

Research progress on risk factors for rebleeding in spontaneous subarachnoid hemorrhage

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Abstract Spontaneous subarachnoid hemorrhage (SAH) is a common clinical cerebrovascular disease. Based on etiology, it can be classified into two major categories: aneurysmal subarachnoid hemorrhage (aSAH) and non-aneurysmal subarachnoid hemorrhage (nSAH). Among these, aSAH accounts for more than 80% and is the primary type of clinical concern. Rebleeding refers to the recurrence of intracranial hemorrhage within a period after the initial onset in SAH patients. It is a critical complication leading to worsening conditions, death, and long-term neurological dysfunction. Given its high incidence, identifying and intervening in its risk factors is the core of clinical prevention and control. This article summarizes the risk factors for rebleeding in spontaneous subarachnoid hemorrhage as follows.

Keywords: Spontaneous subarachnoid hemorrhage; Rebleeding; Aneurysm; Risk factors

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Introduction

Spontaneous subarachnoid hemorrhage (SAH) is a common clinical emergency and critical cerebrovascular disease characterized by acute onset and rapid progression. Currently, the incidence of SAH in China is 2/100,000. Approximately 85% of cases are caused by the rupture of intracranial aneurysms, known as aneurysmal subarachnoid hemorrhage (aSAH), while the remaining 15% are termed non-aneurysmal subarachnoid hemorrhage (nSAH). SAH has high disability and mortality rates, posing a serious threat to patient life and leading to long-term complications such as neurological deficits, which significantly reduce quality of life and social adaptability [1-3]. Rebleeding is the recurrence of intracranial hemorrhage shortly after the initial event; it occurs frequently and often leads to a sharp deterioration in the patient's condition, severely affecting

prognosis. Therefore, accurately predicting the risk of SAH rebleeding is crucial for clinical treatment and patient recovery.

1 Epidemiology

In a 9-year stroke follow-up study conducted in China, Chen et al. [4,5] found that the 28-day mortality rate for SAH patients was 19%, with aSAH accounting for approximately 85% of all SAH cases. The annual incidence of aSAH varies considerably across different regions of the world. High-incidence areas include Finland and Japan, with 19.7 and 22.7 per 100,000 people, respectively; low-incidence areas include Central and South America at 4.2 per 100,000, while the incidence in other regions is approximately 9.1 per 100,000 [6]. The mortality rate of aSAH remains high; a study on global aSAH mortality

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from 1995 to 2007 showed that the mortality rate in Asia ranged from 26.7% to 35.8% [7]. Therefore, improving the prognosis of aSAH is essential. Relevant literature reports that preoperative rebleeding is a major factor leading to poor outcomes in aSAH [8-11]. A meta-analysis involving 14 studies and 5,693 patients showed a rebleeding incidence of 7-26%, with a mortality rate for rebleeding patients between 20% and 60% [12,13].

2 Patient Condition

2.1 Clinical Manifestations

Severe headache is a typical symptom of SAH. Studies indicate that patients with more severe headaches at onset may face a higher risk of rebleeding [14]. Severe pain may reflect a sharp increase in intracranial pressure (ICP), indicating severe bleeding and poor stability at the rupture site, and thus increasing the risk of rebleeding [15]. Consciousness disorders such as coma or somnolence suggest a severe condition and an increased risk of rebleeding [16]. These disorders may be related to large hemorrhage volumes, increased ICP, and impaired brain function, all of which increase the likelihood of rebleeding.

2.2 Medical History

Hypertension is considered a risk factor for aneurysm formation and rebleeding, and a systolic blood pressure \geq 160mmHg is a significant risk factor for rebleeding in aneurysmal subarachnoid hemorrhage [17]. Patients with a significant increase in blood pressure at onset are more likely to experience rebleeding, as hypertension can increase the pressure on the vascular wall, making the ruptured blood vessel more prone to re-rupture [18].

Strict blood pressure control can reduce vascular wall pressure and prevent re-rupture of the damaged vessel.

