecting patient prognosis [5]. Therefore, combined antiplatelet therapy is very important during thrombectomy. Tirofiban, a potent platelet aggregation inhibitor, effectively dissolves thrombus and prevents re-thrombosis, offering advantages such as short half-life, quick intravenous administration, and rapid platelet recovery after drug withdrawal [6]. Studies [7] have pointed out that tirofiban com-

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bined with mechanical thrombectomy effectively improves vascular recanalization in the treatment of cerebral infarction. However, there is limited clinical research on the short-term prognosis of patients with acute large atherosclerotic cerebral infarction. This study aimed to investigate the clinical efficacy of Tirofiban combined with mechanical thrombectomy for treating acute intracranial atherosclerotic cerebral infarction and its influence on the level of inflammatory factors.

Materials and methods

General information

This single-center retrospective study was approved by the Medical Ethics Committee of Jili Hospital, Liuyang City, Hunan Province. A total of 102 patients with acute intracranial large atherosclerotic cerebral infarction, admitted to Jili Hospital, Liuyang City, Hunan Province from December 2022 to December 2024, were included in the study. The patients were divided into a control group and an observation group, with 51 cases in each group.

Inclusion criteria: (1) Diagnosis of intracranial large ACI based on established criteria [8]; (2) Availability of complete clinical data; (3) Onset of symptoms within 6 h; (4) Preoperative National Institutes of Health Stroke Scale (NIHSS) >6. Exclusion criteria: (1) Coagulation dysfunction; (2) Severe cardiovascular disease; (3) Allergies or contraindications to the medications used in the study; (4) A history of head trauma or stroke within the past 3 months.

Sample size calculation

According to the literature review, the expected vascular recanalization rate was 70% in the control group and 90% in the observation group. With a significance level (α) of 0.05 and a test power (1-β) of 0.8, sample size calculations using PASS software indicated that at least 29 patients were needed per group. Considering a 10% loss to follow-up, 51 patients were included in each group.

Treatment method

The patients in the control group underwent mechanical thrombectomy. Upon admission, necessary tests were conducted, followed by the procedure. A preoperative head computed tomography (CT) was performed to rule out bleeding complications. The patient was placed supine, and after anesthesia, a microcatheter was inserted through the femoral artery. Cerebral angiography was performed to identify the thrombus and confirm occlusion. An appropriate stent was selected, advanced through the microcatheter to the thrombus, and released upon reaching the occlusion. The stent was held in place for 5-10 minutes, after which both the stent and microcatheter were withdrawn. Post-procedure, conventional treatment, including antiplatelet therapy and brain circulation enhancement, were administered.

Patients in the observation group underwent the same mechanical thrombectomy procedure as the control group, with the addition of tirofiban (Yuanda Medical China Co., LTD., approved by SinopMA H20041165). The initial dose of tirofiban was 10 μg/kg, injected over 3 minutes, followed by continuous intravenous infusion at a rate of 0.1 μg/kg per minute, with a total dose not exceeding 0.5 mg.

Observation index

(1) Vascular recanalization rate: The vascular recanalization rate was assessed using Modified Thrombolysis in Cerebral Infarction (mTICI) scale [9]. Grade 3 indicated complete recanalization, grade 2b indicated effective recanalization, and grades 0-2A indicated no or partial recanalization. The vascular recanalization rate was defined as the sum of the complete and effective recanalization rates.

(2) Neurological impairment: Neurological impairment was assessed using the National Institutes of Health Stroke Scale (NIHSS) score [10] before and 2 weeks after treatment. The scale has a total score of 42, with higher scores indicating more severe neurological dysfunction. The Minimum Mental State Examination (MMSE) [11] was used to assess the mental state of patients, with a total score of 30, where higher scores reflect better cognitive status.

(3) Inflammatory level: Fasting venous blood (5 mL) was collected before and two weeks after treatment. After centrifugation (3,500 rpm, 10 min), serum was extracted to measure the levels of hs-CRP, TNF-α, and IL-6 using enzyme-linked immunosorbent assay on a Bio-Rad 450 automatic enzyme labeler (Bell Corporation, USA).

