

Effect of Sequential Therapy with Edaravone Dexborneol on Prognosis in Patients with Progressive Cerebral Infarction

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Abstract: *Objective:* To analyze the effects of sequential therapy with edaravone dexborneol on neurological function, inflammatory factors, and prognosis in patients with progressive cerebral infarction. *Methods:* Sixty patients with acute progressive cerebral infarction treated in our hospital from June 2025 to December 2025 were enrolled and divided into two groups by the random number table method, with 30 subjects in each group. The control group received conventional antiplatelet therapy, while the observation group received sequential therapy with edaravone dexborneol in addition to the control treatment. The intervention outcomes were compared between the two groups. *Results:* The total clinical effective rate and improvements in neurological function and inflammatory indicators in the observation group were better than those in the control group, and Hcy, NSE, and the incidence of adverse reactions were lower ($P < 0.05$). *Conclusion:* Sequential therapy with edaravone dexborneol can improve neurological function, reduce inflammation and neurological injury, and has good safety in patients with progressive cerebral infarction, warranting clinical promotion.

Keywords: Edaravone dexborneol; Sequential therapy; Progressive cerebral infarction; Neurological function; Inflammatory factors

Online publication: October 19, 2026

1. Introduction

Progressive cerebral infarction is a type of ischemic stroke. Within 6 hours to 7 days after onset, neurological function may still deteriorate despite active treatment, accounting for approximately 25%–40% of cerebral infarctions ^[1]. Its onset is insidious, and progression is rapid; the infarct focus expands, neurological injury worsens, and disability and mortality rates are high. Inflammatory responses damage the ischemic penumbra. After ischemia, proinflammatory mediators are released, inflammatory cells infiltrate, the blood-brain barrier is disrupted, and neuronal injury is aggravated ^[2]. Timely clearance of inflammatory factors and inhibition of oxidative stress can promote angiogenesis and ameliorate neurological injury. Currently, reperfusion therapy is limited by the time window, and many patients miss the optimal opportunity ^[3]. Neuroprotective therapy

can serve as a supplement. Edaravone dexborneol is composed at a 4:1 ratio and can scavenge free radicals and exert anti-inflammatory effects. Since the launch of sublingual tablets, post-discharge sequential therapy has become possible. This study aimed to investigate its effects on neurological function, inflammatory factors, and prognosis in such patients. The details are as follows:

2. Materials and methods

2.1. General data

Sixty patients with acute progressive cerebral infarction treated in our hospital from June 2025 to December 2025 were enrolled and divided into two groups by the random number table method, with 30 subjects in each group. In the observation group, there were 16 males and 14 females; age ranged from 56 to 78 years, with a mean age of (63.28 ± 2.01) years; 18 had hypertension, 12 had diabetes, and 15 had hyperlipidemia. In the control group, there were 16 males and 14 females; age ranged from 57 to 76 years, with a mean age of (64.01 ± 1.90) years; 17 had hypertension, 11 had diabetes, and 14 had hyperlipidemia. There was no significant difference between the groups ($P > 0.05$). This study was approved by the hospital ethics committee.

2.1.1. Inclusion criteria

- (1) all met the clinical diagnostic criteria for acute progressive cerebral infarction;
- (2) all were definitively diagnosed by CT or MRI;
- (3) neurological deficit signs showed continuous deterioration from 6 h to 7 d after onset;
- (4) patients and their relatives were willing to cooperate with this study.

2.1.2. Exclusion criteria

- (1) concurrent major diseases of the liver, kidney, cardiovascular system, etc.;
- (2) concurrent malignant tumors;
- (3) incomplete follow-up data;
- (4) allergy to the study drugs.

2.2. Methods

The control group received conventional antiplatelet therapy with oral aspirin enteric-coated tablets (manufactured by Bayer HealthCare Co., Ltd.; National Drug Approval No. J20171021; 100 mg per tablet), once daily, 100 mg each time, for 30 consecutive days. Meanwhile, basic regulatory treatment for blood pressure, blood glucose, and blood lipid levels and symptomatic adjunctive therapy were administered.

