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Clinical Effect of Nimodipine Combined with Rosuvastatin Calcium in the Treatment of Hypertensive Intracerebral Hemorrhage

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Abstract: *Objective*: To explore the clinical effect of nimodipine combined with rosuvastatin calcium in the treatment of hypertensive intracerebral hemorrhage. *Methods*: Sixty patients with hypertensive intracerebral hemorrhage from January 2023 to December 2023 were randomly divided into a control group (n = 30) and an observation group (n = 30). The control group received conventional treatment, while the observation group was treated with nimodipine + rosuvastatin calcium tablets. The neurological function, edema volume, hematoma volume, adverse reactions, and treatment efficiency were compared between the two groups. *Results*: After treatment, compared with the control group, the edema volume, hematoma volume index, and NIHSS score in the observation group were all reduced, and the differences were extremely significant ($P < 0.001$). The clinical efficacy of the observation group was 93.33%, which was much higher than the 66.67% of the control group, and the difference was significant ($P < 0.05$). The incidence of adverse reactions in the observation group was 3.33%, which was far lower than the 30.00% of the control group, and the difference was significant ($P < 0.05$). *Conclusion*: Nimodipine combined with rosuvastatin calcium in the treatment of hypertensive intracerebral hemorrhage shows significant advantages in improving edema volume and hematoma volume, promoting neurological recovery, improving clinical efficacy, and reducing the incidence of adverse reactions, thus having broad clinical application prospects.

Keywords: Nimodipine; Rosuvastatin calcium; Hypertensive intracerebral hemorrhage; Clinical effect

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1. Introduction

Hypertensive intracerebral hemorrhage is a common and serious cerebrovascular disease characterized by high morbidity, high disability rate, and high mortality rate [1]. Its pathogenesis is mainly due to pathological changes in small cerebral arteries caused by long-term hypertension, leading to cerebral vascular rupture and bleeding

when blood pressure rises suddenly. After a cerebral hemorrhage, local brain tissue will undergo pathological changes such as hematoma compression and secondary cerebral edema, which can lead to neurological deficits [2]. Currently, there are various clinical treatments for hypertensive intracerebral hemorrhage, but the efficacy still needs to be further improved. Nimodipine is a neuroprotective calcium channel blocker, and rosuvastatin calcium is a statin lipid-lowering drug. Recent studies have found that it has pleiotropic effects, including anti-inflammatory, antioxidant, and improvement of endothelial function. This study aims to explore the clinical effect of nimodipine combined with rosuvastatin calcium in the treatment of hypertensive intracerebral hemorrhage, providing a reference for clinical treatment.

2. Materials and methods

2.1. General information

Sixty patients with hypertensive intracerebral hemorrhage admitted between January 2023 and December 2023 were selected as research subjects. The patients were divided into a control group and an observation group according to the random number table method, with 30 patients in each group.

Control group: 18 males and 12 females; aged between 45–78 years, with an average age of (62.51 ± 8.34) years; bleeding site: 15 cases in the basal ganglia region, 10 cases in the lobes, and 5 cases in the thalamus; bleeding volume: 5–15ml, with an average of (10.55 ± 2.21) ml.

Observation group: 16 males and 14 females; aged between 42–75 years, with an average age of (60.82 ± 7.93) years; bleeding site: 13 cases in the basal ganglia region, 12 cases in the lobes, and 5 cases in the thalamus; bleeding volume: 5–15ml, with an average of (11.32 ± 1.52) ml. There was no statistically significant difference in general information such as gender, age, bleeding site, and bleeding volume between the two groups ($P > 0.05$), indicating comparability.

Inclusion criteria: (1) Meet the diagnostic criteria for hypertensive intracerebral hemorrhage; (2) The onset time is within 24–72 hours; (3) The patient or family members have signed an informed consent form.

Exclusion criteria: (1) Those with severe cardiac, liver, or kidney dysfunction; (2) Those with a bleeding tendency or coagulation dysfunction; (3) Those who are allergic to nimodipine or rosuvastatin calcium; (4) Those with a history of cerebral vascular malformations, brain tumors, or other brain diseases.

2.2. Methods

2.2.1. Control group

Conventional treatment methods were adopted: (1) Patients need to rest in bed, maintain a quiet environment, and avoid emotional excitement, as these external stimuli may cause blood pressure fluctuations and aggravate the condition of cerebral hemorrhage. (2) Based on the patient's intracranial pressure, mannitol, a dehydrating agent that reduces intracranial pressure, was administered. Mannitol can effectively reduce brain tissue edema and lower intracranial pressure. However, the specific dosage must be precisely adjusted based on the patient's actual intracranial pressure to ensure both therapeutic effectiveness and the avoidance of adverse reactions. (3) Strictly control the patient's blood pressure, maintaining it within the range of 140–160/90–100 mmHg. Stable blood pressure is crucial to prevent further deterioration of cerebral hemorrhage. (4) Maintain the patient's water and electrolyte balance and provide nutritional support to ensure the normal functioning of the patient's bodily functions. (5) Actively prevent and treat complications such as pulmonary infections and stress ulcers.