Table 1. Comparison of general information between the two groups [$\bar{x}$±s, n (%)]

Group	Gender		Age (yrs)	Onset to admission time (h)	Underlying disease		
	female	male			hypertension	diabetes	Hyperlipidemia
Control group (n=51)	22 (43.14)	29 (56.86)	67.90±4.54	3.51±1.01	23 (45.10)	25 (49.02)	22 (43.14)
Observation group (n=51)	23 (45.10)	28 (54.90)	68.73±4.83	3.59±1.08	20 (39.22)	18 (35.29)	19 (37.25)
χ^2/t	0.040		0.888	0.379	0.362	1.970	0.367
P	0.842		0.377	0.705	0.547	0.160	0.545

Table 2. Comparison of vascular recanalization rate between the two groups [n (%)]

Group	Complete vascular recanalization	Effective vascular recanalization	No or partial vascular recanalization	Vascular recanalization rate
Control group (n=51)	19 (37.25)	21 (41.18)	11 (21.57)	40 (78.43)
Observation group (n=51)	27 (52.94)	21 (41.18)	3 (5.88)	48 (94.12)
χ^2				5.299
P				0.021

(4) Coagulation indices: Fasting venous blood (2 mL) was collected before treatment and two weeks after treatment. Plasma was obtained through centrifugation (3,500 r/min, 10 min). The levels of D-D, PT, and APTT were determined using an EC6800 automatic hemagglutination instrument (Duecoslen Medical Technology Co., LTD).

(5) Functional ability: The modified Barthel index (MBI) [12] was used to evaluate patients' ability to perform activities of daily living before and two weeks after treatment. A lower MBI score indicates poorer functional ability. The Fugl-Meyer assessment (FMA) [13] was used to evaluate limb motor function, with a total score of 100; lower the scores indicate greater motor impairment.

(6) Neurobiochemical markers: Blood collection and serum preparation were the same as for inflammatory marker analysis. The levels of neural factors, including neuron-specific enolase (NSE), central nerve-specific protein (S-100β), and myelin basic protein (MBP) were determined by chemiluminescence assays.

(7) Reclosure of blood vessels: Re-occlusion of blood vessels was recorded and compared between groups three months after treatment, using digital subtraction angiography.

Statistical method

Data were processed and analyzed using SPSS 26.0. Measured data were expressed as ($\bar{x}$±s), and comparisons between groups were made using the independent T-test. Counted data were expressed as frequency or rate (%), with comparisons between groups conducted using the Chi-square test. Multivariate logistic analysis was performed to identify the independent risk factors for re-occlusion. A *P*-value of <0.05 was considered significant.

Results

Comparison of general information between the two groups

There were no significant differences in age, gender, or other baseline characteristics between the two groups (P>0.05), indicating comparability (**Table 1**).

Comparison of vascular recanalization rate between the two groups

After treatment, the vascular recanalization rate in the observation group was 94.12%, significantly higher than 78.43% in the control group (P<0.05, **Table 2**).

Comparison of NIHSS and MMSE scores between the two groups

Two weeks after treatment, the NIHSS scores were lower in both groups compared to pre-treatment, with the observation group showing a significantly lower score than the control group (P<0.001). The MMSE score increased in both groups, with the observation group achiev-

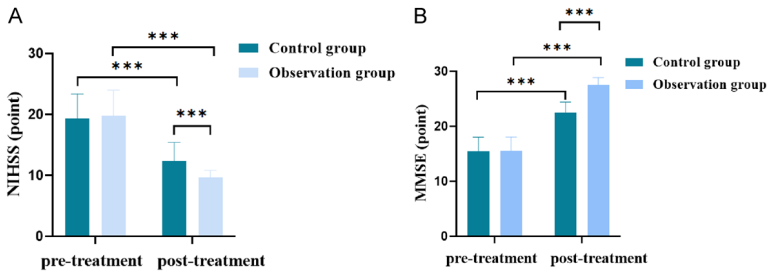

Figure 1. Comparison of NIHSS (A) and MMSE (B) scores between the two groups before and after the treatment. *Note:* NIHSS: National Institutes of Health Stroke Scale; MMSE: Minimum Mental State Examination; ***P<0.001.

ing a significantly higher score than the control group (P<0.001) (**Figure 1**).

