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Research Progress in Emergency Treatment for Patients with Cerebral Hemorrhage

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Abstract: As a severe neurological disease, cerebral hemorrhage is dangerous and progresses rapidly, with high disability and fatality rates. Its occurrence seriously harms patients' lives and health, and also causes a heavy social burden. Timely diagnosis and treatment of cerebral hemorrhage are essential for improving patients' prognosis. This article reviews the research progress in emergency treatment for patients with cerebral hemorrhage, providing a basis for diagnosis and treatment.

Keywords: Cerebral hemorrhage; Assessment; Diagnosis; Examination; Treatment

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1. Introduction

Cerebral hemorrhage, or primary non-traumatic parenchymal hemorrhage, accounts for 20–30% of acute strokes. It is characterized by high incidence, disability, and mortality rates. Epidemiological data show that there are 350,000 to 550,000 new cases annually in China, and the incidence rate increases significantly with age, reaching (120–180)/100,000 among people aged 45–75 [1]. The core etiology is hypertension combined with cerebral arteriolar sclerosis, accounting for about 60–70% of cases, followed by cerebral aneurysms (10–15%), arteriovenous malformations (5–10%), and coagulation dysfunction [2]. The influencing factors are diverse, such as age, gender, uncontrolled hypertension, long-term alcohol abuse, smoking, etc. About 40–60% of survivors of cerebral hemorrhage suffer from neurological impairments such as hemiplegia and aphasia, and the long-term care dependence rate for severe patients exceeds 70%, posing continuous pressure on families and the social medical system [3]. Early identification of abnormal blood pressure and control of risk factors are key strategies to reduce the disease burden. Timely diagnosis and treatment are crucial for reducing brain damage and improving the prognosis of patients with cerebral hemorrhage.

2. Early assessment of cerebral hemorrhage

2.1. Early imaging examination and laboratory testing

2.1.1. Imaging examination

Cranial CT and MRI can distinguish between cerebral hemorrhage, cerebral infarction, and primary or secondary space-occupying lesions. Compared to conventional MRI, CT examination has higher sensitivity in diagnosing edema. Therefore, in the early assessment of patients with cerebral hemorrhage, CT examination can be preferred to determine the patient's clinical manifestations, bleeding site and volume, and the presence of intraventricular hemorrhage and subarachnoid hemorrhage. To clarify the intracranial hemorrhage, especially for young patients with no history of hypertension and uncommon sites of cerebral hemorrhage, head MRI, head magnetic resonance angiography (MRA), or digital subtraction angiography (DSA) can be performed. The first classification based on the etiology of cerebral hemorrhage was released in 2012, indicating that the etiologies include drug-induced, hypertension, amyloid vascular disease, systemic diseases, structural etiologies, and unknown etiologies. This classification can provide a basis for determining the etiology of clinical cerebral hemorrhage and assessing patient prognosis.

2.1.2. Laboratory testing

There are many laboratory tests, including complete blood count, serum glucose, serum electrolytes, liver function, CRP, coagulation function, international normalized ratio, etc. Toxicology testing can be performed when drug abuse is suspected. Besides, patients' vital signs should be monitored using electrocardiographic monitoring.

2.2. Scales related to cerebral hemorrhage

2.2.1. Glasgow Coma Scale (GCS)

Since its introduction in 1974, the Glasgow Coma Scale (GCS) has become the most widely used quantitative assessment tool for coma severity in the world. This scale establishes a 15-point evaluation system based on three dimensions: eye-opening response (1–4 points), verbal response (1–5 points), and motor response (1–6 points). It boasts three core features: standardized operation, cross-cultural universality, and dynamic monitoring value. The GCS establishes objective quantitative criteria, providing an accurate basis for clinical decision-making through grading. The feasibility and professionalism of the scale design are strong. Currently, GCS has been included in 85% of stroke diagnosis and treatment guidelines in China, becoming a standard tool for assessing neurological function in patients with acute brain injury. The GCS score is widely used in the assessment of acute cerebral hemorrhage status, providing a basis for medical staff to assess the patient's condition early and helping to formulate timely response measures. However, the GCS score is not applicable to some patients, including children under 3 years old, patients with mental illness, communication disorders, or hearing loss. Applying this score to such patients may affect the accuracy of the assessment results. Additionally, the scoring can be influenced by the scorer's subjective consciousness, and the use of related drugs may also affect the assessment results.