The observation group received sequential therapy with edaravone dexborneol on the basis of the control treatment. In the initial phase, edaravone dexborneol injection (produced by Simcere Pharmaceutical Co., Ltd.; National Drug Approval No. H20200007; 5 mL per vial) was administered twice daily, 15 mL per dose, by intravenous infusion for 7 consecutive days. From the 7th day onward, edaravone dexborneol sublingual tablets were used for subsequent treatment, twice daily, one tablet sublingually each time, for 4 consecutive weeks. After 14 days from discontinuation of edaravone dexborneol sublingual tablets, a second cycle of sequential therapy was initiated, lasting 14 days.

2.3. Outcome measures

- (1) Clinical efficacy: marked improvement was defined as a reduction in neurological deficit score $\geq 90\%$; effective as a reduction of 50%–89%; ineffective as failure to meet the above criteria or deterioration. Total effective rate = (marked improvement + effective)/total cases $\times 100\%$.
- (2) Neurological deficit score: the criteria of the Academic Conference on Cerebrovascular Diseases were used; the higher the score, the more severe the deficit.
- (3) Inflammatory factors: hs-CRP and IL-6 were measured before and after treatment.
- (4) Hcy and NSE were measured.
- (5) Adverse reactions such as dizziness, fatigue, and somnolence were recorded.

2.4. Statistical analysis

SPSS 26.0 was used. Continuous variables were expressed as mean $\pm$ standard deviation and compared between groups using the *t*-test; categorical data were expressed as n (%) and tested by the chi-square test. $P < 0.05$ was considered a significant difference.

3. Results

3.1. Comparison of clinical efficacy between the two groups

The total effective rate was higher in the observation group ($P < 0.05$), as shown in **Table 1**.

Table 1. Comparison of clinical efficacy between the two groups [n (%)]

Group	<i>n</i>	Marked improvement	Effective	Ineffective	Total effective rate
Observation group	30	18 (60.00)	10 (33.33)	2 (6.67)	28 (93.33)
Control group	30	12 (40.00)	10 (33.33)	8 (26.67)	22 (73.33)
χ^2					4.320
<i>P</i>					0.038

3.2. Comparison of neurological deficit scores between the two groups

After treatment, the neurological function score in the observation group was lower than that in the control group ($P < 0.05$), as shown in **Table 2**.

Table 2. Comparison of neurological deficit scores between the two groups ($\bar{x} \pm s$, points)

Group	<i>n</i>	Before treatment	After treatment	<i>t</i>	<i>P</i>
Observation group	30	22.45 $\pm$ 3.67	8.32 $\pm$ 2.15	18.196	0.000
Control group	30	22.78 $\pm$ 3.82	13.56 $\pm$ 2.87	10.569	0.000
<i>t</i>		0.341	8.004		
<i>P</i>		0.734	0.000		

3.3. Comparison of inflammatory factor levels between the two groups before and after treatment

After treatment, hs-CRP and IL-6 in the observation group were lower than those in the control group ($P < 0.05$), as shown in **Table 3**.

Table 3. Comparison of inflammatory factor levels between the two groups ($\bar{x} \pm s$)

Group	<i>n</i>	hs-CRP (mg/L)	hs-CRP (mg/L)	IL-6 (pg/mL)	IL-6 (pg/mL)
		Before treatment	After treatment	Before treatment	After treatment
Observation group	30	12.56 ± 3.21	4.23 ± 1.15	28.67 ± 5.34	12.45 ± 3.21
Control group	30	12.89 ± 3.45	7.56 ± 1.87	29.12 ± 5.67	18.76 ± 4.12
<i>t</i>		0.384	8.308	0.316	6.617
<i>P</i>		0.703	0.000	0.753	0.000

3.4. Comparison of Hcy and NSE levels between the two groups

After treatment, Hcy and NSE in the observation group were lower than those in the control group ($P < 0.05$), as shown in **Table 4**.

