

Original Article

Edaravone dextrocamphorol combined with intravenous thrombolysis enhances recovery in acute ischemic stroke

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Abstract: Objective: To evaluate the effect of edaravone dextrocamphorol on neurotrophic status and functional recovery in patients undergoing intravenous thrombolysis for acute ischemic stroke. Methods: This single-center retrospective study enrolled 100 patients with acute ischemic stroke. Patients were divided into two groups: the observation group (n = 45), who received both intravenous alteplase and edaravone dextrocamphorol treatment, and the control group (n = 55), who received intravenous thrombolysis with alteplase alone. Clinical indicators that were compared between groups included baseline characteristics, NIHSS scores, nutritional status (albumin, hemoglobin, prealbumin, and transferrin), diet satisfaction, and quality of life (SF-36), and negative psychological symptoms. Results: Compared to the control group, the observation group demonstrated significantly higher diet satisfaction, improved dietary-related symptoms, and better neurological function recovery (all $P < 0.05$). SF-36 scores, including psychological, material, and total functional domains, were also superior in the observation group ($P < 0.05$ for all). Additionally, nutritional status and negative psychological symptoms ($P < 0.05$) improved significantly in the observation group. Conclusion: Edaravone dextrocamphorol, when added to intravenous thrombolysis, significantly enhances recovery in acute ischemic stroke. This strategy improves diet satisfaction, neurological function, quality of life, nutritional status, and psychological well-being.

Keywords: Edaravone dextrocamphorol, acute ischemic stroke, intravenous thrombolysis, neurotrophic status

Introduction

Acute ischemic stroke (AIS) remains one of the leading causes of death and disability worldwide [1, 2]. The current standard of care involves intravenous thrombolysis with recombinant tissue plasminogen activator (rtPA), which restores cerebral perfusion and reduces ischemic brain injury [3-5]. However, despite its proven efficacy, a substantial proportion of patients fail to achieve satisfactory outcomes, with incomplete functional recovery and persistent neurological deficits. This is largely attributed to the complex pathophysiology of AIS, which extends beyond the instant loss of cerebral blood flow to include secondary damages, such as oxidative stress, neuroinflammation, and neuronal apoptosis [6]. These process-

es worsen brain damage and hinder neural repair.

In recent years, increasing attention has been directed toward neuroprotective measures designed to enhance recovery following thrombolysis by targeting the cellular and molecular cascades underlying ischemic injury. Neurotrophic factors play a pivotal role in post-stroke neuroplasticity and functional restoration by promoting neuronal survival, growth, and differentiation [7, 8]. Among them, brain-derived neurotrophic factor (BDNF) and vascular endothelial growth factor (VEGF) are particularly well-studied, with evidence demonstrating their ability to promote neurogenesis and angiogenesis after brain injury [9, 10]. Despite these promising findings, the clinical translation of neurotrophic

factors remains limited due to challenges in delivery and regulatory control.

Edaravone is a potent free radical scavenger shown to reduce oxidative stress and neuronal apoptosis in stroke models [11-13]. Dextrocamphorol, a camphor derivative, has been investigated for its neuroprotective properties, particularly its ability to modulate neuroinflammation and promote neural tissue repair [14-16]. While each agent demonstrates benefits individually, their combined effect on neurotrophic status and functional recovery after intravenous thrombolysis in AIS patients remains limited.

This study aimed to evaluate the effects of edaravone dextrocamphorol on neurotrophic status and the functional outcomes in AIS patients undergoing intravenous thrombolysis. By exploring possible synergistic actions of these agents on the expression of neurotrophic factors and clinical recovery, this study seeks to advance therapeutic strategies for post-stroke management. We hypothesized that edaravone dextrocamphorol would increase neurotrophic support and attenuate secondary brain injury, ultimately improving neurological functional improvement and providing a unique adjunctive approach for optimizing outcomes following thrombolysis in AIS patients.

Methods

Patient inclusion

Sample size calculation

The sample size was estimated using power analysis. The corrected sample size was calculated according to the formula: $N_{\text{corrected}} = N / [1 - (\text{attrition rate} / 100)]$ [22]. Considering attrition, the final target enrollment was approximately 100 patients.