Comparison of neurobiochemical markers between the two groups

After treatment, the serum levels of NSE, MBP, and S-100β decreased in both groups. The levels in the observation group were significantly lower than those of the control group (all P<0.001), as shown in **Table 3**.

Comparison of inflammatory marker levels between the two groups

Before treatment, there were no significant differences in the levels of hs-CRP, TNF-α, and IL-6 between the two groups (all P>0.05). After treatment, the levels of hs-CRP, TNF-α, and IL-6 were significantly lower in both groups compared to pre-treatment, with the observation group showing significantly lower levels than the control group (all P<0.001) (**Figure 2**).

Comparison of coagulation indices between the two groups

After treatment, both APTT and PT were prolonged in both groups, with the observation group showing significantly higher values than the control group (P<0.05). After treatment, the D-D level decreased in both groups, and the observation group showed significantly lower levels than the control group (P<0.001) (**Table 4**).

Comparison of MBI and FMA scores between the two groups

Before treatment, there were no significant differences in MBI and FMA scores between the two groups (all P>0.05). After treatment, both MBI and FMA scores were significantly higher compared to those before treatment, and the observation group performed better than the control group (all P<0.001) (**Table 5**).

Multivariate analysis of post-treatment vascular reclosure

Three months after treatment, 11 cases of vascular re-closure were observed in the control group, compared to 3 cases in the observation group (χ^2=5.299, P=0.021). Univariate analysis revealed significant differences in NIHSS, MMSE scores, hs-CRP, TNF-α, IL-6 levels, APTT, and MBI scores between the two groups (P<0.05). Logistic regression analysis, with vascular reclosure as the dependent variable (occurrence =1, non-occurrence =0) and the significant variables from univariate analysis as independent variables, identified post-treatment NIHSS score as an independent variable affecting the occurrence of vascular re-occlusion (P<0.05, **Table 6**).

Typical case

A 67-year-old male was admitted to the hospital with right limb weakness and unclear speech, presenting 5 hours after symptom onset. His NIHSS score was 14, and diagnosis confirmed occlusion of the left internal carotid artery. Preoperative and postoperative CT angiography (CTA) and head CT are shown in **Figures 3, 4**.

Discussion

Acute intracranial atherosclerotic cerebral infarction is a major subtype of ACI. Its pathophysiology mainly involves the rupture of intracranial atherosclerotic plaques, leading to thrombosis, which causes vascular stenosis or occlusion and subsequent ischemic brain injury [14]. This type of cerebral infarction is associated not only with a high incidence but also with significant disability and mortality rates, severely worseningthe prognosis. In addition, the load of intracranial atherosclerotic plaque can influence the severity of the disease, and most patients experience multi-segmental artery involvement [15]. Therefore, it is

Table 3. Comparison of neurobiochemical markers between two groups of patients before and after treatment ($\bar{x}\pm s$)

Group	NSE		MBP		S100β	
	pre-treatment	post-treatment	pre-treatment	post-treatment	pre-treatment	post-treatment
Control group (n=51)	22.31±4.00	11.35±2.16*	12.68±3.46	5.43±1.24*	2.35±0.22	1.63±0.16*
Observation group (n=51)	22.70±2.30	6.33±1.64*	12.32±3.28	3.09±0.97*	2.39±0.22	1.23±0.12*
t	0.555	13.218	0.545	10.604	1.085	14.28
P	0.581	<0.001	0.587	<0.001	0.280	<0.001

Note: Compared with before treatment, *P<0.05; NSE: Neuron-specific enolase; MBP: Myelin Basic protein; S100β: A specific protein for the central nervous system.