2.2.2. Observation group

In addition to conventional treatment, a treatment regimen combining nimodipine and rosuvastatin calcium was added. The initial dose of nimodipine (manufacturer: Tianjin Central Pharmaceutical Co., Ltd.; specification: 20 mg x 50 tablets) was 30 mg per time, taken orally three times a day. The dosage could be gradually increased to 60 mg per time, also taken orally three times a day, based on the patient's condition and tolerance. Rosuvastatin calcium (manufacturer: Wuhan Mingsheng Technology Co., Ltd.; specification: 10 mg x 28 tablets) was administered at a dose of 10 mg per time, taken orally once a day. Both groups of patients were treated continuously for 4 weeks.

2.3. Observation indices

2.3.1. Imaging indices

Edema volume: The volume of cerebral edema in patients was measured using head CT scans before treatment and after 4 weeks of treatment. The edema volume was calculated using a specific formula through professional image analysis software.

Hematoma volume: Similarly, before treatment and after 4 weeks of treatment, the hematoma volume was calculated using the Tada formula (hematoma volume = $\pi/6$ x length axis x short axis x slice number) through head CT scans.

2.3.2. Neurological function

The National Institutes of Health Stroke Scale (NIHSS) score was used to evaluate patients' neurological function, with assessments conducted before treatment and after 4 weeks of treatment. The maximum score is set to 42, and a higher score indicates a more severe degree of neurological impairment.

2.3.3. Clinical efficacy

Based on the clinical symptoms, signs, and imaging examination results of patients after treatment, the treatment effect was divided into three levels: significantly effective, effective, and ineffective.

Significantly effective: The clinical symptoms and signs of patients improved significantly, NIHSS score decreased by ≥70%, and edema and hematoma volumes decreased by ≥50%.

Effective: The clinical symptoms and signs of patients improved somewhat, with a 30% ≤ NIHSS score decrease < 70%, and a 20% ≤ decrease in edema and hematoma volumes < 50%.

Ineffective: The clinical symptoms and signs of patients showed no significant improvement or even worsened, NIHSS score decreased by <30%, and edema and hematoma volumes decreased by <20%.

Treatment effectiveness rate = (number of significantly effective cases + number of effective cases) / total number of cases x 100%.

2.3.4. Adverse reactions

Adverse reactions that occurred during treatment in both groups of patients were observed and recorded, including nausea, gastrointestinal bleeding, transient dizziness, etc.

2.4. Statistical methods

Data analysis was performed using SPSS 27.0. Measurement data were expressed as (± SD) and analyzed using the *t*-test. Count data were expressed as [n(%)] and analyzed using the x^2 test. A *P*-value < 0.05 was considered statistically significant.

3. Results

3.1. Comparison of imaging indices between the two groups

Before treatment, there was no significant difference in edema volume and hematoma volume between the two groups. After treatment, compared with the control group, the observation group showed significantly reduced edema volume (1.30 ± 0.15 mL vs. 2.34 ± 0.33 mL) and hematoma volume (9.84 ± 1.16 mL vs. 15.29 ± 1.21 mL), with extremely significant differences ($P < 0.001$), as shown in **Table 1**.

Table 1. Comparison of edema volume and hematoma volume indices between the two groups (± SD, mL)

Group	Number of cases (n)	Edema volume		Hematoma volume	
		Before treatment	After treatment	Before treatment	After treatment
Control group	30	6.41 ± 1.26	2.34 ± 0.33	20.15 ± 3.50	15.29 ± 1.21
Observation group	30	6.38 ± 1.20	1.30 ± 0.15	20.24 ± 3.36	9.84 ± 1.16
t value		0.094	15.714	0.102	17.809
P value		0.925	<0.001	0.919	<0.001

3.2. Comparison of neurological function between the two groups of patients

Before treatment, the NIHSS scores of the two groups were similar, and there was no statistically significant difference. After treatment, the NIHSS score of the observation group was lower than that of the control group, and the difference was extremely significant ($P < 0.001$), as shown in **Table 2**.

Table 2. Comparison of NIHSS scores between the two groups of patients (± SD, score)

Group	Number of cases (*n*)	NIHSS score	
		Before treatment	After treatment
Control group	30	23.11 ± 3.20	15.27 ± 1.35
Observation group	30	23.20 ± 3.17	6.47 ± 1.24
t-value		0.109	27.375
P-value		0.913	<0.001

3.3. Comparison of clinical efficacy between the two groups of patients

The clinical efficacy of the observation group was 93.33%, which was much higher than the 66.67% of the control group, and the difference was significant ($P = 0.010 < 0.05$), as shown in **Table 3**.