However, excessively low blood pressure can also lead to cerebral hypoperfusion and aggravate brain injury. Therefore, when managing blood pressure, it is necessary to select appropriate antihypertensive drugs and target blood pressure based on the patient's individual condition. Currently, there is no clear target blood pressure value proven to reduce rebleeding risk, but maintaining systolic blood pressure below 160mmHg is a relatively reasonable approach. In clinical practice, blood pressure regulation must balance the risk of stroke, hypertension-related rebleeding, and the maintenance of cerebral perfusion pressure, striving to achieve equilibrium between these factors [19].

Hyperglycemia may be associated with poor prognosis in SAH patients, but its relationship with rebleeding remains unclear. Hyperglycemia can damage vascular endothelial cells, promote the development of atherosclerosis, and potentially increase the risk of rebleeding [20].

Some studies suggest that a history of heart disease may increase the risk of rebleeding in subarachnoid hemorrhage [21]. On one hand, heart disease itself can cause pathological manifestations such as blood pressure fluctuations and arrhythmias, which adversely affect the stability of cerebral blood vessels. On the other hand, patients with heart disease often require long-term use of anticoagulant drugs, which interfere with the body's normal coagulation function. In addition, physical frailty in patients can significantly reduce recovery efficiency after bleeding, thereby indirectly increasing the potential risk of rebleeding [21].

Long-term alcohol consumption leads to decreased vascular elasticity and blood pressure fluctuations [20]. Alcohol may also affect coagulation function, making hemostasis and tissue repair after bleeding more difficult.

Meanwhile, alcohol-induced emotional excitement can further increase the burden on cerebral blood vessels, raising the risk of rebleeding.

Smoking can damage vascular endothelial cells, promote atherosclerosis, and increase the risk of SAH rebleeding. Smoking also causes vasoconstriction and elevated blood pressure, further exacerbating vascular burden [22].

2.3 mFisher Grade and Intracranial Hematoma

Whether intracranial hematoma (including intracerebral and intraventricular hematoma) is an independent risk factor for aSAH rebleeding remains controversial. Some studies suggest that intracerebral hematoma is associated with the risk of rebleeding. Zhang Gaoqi et al.'s analysis of intracerebral hematoma identified it as an independent risk factor for aSAH rebleeding, suggesting that agitation and hypertension caused by the stimulation of brain tissue by intracranial hematoma may be part of the incentives for rebleeding. However, other studies argue that when factors such as clinical grading are comprehensively considered, the independent predictive effect of intracerebral hematoma may be weakened [23]. The mFisher grade is a classification based on the amount and location of subarachnoid hemorrhage on CT scans, and is more widely used than intracranial hematoma [23]. The mFisher grade is usually used to predict cerebral vasospasm after aneurysmal subarachnoid hemorrhage, and is an indicator reflecting the amount of subarachnoid hemorrhage. The greater the amount of bleeding, the higher the mFisher grade, and the more severe the patient's condition [24]. Some studies suggest that the modified Fisher grade is not related to rebleeding [10], but more studies have shown that a higher Fisher grade may be a strong risk factor for rebleeding, because a large amount of bleeding is often

regarded as a sign of impaired stability of the aneurysm wall in aSAH patients [12,25]. Relevant studies have shown that within 72 hours after intracranial aneurysm rupture, the rebleeding rate of patients with mFisher grade 0-2 is significantly lower than that of patients with grade 4-5, and this grade is significantly positively correlated with patient prognosis. Further confirmation through univariate and multivariate stratified analysis indicates that mFisher grade is an independent risk factor for rebleeding after aneurysm rupture [26]. In addition, several other clinical studies have reached the same conclusion that a high mFisher grade is an independent influencing factor leading to an increased risk of rebleeding in such patients [22,24,27].