A total of 100 AIS patients who underwent intravenous thrombolysis between January 2022 and December 2024 were retrospectively enrolled. Based on treatment regimens, 45 patients who additionally received edaravone dextrocamphorol were assigned to the observation group, and 55 patients treated with alteplase alone constituted the control group. The study protocol was approved by the Ethics Committee of the Third Clinical Hospital

Affiliated to Changchun University of Traditional Chinese Medicine.

Inclusion and exclusion criteria

Inclusion criteria: (1) Age ≥ 18 years; (2) Diagnosis of AIS in accordance with the *Diagnostic Guidelines for Acute Ischemic Stroke in China (2018 Edition)*, confirmed by cranial CT and/or MRI [17]; (3) Presentation within the intravenous thrombolysis time window (≤ 4.5 hours from symptom onset); (4) Receipt of intravenous rt-PA (recombinant tissue plasminogen activator) as part of their treatment plan; (5) Availability of complete clinical records required for this study.

Exclusion criteria: (1) History of significant head trauma or ischemic stroke within the preceding 3 months; (2) Suspected subarachnoid hemorrhage or other major intracranial pathology; (3) Arterial puncture at a non-compressible site within the past week; (4) History of intracranial hemorrhage or other significant hemorrhagic events; (5) Presence of intracranial tumors, arteriovenous malformations, or aneurysms; (6) Recent intracranial or intraspinal surgeries (≤ 6 months); (7) Uncontrolled hypertension at baseline: systolic blood pressure ≥ 180 mmHg, and/or diastolic blood pressure ≥ 100 mmHg; (8) Active internal bleeding or a known bleeding disorder; (9) Platelet count $< 100 \times 10^9/L$ or abnormal coagulation values (prolonged prothrombin time (PT) or activated partial thromboplastin time (aPTT)); (10) Baseline blood glucose < 2.8 mmol/L or > 22.22 mmol/L; (11) Severe organ failure (liver, kidney, heart, lung, or brain), including end-stage renal disease or cirrhosis; (12) Pregnancy or lactation; (13) Severe cognitive impairment or a history of dementia precluding reliable neurocognitive assessment.

Methods

Control Group: Patients received intravenous thrombolysis with alteplase (Boehringer Ingelheim, product approval number SJ20160054; specification: 20 mg). The alteplase dose was 0.9 mg/kg (maximum 90 mg), with 10% of the total dose administered as an intravenous bolus over 1 minute, followed by infusion of the remaining dose over 1 hour. This regimen adhered to standard AIS treatment protocols

aimed at restoring cerebral perfusion and minimizing ischemic damage.

Observation Group: In addition to the above regimen, patients received edaravone dextrocamphorol (Shensheng Pharmaceutical Co., Ltd., product approval number H20200007; specification: 5 mL/vials containing 10 mg edaravone and 2.5 mg dextrocamphorol, 6 vials per package). A dose of 15 mL was diluted with 100 mL of normal saline and administered intravenously. The infusion was given twice daily for 14 consecutive days. The dosage regimen was selected to optimize neuroprotective effects while ensuring patient safety and tolerability.

Data collection

Primary outcomes

Nutritional status: Peripheral venous blood (2-3 mL) was collected after overnight fasting at baseline and 1 month post-intervention. Samples were centrifuged at 3,000 r/min (radius 3 cm) to separate serum. Serum levels of prealbumin (PA), albumin (ALB), hemoglobin (Hb), and transferrin (TRF) were measured using enzyme-linked immunosorbent assay (ELISA).

Quality of life was assessed with the 36-Item Short Form Health Survey (SF-36) [16], covering five dimensions of physical function, psychological function, social function, common symptoms, and treatment-related side effects. Each dimension is scored from 0 to 100, with higher scores indicating better quality of life.

Secondary outcomes

Neural function was evaluated using the National Institutes of Health Stroke Scale (NIHSS) [17] at baseline and 12 weeks post-intervention. The scale consists of 11 items (e.g., consciousness, neglect, dysarthria, limb movement), with a total score of 42 points. Lower scores indicate better neurological recovery.

Dietary satisfaction was measured using the dietary satisfaction subscale of a validated quality-of-life questionnaire [18]. The subscale includes 6 items rated on a 4-point Likert scale (1 = never, 2 = occasionally, 3 = sometimes, 4 = often). Jospe MR et al. [19] demonstrated

good internal consistency (Cronbach's $\alpha = 0.83$) and construct validity (KMO = 0.676, $P < 0.01$).