Table 3. Comparison of clinical efficacy between the two groups of patients [n(%)]

Group	n	Marked effect	Effective	Ineffective	Total effective rate
Control group	30	10 (33.33%)	10 (33.33%)	10 (33.33%)	20 (66.67%)
Observation group	30	20 (66.67%)	8 (26.67%)	2 (6.67%)	28 (93.33%)
x^2 value	-	-	-	-	6.667
P value	-	-	-	-	0.010

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

The incidence of adverse reactions in the observation group was 3.33%, which was significantly lower than the 30.00% in the control group (P = 0.006 < 0.05), as shown in **Table 4**.

Table 4. Comparison of the incidence of adverse reactions between the two groups [n(%)]

Group	n	Gastrointestinal bleeding	One-time dizziness	Nausea	Total adverse reaction rate
Control group	30	3 (10.00%)	4 (13.33%)	2 (6.67%)	9 (30.00%)
Observation group	30	0 (0.00%)	1 (3.33%)	0 (0.00%)	1 (3.33%)
x^2 value					7.680
P value					0.006

4. Conclusion

In the field of neurosurgery, hypertensive intracerebral hemorrhage is a quite common disease with a persistently high incidence rate. As the incidence of hypertension continues to rise in the population, the occurrence of hypertensive intracerebral hemorrhage has shown a more pronounced growth trend in recent years. According to relevant research data and clinical observations, this increasing trend is closely related to the increase in the number of hypertensive patients [3]. Nimodipine is a commonly used antihypertensive drug in clinical treatment, and as a typical representative of calcium channel blockers, it has a unique pharmacological mechanism. Besides its basic effect of lowering blood pressure, nimodipine can also exert specific effects on nerve cells and cerebrovascular vessels. By regulating calcium ion channels, nimodipine can affect the excitability of nerve cells and the contractile state of cerebrovascular vessels, thereby playing a positive role in improving cerebral blood circulation, reducing cerebral ischemic injury, and protecting nerve cells [4–5]. Rosuvastatin calcium, on the other hand, is a commonly used lipid-lowering drug in clinical practice. Its main pharmacological effect lies in its ability to effectively reduce the level of lipoprotein cholesterol in the plasma and inhibit the synthesis process of cholesterol. In long-term clinical application, the lipid-lowering effect of rosuvastatin calcium has been widely confirmed and recognized. Many patients have achieved significant improvements in their blood lipid indicators after using this drug, thereby reducing the risk of cardiovascular disease and other related complications [6–7]. Both drugs occupy important positions in their respective pharmacological fields, laying a foundation for subsequent research on combination therapy for hypertensive intracerebral hemorrhage.

This study suggests that the observation group exhibited significant advantages over the control group in multiple aspects in the treatment of hypertensive intracerebral hemorrhage. After treatment, the observation group showed significant improvement in key indicators such as edema volume, hematoma volume, and NIHSS score. This is primarily attributed to the synergistic mechanism of the combined drug regimen. As a calcium channel blocker, nimodipine can effectively dilate cerebral blood vessels, improve cerebral blood circulation, and reduce a series of pathophysiological changes caused by local ischemia and hypoxia. This inhibits the further development of edema around the hematoma and promotes hematoma absorption [8]. At the same time, it has a protective effect on nerve cells, can reduce nerve damage, and contribute to the recovery of nerve function. Rosuvastatin calcium plays an important role due to its pleiotropic effects. It can regulate lipid metabolism, stabilize vascular endothelial function, and reduce vascular wall damage and inflammation. Through anti-inflammatory and antioxidant effects, it can reduce the inflammatory cascade reaction after cerebral hemorrhage, alleviate secondary damage to brain

tissue, further promote edema subsidence and hematoma absorption, and create favorable conditions for the repair of nerve function [9]. In terms of clinical efficacy, the significant improvement in the observation group benefits from the cooperation of the two drugs, which intervene in the complex pathological process after hypertensive intracerebral hemorrhage from different pathological links, thereby effectively improving patients' symptoms and signs and enhancing the effectiveness of treatment. Regarding the incidence of adverse reactions, the advantage of the observation group may be due to the reasonable dosing combination of the two drugs during combination therapy, and their respective mechanisms of action are adjusted to a certain extent, reducing the possible adverse reactions caused by a single drug and making the overall treatment process safer and more tolerable [10–11].

In summary, the combination of nimodipine and rosuvastatin calcium has a significant clinical effect in the treatment of hypertensive intracerebral hemorrhage. It stands out in improving patients' conditions, enhancing treatment efficiency, and reducing the risk of adverse reactions. This provides a more effective treatment strategy for the clinical treatment of hypertensive intracerebral hemorrhage and is worthy of further promotion and application in clinical practice, as well as deeper research on its mechanism of action, to better serve patients.

Disclosure statement

The authors declare no conflict of interest.

Reference

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