2.4 Hunt-Hess Grade

Studies [10,28-30] have conducted multivariate logistic analysis on the relationship between rebleeding and Hunt-Hess grade. These studies divided Hunt-Hess grade into good group (grades I-II) and poor group (grades III-V), and listed the number of rebleeding patients in different Hunt-Hess grades. The results showed that the increase of Hunt-Hess grade was associated with an increase in the risk of rebleeding in aSAH patients. Meanwhile, a meta-analysis [12] involving 5,693 patients from many different countries, including the United States and European countries, identified a poor Hunt-Hess grade as a risk factor for preoperative rebleeding.

3 Aneurysm Morphological Characteristics

3.1 Size and Shape

- **Size:** Larger aneurysms are more likely to rebleed [31]. Studies by Andrew M, Rahman M et al. [32,33] point out that the size of the aneurysm

is an independent risk factor for spontaneous re-rupture and bleeding. Andrew M further proposes that the larger the volume of the aneurysm, the higher the probability of spontaneous re-rupture and bleeding, and the study also confirms that the size of the aneurysm has no correlation with the time of rebleeding. Studies by Donkelaar, Bakker et al. also indicate a certain association between aneurysm size and rebleeding, with patients with an aneurysm diameter > 20mm having a significantly higher risk of rebleeding [34]. All the above studies show that aneurysm size is a risk factor for rebleeding. However, other studies suggest that aneurysm size is not related to aneurysm re-rupture and bleeding [35].

- **Shape:** Irregularly shaped aneurysms are considered risk factors for rupture. Japanese scholars have reported that among unruptured aneurysms, irregular aneurysms with daughter sacs have a higher rupture risk than regular ones. A prospective study on Japanese patients with unruptured intracranial aneurysms (UIAs) published in the *New England Journal of Medicine* in 2012 also identified the presence of daughter sacs as an independent predictor of aneurysm rupture [31]. These aneurysms have unstable structures, and the vascular wall at the rupture site is easily impacted by blood flow, leading to re-rupture and bleeding.

3.2 Location

Aneurysms located in special sites such as the posterior circulation or the bifurcation of the middle cerebral artery

have a higher risk of rebleeding. Aneurysms in these locations are more difficult to operate on, with relatively poor treatment effects, and are prone to rebleeding [36]. However, most studies suggest that the location of the aneurysm is not related to the occurrence of rebleeding after aneurysmal subarachnoid hemorrhage [37].

3.3 Number of Aneurysms

Jurgen Beck et al. found that multiple aneurysms are a risk factor for aneurysm re-rupture and bleeding. They believe that the vascular fragility of multiple aneurysms is relatively high, which is the reason for the easy formation of multiple aneurysms and also the easy cause of aneurysm re-rupture and bleeding. However, most studies believe that the number of aneurysms is not related to rebleeding [37].

4 Cerebral Vasospasm and Acute Hydrocephalus

Cerebral Vasospasm (CVS): After aneurysm rupture and bleeding, decomposition products of red blood cells (such as hemoglobin) can stimulate the production of vasoconstrictive substances (such as endothelin), causing cerebral vasoconstriction [14]. Patients with severe vasospasm may have an increased risk of rebleeding. Vasospasm can lead to insufficient cerebral blood supply, cause cerebral ischemia and hypoxia, aggravate brain damage, and also affect vascular stability, increasing the risk of rebleeding. It is reported that 50% of patients with cerebral vasospasm eventually develop cerebral infarction, and the mortality rate due to CVS is as high as 15-20% [38].

N Aoyagi et al. [39] believe that acute hydrocephalus is a risk factor for aneurysm re-rupture and bleeding. Acute hydrocephalus obstructs the cerebrospinal fluid (CSF)