Negative psychological status was assessed with both subjective and objective scales: the Self-Rating Anxiety Scale (SAS) [20] and the Hamilton Anxiety Scale (HAMA) [21], respectively. SAS scores were classified as follows: < 40 , no anxiety; 40-47, mild anxiety; 48-55, moderate anxiety; > 56 , severe anxiety.

Statistical analysis

All statistical analyses were performed using SPSS 26.0 software. Categorical variables were presented as frequencies and percentages, and between-group comparisons were conducted using the chi-square test. Continuous variables with normal distribution were expressed as mean \pm standard deviation (SD), and analyzed using the independent-samples t-test (between groups) or paired t-test (within groups). A p -value < 0.05 (bilateral) was considered significant.

Results

Clinical characteristics

In the observation group, there were 31 males and 14 females, aged 20-70 years, with an average disease course of 54.36 ± 1.67 months. In the control group, there were 44 males and 11 females, aged 19-72 years, with an average disease course of 54.27 ± 0.68 months. No significant differences were observed between the two groups in terms of age, sex, smoking history, hypertension, diabetes, atrial fibrillation, hyperlipidemia, infarction in anterior circulation vessels, infarction in posterior circulation vessels, hyperhomocysteinemia, or coronary heart disease (**Table 1**).

Diet satisfaction

Prior to the intervention, diet satisfaction did not differ significantly between the two groups ($P = 0.224$). After intervention, the observation group showed a significant increase in diet satisfaction compared with the control group ($P < 0.001$) (**Table 2**).

Dietary-related symptoms and neurological function

At baseline, no significant differences were observed in dietary-related symptom scores

Table 1. Comparison of clinical characteristics between the two groups

	Observation group (n = 45)	Control group (n = 55)	t/ χ^2	P
Age	54.36±1.67	54.27±0.68	0.336	0.737
Gender			1.630	0.202
Male	31 (68.89%)	44 (80.00%)		
Female	14 (31.11%)	11 (20.00%)		
Smoking history	14 (31.11%)	17 (30.91%)	0.004	0.983
Hypertension	14 (31.11%)	23 (41.82%)	1.217	0.270
Diabetes	12 (26.67%)	14 (25.45%)	0.019	0.891
Atrial fibrillation	3 (6.67%)	6 (10.91%)	0.544	0.461
Hyperlipidemia	9 (20.00%)	6 (10.91%)	1.604	0.205
Infarction in anterior circulation vessel	39 (86.67%)	44 (80.00%)	0.780	0.377
Infarction in posterior circulation vessel	6 (13.33%)	11 (20.00%)	0.781	0.376
Hyperhomocysteinemia	4 (0.89%)	5 (9.09%)	0.001	0.972
Coronary heart disease	5 (11.11%)	5 (9.09%)	0.112	0.738

Table 2. Comparison of diet satisfaction between the two groups

	Observation group (n = 45)	Control group (n = 55)	t	P
Before intervention	10.56±1.78	10.95±1.41	1.224	0.224
After intervention	18.22±1.99	12.89±2.27	12.335	< 0.001
t	19.286	5.395	-	-
p	< 0.001	< 0.001	-	-

Table 3. Comparison of dietary-related symptoms and NIHSS scores between the two groups

Group		Control group (n = 55)	Observation group (n = 45)	t	P
Dietary related symptoms	Before intervention	12.73±3.21	13.24±3.66	0.752	0.124
	After intervention	11.49±4.19	9.09±3.37	3.109	0.002
	t	1.737	5.601	-	-
	p	0.085	< 0.001	-	-
NIHSS Scores	Before intervention	18.29±1.51	18.20±1.38	0.312	0.756
	After intervention	12.51±1.02	7.49±1.24	22.294	< 0.001
	t	23.545	38.858	-	-
	p	< 0.001	< 0.001	-	-

Note: NIHSS: National Institutes of Health Stroke Scale.

between the two groups ($P > 0.05$). After intervention, the observation group exhibited significantly greater improvement compared to the control group ($P < 0.001$).