Table 4. Comparison of Hcy and NSE levels between the two groups ($\bar{x} \pm s$)

Group	<i>n</i>	Hcy (μmol/L)	Hcy (μmol/L)	NSE (μg/L)	NSE (μg/L)
		Before treatment	After treatment	Before treatment	After treatment
Observation group	30	18.67 ± 4.23	10.34 ± 2.56	16.78 ± 3.45	8.12 ± 2.01
Control group	30	18.92 ± 4.45	14.67 ± 3.12	17.01 ± 3.67	11.89 ± 2.67
<i>t</i>		0.223	5.876	0.250	6.179
<i>P</i>		0.824	0.000	0.803	0.000

3.5. Comparison of the incidence of adverse reactions between the two groups

The incidence of adverse reactions in the observation group was lower than that in the control group ($P < 0.05$), as shown in **Table 5**.

Table 5. Comparison of the incidence of adverse reactions between the two groups [*n* (%)]

Group	<i>n</i>	Dizziness	Fatigue	Somnolence	Incidence
Observation group	30	1 (3.33)	1 (3.33)	0 (0.00)	2 (6.67)
Control group	30	3 (10.00)	3 (10.00)	2 (6.67)	8 (26.67)
χ^2					4.320
<i>P</i>					0.038

4. Discussion

Progressive cerebral infarction is a common type of ischemic stroke. Its main feature is continuous deterioration of neurological function. Compared with ordinary cerebral infarction, the disease progresses more rapidly. Patients may have only mild symptoms on admission, but within hours to days they may continue to worsen, even to complete paralysis or coma ^[4]. Pathologically, this is related to the continuous loss of the ischemic penumbra. The penumbra is an area around the infarct core that is supplied by collateral circulation, structurally intact but functionally impaired. If blood flow is restored in time, it can survive; if ischemia continues, it becomes part of the infarct. Disease deterioration involves several links: decreased perfusion pressure, thrombus enlargement or embolization, and aggravated inflammatory response. Advanced age, diabetes, blood pressure fluctuation, and fever can also promote progression. Therefore, in addition to

reperfusion therapy, a regimen of continuous neuroprotection is clinically needed.

This study showed that the total effective rate was higher in the observation group. This indicates that sequential therapy with edaravone dexborneol can improve outcomes in patients with progressive cerebral infarction. Efficacy was determined by the degree of reduction in neurological deficit score. The drug can increase the effective rate because it continuously interferes with key links of ischemic injury. In the past, only injection was used, and treatment was interrupted after discharge; residual neurons in the penumbra lost protection and might die due to inflammation or oxidative stress. Sequential therapy ensures continuity of protection from the acute phase to the recovery phase. Intravenous administration acts quickly, scavenging free radicals and inhibiting early inflammation; sublingual tablets maintain stable drug levels, continuously inhibiting delayed inflammation and creating a good environment for neurological recovery. This “acute-phase attack + recovery-phase maintenance” strategy is more consistent with the developmental pattern of the disease. A higher total effective rate means that more patients change from ineffective to effective or markedly improved, which can reduce disability and may also reduce rehabilitation and nursing costs.

After treatment, the neurological deficit score in the observation group was lower. This again demonstrates the neuroprotective effect of this sequential therapy. The greater reduction in scores in this study indicates that the drug can prevent disease progression and promote functional recovery. Edaravone scavenges free radicals, reduces oxidative damage, and protects penumbral neurons. Dexborneol exerts anti-inflammatory effects, inhibits inflammatory damage to the neurovascular unit, and reduces secondary injury. After penumbral neurons are protected, electrophysiological and synaptic functions gradually recover, and scores decrease accordingly ^[5]. Neurological recovery is a dynamic process. The advantage of sequential therapy over conventional therapy lies mainly in the longer administration time, covering the critical period of neural repair. Recovery is most active within weeks after cerebral infarction; without protection, recovery may be hindered. Continuous use of sublingual tablets provides support for post-discharge repair, which may explain why the difference in scores between the two groups widened over time.