circulation pathway, leading to a rapid increase in ICP. The rapid increase in ICP significantly reduces cerebral perfusion pressure, endangering cerebral tissue blood supply. To maintain cerebral blood flow, the body overactivates the sympathetic nervous system, reflexively causing a sharp increase in blood pressure. This secondary and uncontrollable hypertension becomes the most direct driver of blood impacting the aneurysm wall and causing rebleeding. At the same time, external ventricular drainage (EVD), as a standard method for treating acute hydrocephalus, is also associated with the occurrence of rebleeding in aSAH patients. Placing EVD to drain cerebrospinal fluid in aSAH patients before aneurysm treatment may cause sudden changes in intracranial pressure or transmural pressure of the aneurysm wall, thereby increasing the incidence of rebleeding. Many studies have shown that EVD is an independent risk factor for rebleeding [40-42]. Federico Cagnazzo et al. conducted a meta-analysis on the relevant research data of EVD in aSAH patients. The results showed that the incidence of rebleeding in patients receiving EVD treatment was as high as 18.8%, while that in patients not receiving this surgical method was only 6.4%. The difference between the two groups was statistically significant ($P < 0.0001$). This conclusion clearly confirms that there is a significant correlation between the application of EVD and the risk of rebleeding in aSAH patients [42].

5 Laboratory Indicators

- **Inflammation:** Inflammatory responses can exacerbate vascular wall damage and increase the likelihood of rebleeding [43].
- **Thrombocytopenia:** Thrombocytopenia may increase the risk of bleeding, but its role in SAH

rebleeding remains unclear. Platelets play an important role in hemostasis, and thrombocytopenia may lead to coagulation disorders, increasing the risk of rebleeding [20].

- **Coagulation Function Abnormality:** This may be related to rebleeding, but the specific mechanism still needs further research [44]. Abnormal coagulation function will affect the blood's coagulation ability, making it difficult to stop bleeding from the ruptured blood vessel, which is prone to rebleeding.
- **Neutrophil-Lymphocyte Ratio (NLR):** It is reported that elevated neutrophils and decreased lymphocytes at admission are associated with delayed cerebral ischemia and poor prognosis in aSAH patients. Subarachnoid hemorrhage can stimulate systemic cellular responses, induce leukocytosis and neutrophilia, leading to brain injury and delayed cerebral ischemia [45]. Studies have shown that elevated NLR (increased neutrophils and decreased lymphocytes) and subsequent inflammation lead to weakened aneurysm walls and coagulation disorders, thereby causing rebleeding [46].

6 Treatment Choices

6.1 Surgical Timing

Early surgery (within 72 hours of onset) can reduce rebleeding risk by clearing hematomas and stabilizing the aneurysm, though it may increase the risk of surgical complications [47-49]. For patients in stable condition, delayed surgery may be considered. Early surgery can clear intracranial hematomas and accumulated blood, reduce

intracranial pressure, and prevent rebleeding. However, early surgery may also aggravate brain injury and increase the risk of surgical complications. Therefore, the choice of surgical timing needs to comprehensively consider factors such as the patient's condition, aneurysm characteristics, and surgical risks [9].

6.2 Antifibrinolytic Therapy

Antifibrinolytic drugs can inhibit fibrinolysis and prevent the dissolution of blood clots at the aneurysm rupture site, thereby reducing the occurrence of rebleeding [50]. However, antifibrinolytic drugs can also inhibit the normal coagulation process and increase the risk of cerebral infarction [51]. Their use remains controversial and requires weighing the risk of rebleeding against ischemic events. Currently, there is still controversy regarding the indications and course of antifibrinolytic therapy.

7 Conclusion

Predicting rebleeding in non-traumatic SAH is complex, involving patient condition, aneurysm morphology, vasospasm, hydrocephalus, laboratory markers, and treatment factors. Although many studies have evaluated the relevant risk factors for rebleeding in non-traumatic SAH, due to the small sample size of these studies, there are certain controversies, and no expert consensus has been formed. In addition, previous studies only covered a few geographical regions, and some studies have discrepancies. Comprehensive analysis of these factors is essential for early identification of high-risk patients, taking active prevention and treatment measures, reducing the risk of rebleeding, and improving the prognosis of patients. However, there are still certain limitations in the prediction of rebleeding at present, and further research is needed to

refine predictive models and enhance the accuracy of prediction. At the same time, in clinical treatment, appropriate treatment plans should be selected according to the specific conditions of patients, so as to not only prevent the occurrence of rebleeding but also avoid the adverse consequences caused by treatment measures.

Conflict of Interest

None.

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