Similarly, although both groups showed significant reductions in NIHSS scores post-intervention, the improvement in the observation group was significantly greater than in the control group ($P < 0.001$) (Table 3).

Nutritional status

Before intervention, no significant differences were found in PA, ALB, Hb, or TRF levels

between the two groups (all $P > 0.05$). After intervention, patients in the observation group exhibited significantly higher levels of PA, ALB, Hb, and TRF compared to the control group ($P < 0.001$) (Figure 1).

Negative psychological status

After the intervention, both groups showed reductions in SAS and HAMA scores compared to those at baseline. However, the decrease in negative psychological scores was significantly greater in the observation group than in the control group ($P < 0.05$) (Figure 2).

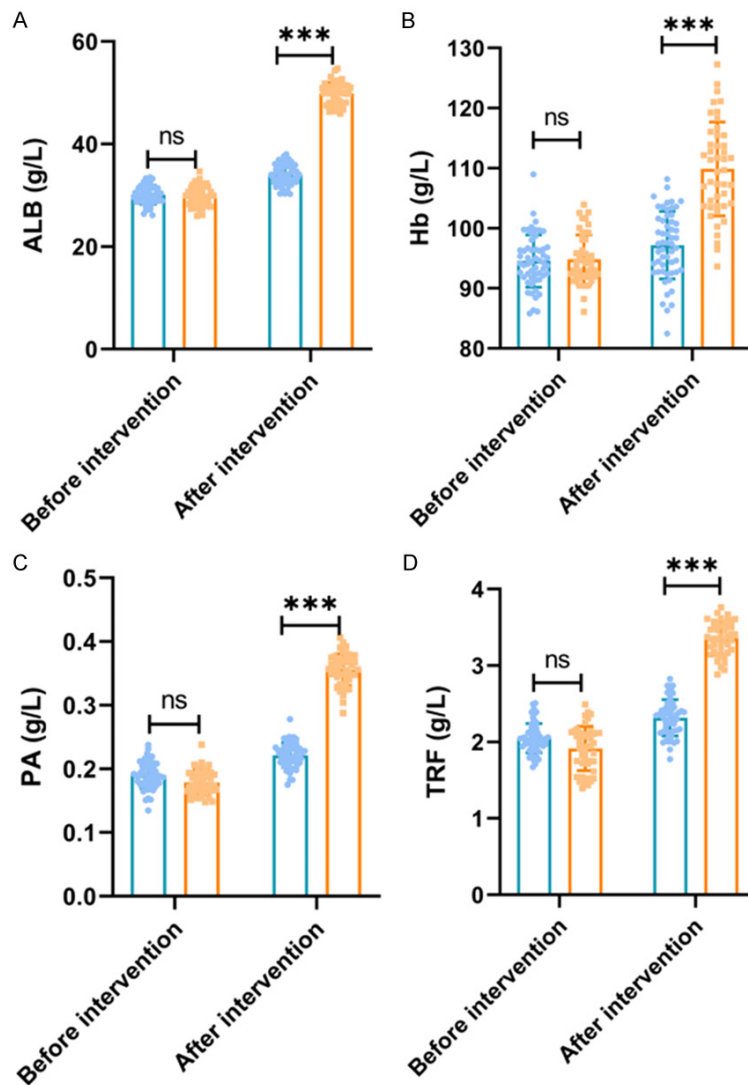


Figure 1. Comparison of nutritional status between the two groups. (A) ALB, (B) Hb, (C) PA, (D) TRF. Note: ALB: albumin; Hb: hemoglobin; PA: prealbumin; TRF: transferrin. ^{ns}P > 0.05, ***P < 0.001, compared to the control group.

Liver function

No significant differences were observed in ALT, AST, or TBIL levels between the two groups, either before or after the intervention (all P > 0.05) (**Figure 3**).

Blood coagulation values

PT, aPTT, international normalized ratio (INR), fibrinogen, and D-dimer levels were between the two groups (**Figure 4**). No significant differences were found within or between groups before and after treatment (all P > 0.05), indicating that the addition of edaravone-dextro-

camphorol to intravenous thrombolysis did not affect coagulation function.

Quality of life

After intervention, patients in the observation group showed significantly higher quality-of-life scores across all domains compared to the control group (P < 0.05) (**Figure 5**). These results suggest that combined therapy led to superior quality-of-life outcomes relative to thrombolysis alone.