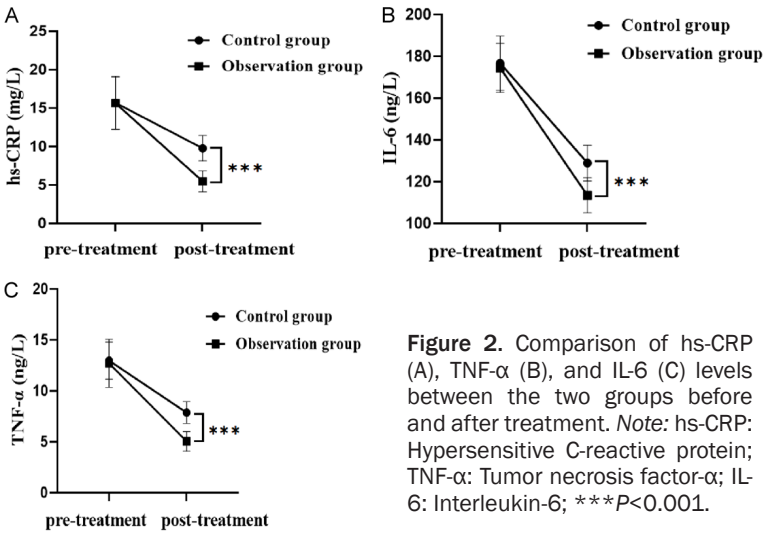

Figure 2. Comparison of hs-CRP (A), TNF-α (B), and IL-6 (C) levels between the two groups before and after treatment. *Note:* hs-CRP: Hypersensitive C-reactive protein; TNF-α: Tumor necrosis factor-α; IL-6: Interleukin-6; ***P<0.001.

of great significance to find a highly effective treatment scheme.

Studies [16, 17] have demonstrated that early endovascular treatment within the therapeutic time window significantly benefits patients with acute large atherosclerotic cerebral infarction. Among the endovascular therapies, mechanical thrombectomy (MT) is widely used to remove thrombi and facilitate rapid vascular recanalization. However, due to the complex structure at the infarction site, many patients require multiple thrombectomy attempts to achieve complete recanalization. Additionally, some patients with chronic metabolic diseases are at a high risk of intraoperative coagulation and bleeding, which adversely affects the prognosis [18]. Relevant studies [19, 20] pointed out that the key to preventing thrombosis is inhibiting platelet aggregation. Without platelet antagonism during MT, there is a risk of thrombus reformation. Tirofiban, an antiplatelet drug, exerts a reversible antagonistic effect on platelet receptors. By binding to glycoprotein receptors on the platelet surface, tirofiban prevents the cross-linking of fibrinogen and platelets, thus inhibiting platelet activation and aggregation, which in turn reduces the likelihood of thrombosis [21]. Zhong et al. [22] conducted a retrospective study on 145 acute ischemic stroke (AIS) patients who received MT and found that adjuvant treatment with tirofiban significantly improved vascular recanalization rates, alleviated symptoms, promoted neurological function recovery, and improved patient prognosis. In this study, the vascular recanalization rate in the observation group was significantly higher than that in the control group, consistent with these findings. The results suggest that tirofiban combined with mechanical thrombectomy offers superior efficacy in enhancing vascular recanalization compared to MT alone. This may be due to the high platelet content of the thrombi, where mechanical thrombectomy clears the obstruction, while tirofiban's platelet antagonism helps prevent re-thrombosis, thereby improving blood perfusion and enhancing the recanalization rate [23].

Studies [24] have shown that patients with acute anterior circulation cerebral infarction often experience neurological impairment and limited ability in daily living, which significantly affects their quality of life. In this study, the NIHSS scores in the observation group were lower after treatment compared to the control group, while MMSE scores, MBI scores, and FMA scores were higher. These results indicate that tirofiban combined with mechanical throm-

Table 4. Comparison of coagulation indices between the two groups before and after treatment ($\bar{x}\pm s$)

Group	D-D		PT		APTT	
	pre-treatment	post-treatment	pre-treatment	post-treatment	pre-treatment	post-treatment
Control group (n=51)	0.70±0.12	0.49±0.08*	10.12±0.68	11.12±0.36*	25.24±4.23	27.82±1.84*
Observation group (n=51)	0.72±0.14	0.36±0.03*	10.14±0.69	12.63±0.39*	24.13±3.93	31.36±1.71*
t	0.945	11.062	0.202	20.186	1.378	9.811
P	0.347	<0.001	0.840	<0.001	0.171	<0.001

Note: Compared with before treatment, *$P<0.05$; D-D: D-dimer; PT: Prothrombin time; APTT: Activated partial thromboplastin time.