2.2.2. Intracerebral Hemorrhage (ICH) clinical grading score

The ICH score is a grading system specifically designed for spontaneous intracerebral hemorrhage. It is simple to operate and highly practical, capable of predicting the risk of death within one month for patients with cerebral hemorrhage. In the early stages of cerebral hemorrhage, assessing the patient's clinical condition using the ICH score can provide a reference for clinicians to formulate treatment plans. However, the ICH score has certain

limitations, as some of its indicators have limited applicability among different global populations.

2.2.3. National Institutes of Health Stroke Scale (NIHSS) score

As a commonly used method to evaluate neurological impairment in stroke patients, the NIHSS score is calculated by summarizing the scores of 15 parameters. The patient's condition is inversely correlated with this score. The NIHSS score can not only assess the severity of stroke patients but also evaluate their treatment effectiveness. Research has shown that among the risk factors for poor prognosis in patients with cerebral hemorrhage, an NIHSS score of 20 or higher is one of the risk factors, suggesting that NIHSS can be used to assess the prognosis of patients with cerebral hemorrhage [4].

3. Emergency treatment of cerebral hemorrhage

3.1. Blood pressure management

Hypertension is not only one of the main causes of cerebral hemorrhage, but can also be induced by cerebral hemorrhage. Hypertension can affect the edema around the hematoma in patients with cerebral hemorrhage and may cause recent rebleeding. Among the factors contributing to a poor prognosis in patients with cerebral hemorrhage, hypertension is a significant one. Therefore, managing the patient's blood pressure is crucial. There is a positive correlation between elevated blood pressure and hematoma expansion in patients with cerebral hemorrhage. Effective blood pressure-lowering treatment during the acute phase can prevent hematoma expansion and improve the patient's prognosis. Guidelines recommend that hypertensive patients should use intravenous medication to control their systolic and diastolic blood pressure below 160 mmHg and 90 mmHg, respectively. Early intensive blood pressure lowering in patients has higher efficacy and safety compared to target blood pressure lowering. Studies by Liu Chang *et al.* [5] have shown that intensive blood pressure lowering treatment for hypertensive cerebral hemorrhage patients can reduce the risk of hematoma expansion, which is significantly positive for improving the patient's prognosis. Wang Yu *et al.*'s [6] research results indicate that using urapidil for intensive blood pressure lowering in cerebral hemorrhage patients can effectively control blood pressure and delay hematoma expansion.

3.2. Intervention for antiplatelet drug-related cerebral hemorrhage

In the early stages of spontaneous cerebral hemorrhage, neurological damage caused by the expansion of the hematoma or the intensification of edema around the hematoma is significant, which can have a notable impact on the patient's prognosis. Especially in acute cerebral hemorrhage, where the hematoma expands rapidly and often appears early, it is crucial to adopt scientific and standardized methods to limit its expansion to prevent further damage to the patient. Antiplatelet drugs, commonly used for various diseases, including stroke, are increasingly widespread. Although these drugs play a vital role in disease treatment, they also have side effects. Overuse can inhibit platelet function, potentially increasing the risk of bleeding events. Multiple studies have been conducted in China on whether to use platelet transfusion in the acute phase of cerebral hemorrhage, but this issue remains controversial. Commonly used antiplatelet drugs such as aspirin and clopidogrel have irreversible inhibitory effects on platelets, leading many scholars to shift their treatment goals to reversing platelet function. A phase III clinical trial involving 190 patients who developed symptoms within 6 hours was divided into a platelet transfusion group and a standard treatment group. The results showed that the fatality rate in the platelet transfusion group was

higher than that in the control group [7]. Thus, platelet transfusion should be performed cautiously in the treatment of cerebral hemorrhage.