Discussion

The present study demonstrated that edaravone dextrocamphorol improved diet satisfaction, dietary-related symptoms, neurological function, quality of life, nutritional status, and psychological well-being in patients with acute ischemic stroke undergoing intravenous thrombolysis. These results highlight the potential of combining neuroprotective therapy with standard thrombolytic treatment to enhance post-stroke recovery, addressing both physical and psychological dimensions of rehabilitation.

A key observation was the marked improvement in diet

satisfaction and dietary-related symptoms among patients receiving edaravone dextrocamphorol. Previous studies have emphasized the importance of nutritional interventions in stroke recovery, showing benefits in both cognitive function and physical outcomes [23, 24]. Our findings extended this evidence by introducing neuroprotective therapy into the post-thrombolysis management paradigm. The improvement in diet satisfaction may be related to better treatment adherence, improved symptom control, and increased patient-perceived benefits of therapy. These positive perceptions are clinically meaningful, as they can facilitate

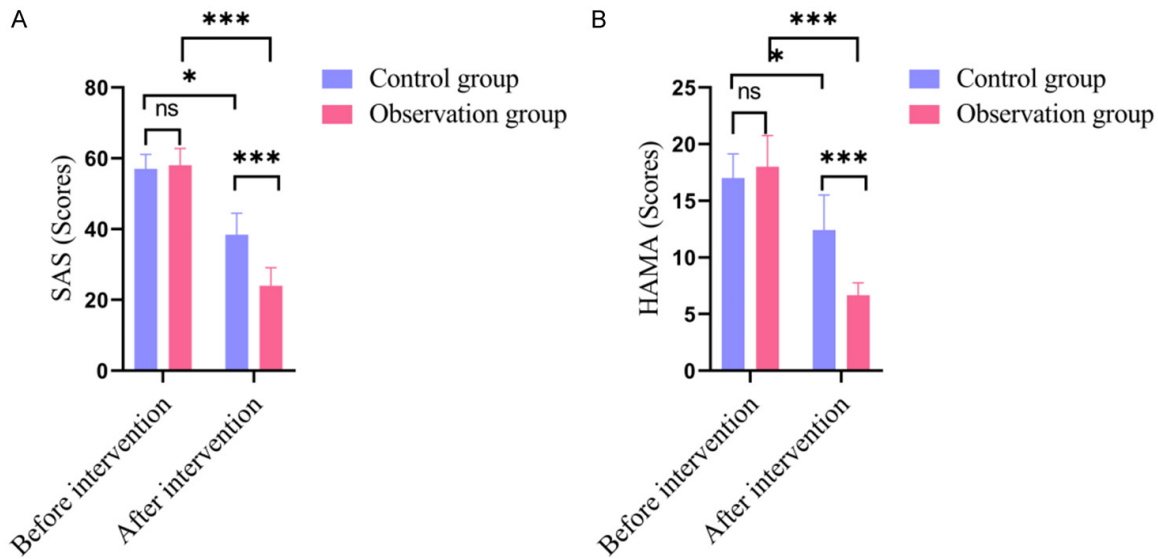


Figure 2. Comparison of negative psychological status between the two groups. (A) SAS, (B) HAMA. Note: SAS: Self-rating Anxiety Scale; HAMA: Hamilton Anxiety Scale. ^{ns}P > 0.05, *P < 0.05, ***P < 0.001, compared to the control group.