Table 5. Comparison of MBI and FMA scores between the two groups before and after treatment ($\bar{x}\pm s$)

Group	MBI		FMA	
	pre-treatment	post-treatment	pre-treatment	post-treatment
Control group (n=51)	40.73±5.35	58.24±5.44*	70.04±5.06	81.59±4.58*
Observation group (n=51)	40.96±4.84	65.08±5.55*	70.37±3.58	89.67±4.09*
t	0.233	6.291	0.384	9.389
P	0.816	<0.001	0.702	<0.001

Note: Compared with before treatment, *$P<0.05$; MBI: Modified Barthel Index; FMA: Fugl-Meyer Assessment.

Table 6. Multivariate logistic analysis of vascular re-occlusion in ACI patients

Variable	*B* value	*SE* value	*Wald* χ^2 value	*P* value	*OR* value (95% *CI*)
NIHSS	0.971	0.360	7.280	0.007	2.640 (1.304-5.343)
MMSE	0.485	0.335	2.091	0.148	1.624 (0.842-3.133)
hs-CRP	-0.329	0.317	1.082	0.298	0.719 (0.387-1.338)
IL-6	0.050	0.059	0.711	0.399	1.051 (0.936-1.181)
TNF-α	0.627	0.489	1.647	0.199	1.873 (0.718-4.88)
APTT	-0.288	0.232	1.547	0.214	0.750 (0.476-1.18)
MBI	0.071	0.103	0.476	0.490	1.073 (0.878-1.312)

Note: NIHSS: National Institutes of Health Stroke Scale; MMSE: Minimum Mental State Examination; hs-CRP: Hypersensitive C-reactive protein; TNF-α: Tumor necrosis factor-α; IL-6: Interleukin-6; APTT: Activated partial thromboplastin time; MBI: Modified Barthel index; ACI: Acute cerebral infarction.

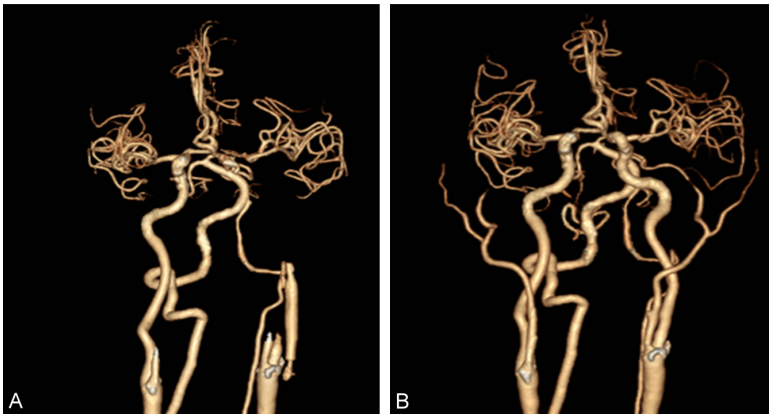

Figure 3. Preoperative and postoperative CT angiography images. A: Preoperative CT angiography; B: Postoperative CT angiography.

bectomy can effectively improve neurological function, mental state, daily living ability, and limb motor function of patients, aligning with the study by Sang [25]. The underlying mechanism may be attributed to tirofiban's specific antagonism of platelet surface receptors. By preventing fibrinogen from binding to glycoprotein IIb/IIIa receptors, tirofiban interferes with platelet aggregation, thus inhibiting thrombus formation [26]. Moreover, the inhibition of tirofiban on receptors is reversible with a short half-life, enabling rapid restoration of platelet function after drug withdrawal, which effectively enhances neurological function recovery in patients [27].