The results showed that both inflammatory indicators were significantly lower in the observation group after treatment. hs-CRP and IL-6 not only reflect the degree of inflammation but are also associated with neurological deterioration. The reduction of inflammatory factors in the observation group indicates that the anti-inflammatory effect of dexborneol is clinically effective. Post-ischemic inflammation is dual in nature: moderate inflammation can clear necrotic tissue, while excessive inflammation aggravates injury. Patients with progressive cerebral infarction often experience uncontrolled inflammation, with massive release of proinflammatory factors, forming a positive feedback loop, recruiting peripheral immune cells, and aggravating blood-brain barrier disruption and neuronal death. Dexborneol inhibits the expression of TNF- α , IL-1 β , COX-2, etc., weakening the initiation of inflammation at the source, thereby reducing hs-CRP and IL-6. This effect improves indicators, protects the blood-brain barrier, and reduces cerebral edema ^[6]. After inflammation is controlled, vascular endothelial injury is reduced, microcirculation is maintained, and penumbral blood supply is improved, laying the foundation for neurological recovery. Better inflammation control in the observation group also explains its efficacy.

In this study, Hcy and NSE were lower in the observation group after treatment. Hcy is an intermediate product of methionine metabolism. Its elevation can damage vascular endothelium and promote atherosclerosis and thrombosis. Hyperhomocysteinemia can damage endothelium through oxidative stress, endoplasmic reticulum stress, and inflammation, promoting thrombus extension and worsening perfusion.

The reduction of Hcy by sequential therapy with edaravone dexborneol may be related to its antioxidant and anti-inflammatory effects. After oxidative and inflammatory responses are inhibited, endothelial function improves, Hcy toxicity is reduced, and the liver's ability to metabolize Hcy may also recover. NSE marks neuronal injury, and its level is positively correlated with infarct volume and degree of deficit. The reduction of NSE in the observation group indicates that this treatment reduced irreversible neuronal injury. Edaravone scavenges free radicals, protects neuronal membranes, and reduces NSE release. Dexborneol inhibits glutamate excitotoxicity and reduces neuronal necrosis. Simultaneous improvement in Hcy and NSE indicates that this treatment protects both blood vessels and neurons.

The incidence of adverse reactions in the observation group was lower, suggesting that sequential therapy with edaravone dexborneol has good safety. In this study, adverse reactions in the observation group were mainly dizziness, fatigue, and somnolence; symptoms were mild, tolerable, and no serious adverse events occurred. Edaravone has clear targets and little interference with normal physiology; dexborneol is derived from natural components and has high safety. Sublingual tablets avoid local irritation and infection risk associated with injections, improving safety. Fewer adverse reactions can improve compliance, reduce discontinuation due to adverse reactions, and ensure completion of sequential therapy. Good safety is a key prerequisite for the widespread use of drugs in patients with progressive cerebral infarction, and this treatment has clear advantages.

5. Conclusion

In summary, sequential therapy with edaravone dexborneol has a clear effect in patients with progressive cerebral infarction. It can improve neurological function, reduce inflammation and neurological injury, and lower Hcy and NSE. The therapy has few adverse reactions and good safety. Sublingual tablets allow patients to continue sequential therapy after discharge, compensating for the previous shortcoming that sequential therapy was difficult with injection alone. After sublingual tablets are launched and promoted, more patients may benefit. However, this study had a small sample size and short follow-up. Future studies should expand the sample and prolong follow-up to verify long-term efficacy and safety.

Disclosure statement

The authors declare no conflict of interest.

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