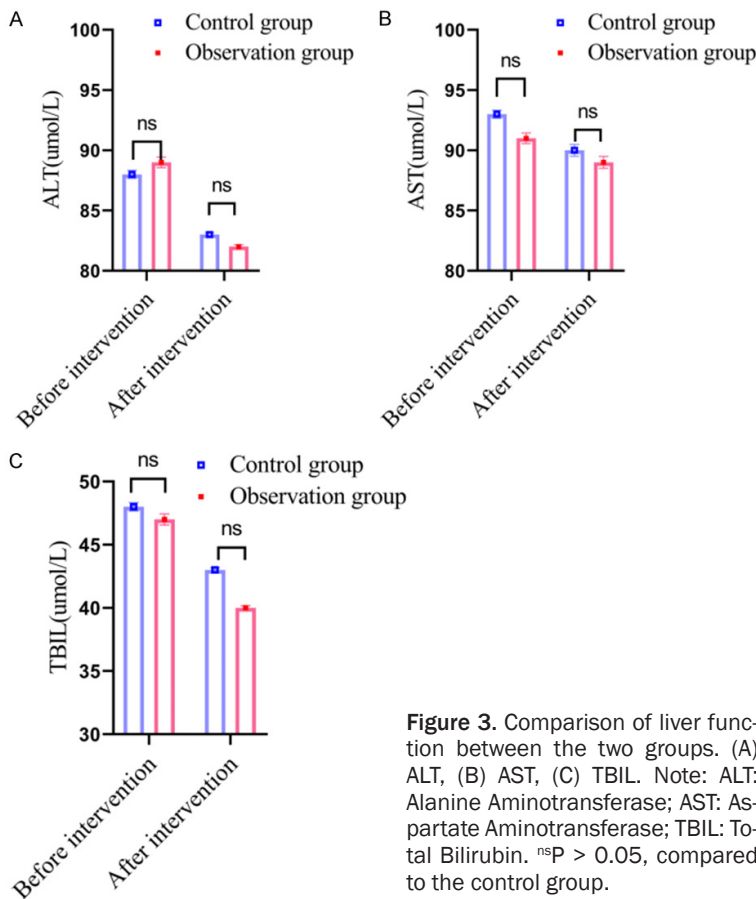
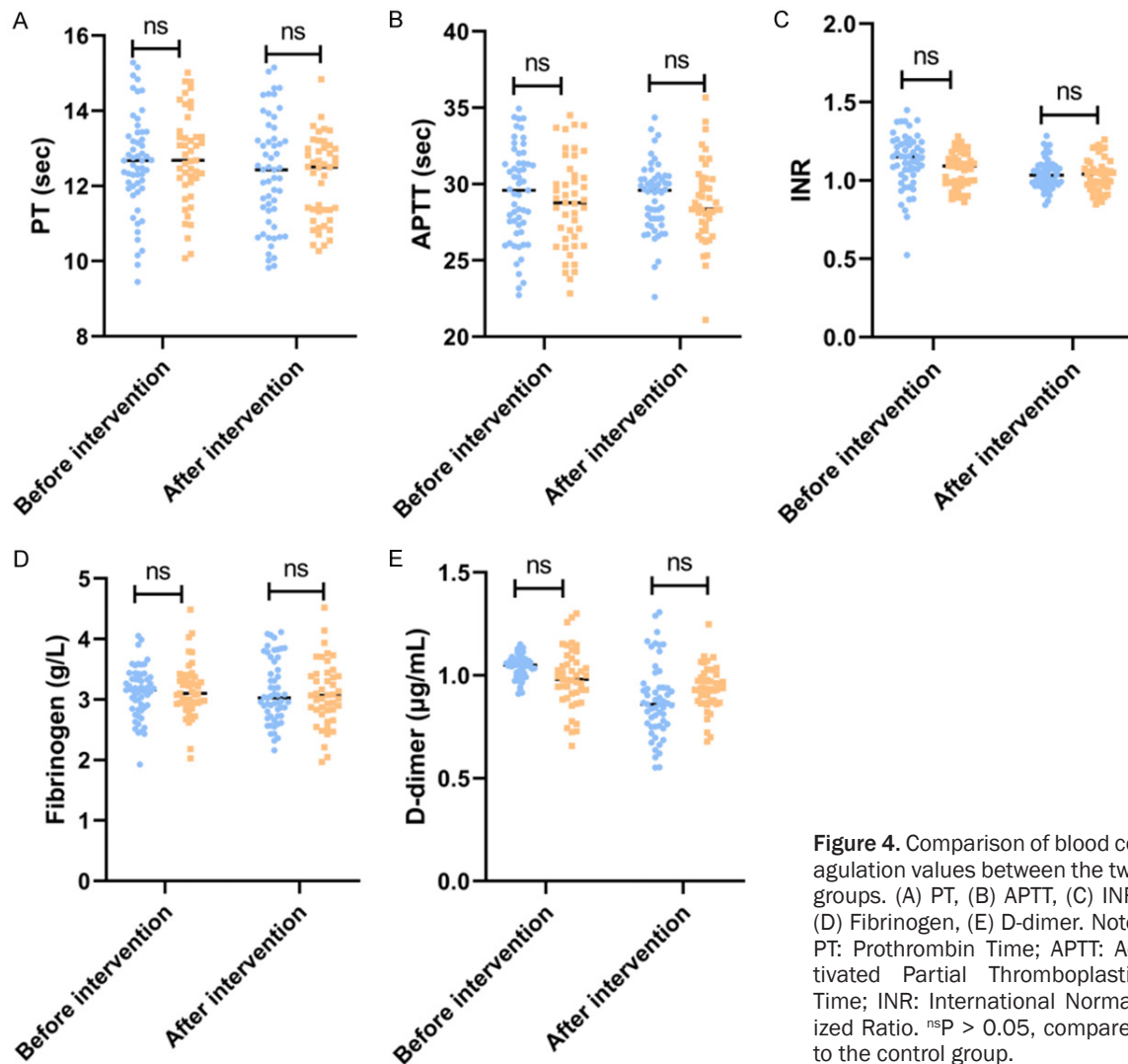