3.3. Intervention for anticoagulant drug-related cerebral hemorrhage

With the global increase in patients with thrombotic diseases such as atrial fibrillation and venous thrombosis, the incidence of anticoagulant drug-related cerebral hemorrhage (including vitamin K antagonists and novel oral anticoagulants) has shown a significant upward trend. The annual risk of bleeding for those receiving warfarin therapy ranges from 0.3% to 2.4%. Although the incidence of cerebral hemorrhage associated with novel oral anticoagulants is lower than that of warfarin, it is higher in certain patient groups, such as elderly patients and those with chronic kidney disease. Anticoagulant drug-related cerebral hemorrhage has unique clinical characteristics, including a hematoma expansion rate higher than 35% and a 24-hour mortality rate exceeding 25%. Due to abnormal coagulation function, the effectiveness of conventional hemostatic treatment is limited, and survivors are more likely to experience moderate to severe neurological dysfunction compared to non-anticoagulant-related cases. For warfarin-related cerebral hemorrhage, if the patient's international normalized ratio (INR) is below 1.3 within 4 hours of onset and their systolic blood pressure is controlled below 160 mmHg, the risk of intracranial hematoma expansion can be reduced. The reversal of warfarin's anticoagulant effect mainly focuses on supplementing coagulation factors, with options including fresh frozen plasma, prothrombin complex concentrates, vitamin K, and recombinant activated coagulation factor VII. Studies have shown that tranexamic acid is effective in treating cerebral hemorrhage caused by non-vitamin K antagonist oral anticoagulants [8]. For anticoagulant drug-related cerebral hemorrhage, anticoagulant reversal agents should be used as early as possible, provided the patient's condition allows.

3.4. Use of hemostatic agents

Studies have explored whether recombinant human coagulation factor VIIa can be used for the treatment of cerebral hemorrhage. The results showed that the emergency use of hemostatic drugs in patients did not prevent hematoma expansion and did not improve patient functional outcomes [9]. Some scholars have also pointed out that the application of tranexamic acid in patients with cerebral hemorrhage can prevent hematoma enlargement and improve hematoma area. It contains thrombin-like substances that can promote platelet aggregation, quickly stop bleeding at the site of cerebral hemorrhage, and prevent rebleeding [10]. However, there are few studies on the use and dosage of hemostatic drugs in patients with cerebral hemorrhage, which requires further exploration.

3.5. Intervention for common complications

Complications of cerebral hemorrhage can aggravate the patient's condition and increase the risk of mortality, so timely intervention is needed for the complications that occur. For patients with fever, prompt cooling measures should be taken to restore normal body temperature. The patient's swallowing function should be evaluated to prevent aspiration and aspiration pneumonia, paying attention to the patient's posture. If the patient is at risk of gastrointestinal bleeding, gastric mucosal protective agents can be administered, strengthening patient observation and watching for active bleeding. For severe patients requiring mechanical ventilation, real-time monitoring of the patient's condition should be implemented to avoid acute respiratory distress syndrome.

4. Conclusion

Cerebral hemorrhage is a rapidly changing and dangerous condition. Early diagnosis and timely treatment are crucial for improving the patient's condition and prognosis. Currently, emergency treatment for cerebral hemorrhage patients mainly involves targeted therapeutic measures and symptomatic treatment based on patient examination and condition. However, there is a lack of standardized treatment processes and standard treatment plans. To ensure treatment effectiveness and improve patient prognosis, it is necessary to explore effective treatment methods and establish standardized treatment processes and plans.

Disclosure statement

The authors declare no conflict of interest.

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