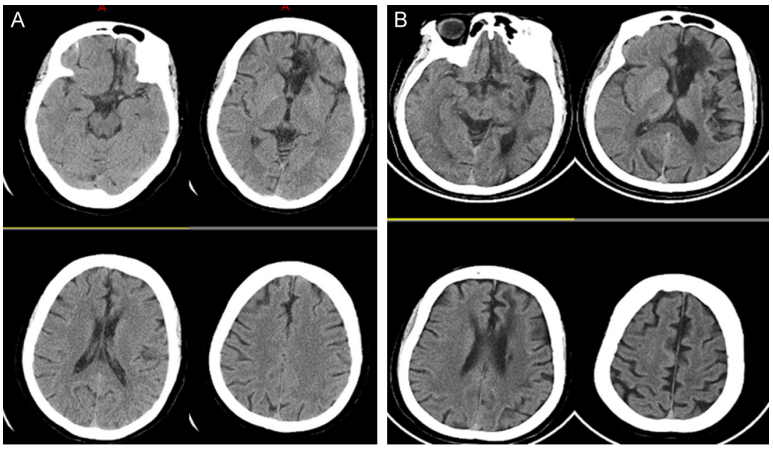

Figure 4. Head CT before and after the operation. A: Preoperative head CT; B: Head CT 3 months after the operation.

NSE, MBP, and S-100β are key neurobiologic markers. NSE has neurotrophic and protective effects, MBP preserves myelin function in the central nervous system, and S100β supports neuronal growth and overall nervous system function. During ischemia and hypoxia, such as in acute intracranial large artery atherosclerotic cerebral infarction, these markers are released into the bloodstream through the blood-brain barrier [28]. In this study, post-treatment levels of these nerve markers were significantly lower in the observation group compared to the control group, indicating that Tirofiban combined with mechanical thrombectomy effectively improved neurological function of patients and promoted their recovery. This effect can be explained by tirofiban's ability to inhibit the release of thromboxane A_2, oxygen free radicals, and serotonin, which act rapidly at the thrombus site to prevent thrombus formation. Tirofiban facilitates the detachment of microthrombi and slows thrombosis formation, reduces vascular endothelial cell damage, enhances cerebral blood flow, and promotes the recovery of cerebral blood circulation, ultimately aiding in neurological recovery [29].

Furthermore, the observation group exhibited significantly lower D-D levels and prolonged APTT and PT, suggesting that the combined therapy effectively improves coagulation function and reduces thrombosis risk. This benefit may stem from the complementary effects of combination therapy, enhancing the overall benefit.

Inflammatory factors are involved in the onset and progression of acute intracranial ACI. Mechanical thrombectomy, while effective in recanalizing occluded vessels, can cause vascular endothelial injury, thereby triggering the release of inflammatory factors. Elevated levels of inflammatory factors can aggravate brain damage and accelerate disease progression. Tirofiban, by reducing ischemia- and hypoxia-induced brain tissue damage, helps mitigate the aggregation of inflammatory factors induced by neuronal lysis [30]. Additionally, it resists platelet aggregation, reduces the production of inflammatory factors during platelet activation, prevents the rupture of vulnerable plaques, and improves blood circulation in brain tissue [31]. In this study, the observation group showed significantly lower levels of inflammatory markers compared to the control group two weeks post-treatment. These results suggest that tirofiban combined with mechanical thrombectomy effectively inhibits the inflammatory response, helping to control disease progression. Furthermore, multivariate analysis revealed that post-treatment NIHSS score was an important factor affecting the occurrence of vascular re-occlusion. This further highlights the advantages of combining tirofiban with mechanical thrombectomy in reducing brain tissue injury and controlling inflammatory response.

Conclusion

Tirofiban combined with mechanical thrombectomy demonstrated significant clinical efficacy in treating acute intracranial ACI. This approach enhances vascular recanalization, improves neurological function and mental state, enhances coagulation function, suppresses inflammatory responses, boosts patients' ability to perform daily activities, and improves limb mobility. Future studies should further validate the long-term effects of this combined therapy and explore its use across various subtypes of cerebral infarction. However, there are still some limitations in this

study, including the relatively small sample size, which may not fully capture the differences in treatment responses across patients with different conditions and constitutions. Future research should aim to expand the sample size, and conduct multi-center studies, to make the findings more representative.

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Disclosure of conflict of interest

None.

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