Figure 3. Comparison of liver function between the two groups. (A) ALT, (B) AST, (C) TBIL. Note: ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; TBIL: Total Bilirubin. ^{ns}P > 0.05, compared to the control group.

Regarding neurological outcomes, patients in the observation group showed significantly greater improvement than those in the control group. This is corroborated by the existing literature reporting the benefits of combining neuroprotective drugs with rehabilitation strategies to enhance neurological recovery after stroke [25]. The mechanism may be related to the neuroprotective role of the edaravone dextrocamphorol. Edaravone has been shown to alleviate oxidative stress, limit neuronal injury, and promote neuronal repair and regeneration in ischemic brain tissue [26]. These effects may explain the superior functional outcomes observed in the intervention group. Collectively, this reinforces the value of adding pharmacological neuroprotection to conventional rehabilitation measures to optimize stroke recovery.

sustained engagement with rehabilitation and long-term lifestyle adjustments.

Another finding was the significant improvement in quality of life and psychological well-



being among patients in the observation group. Similar to prior research, both dietary and pharmacologic interventions have been shown to improve psychological health and overall quality of life in stroke survivors [27-29]. The combined effect of nutritional support and pharmacological treatment in our study likely contributed to the observed psychological benefits. Moreover, this integrative approach may help alleviate anxiety and depression, which are common among stroke patients [30]. Addressing both physical and psychological domains is critical, as stroke recovery is inherently multidimensional and requires comprehensive management strategies.

Another noteworthy finding was the improvement in nutritional status after the treatment

with edaravone dextrocamphorol in addition to thrombolysis. Specifically, serum albumin and hemoglobin levels increased significantly compared to the control group. These results are consistent with previous studies highlighting the critical role of nutritional support in improving recovery trajectories in critically ill patients, including those with stroke [31, 32]. The potential mechanisms underlying these improvements may involve enhanced nutrient absorption and utilization, which are essential for tissue repair and immune function [33]. Moreover, better nutritional status may improve the body's ability to combat oxidative stress and inflammation, thereby facilitating neurological recovery [34]. These findings underscore the importance of adopting a holistic approach that integrates pharmacologic treatment with nutri-

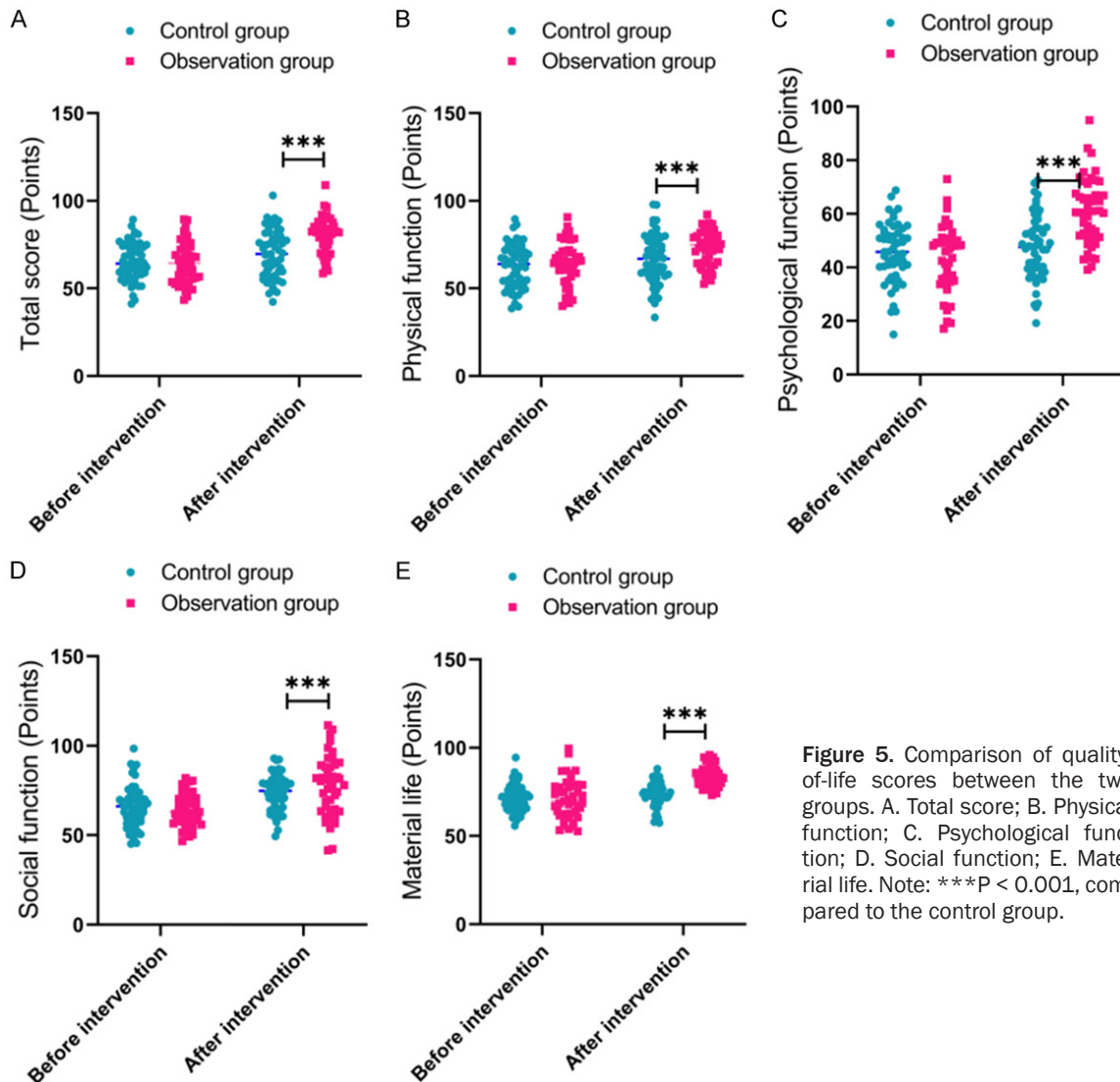


Figure 5. Comparison of quality-of-life scores between the two groups. A. Total score; B. Physical function; C. Psychological function; D. Social function; E. Material life. Note: *** $P < 0.001$, compared to the control group.

tional strategies to optimize post-stroke rehabilitation.

Despite the encouraging outcomes, several limitations should be taken into consideration. First, the sample size was relatively small, which may limit the generalizability of the results. Second, the follow-up period was relatively short, and thus the long-term effects of edaravone dextrocamphorol combined with intravenous thrombolysis remain uncertain. Third, we did not investigate the genetic and molecular mechanisms involved in this intervention. Future studies should involve larger and more diverse populations, adopt longitudinal designs, and explore molecular pathways to better define the therapeutic value of this combined approach.

Conclusion

This study demonstrated that edaravone dextrocamphorol, when combined with intravenous thrombolysis, significantly improved neurotrophic status, neurological function, quality of life, nutritional status, and psychological well-being in patients with acute ischemic stroke. These findings suggest that integrated therapeutic strategies combining pharmacological agents with supportive interventions may enhance post-stroke recovery. However, further research with larger cohorts longer follow-up, and mechanistic exploration is needed to confirm the long-term benefits.

Disclosure of conflict of interest

None.

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