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Research Progress on Risk Factors and Prevention Strategies for Hemorrhagic Transformation After Mechanical Thrombectomy in Acute Ischemic Stroke

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Abstract: Acute ischemic stroke is characterized by high morbidity, high disability rate, and high mortality, representing a major cerebrovascular disease that jeopardizes national health. Severe stroke caused by large vessel occlusion progresses rapidly, and conventional intravenous thrombolysis has notable limitations. Mechanical thrombectomy can rapidly recanalize the occluded vessel and restore cerebral tissue perfusion. It has now become the principal life-saving treatment for severe acute ischemic stroke, effectively improving patients' clinical outcomes. However, post-procedural hemorrhagic transformation, as a frequent and devastating serious complication, can substantially offset the therapeutic benefits of the procedure, aggravate neurological impairment, and increase the risks of disability and death. Currently, no unified understanding has been reached regarding the risk factors and pathogenesis of this complication, and standardized and individualized prevention and treatment protocols are still lacking. Therefore, this article explores the relevant research progress on hemorrhagic transformation after mechanical thrombectomy, aiming to provide a reference for clinical risk prevention and control as well as standardized diagnosis and treatment.

Keywords: Acute ischemic stroke; post-mechanical thrombectomy; hemorrhagic transformation; risk factors; prevention and treatment strategies

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1. Overview of Acute Ischemic Stroke and Mechanical Thrombectomy

Acute ischemic stroke is the most common type of cerebrovascular disease in clinical practice, accounting for an extremely high proportion of all stroke cases, and exhibits epidemiological characteristics of high incidence, high disability rate, high mortality, and high recurrence rate. This disease is mostly caused by the occlusion of cerebral vessels, which interrupts cerebral tissue perfusion, leading to ischemic and hypoxic injury of the brain tissue, and subsequently precipitating sudden neurological deficits such as limb hemiplegia, speech disturbance, and impaired consciousness, severely threatening patients' life and quality of life. The current key points of clinical treatment for acute ischemic stroke are rapid recanalization of the occluded vessel, restoration of cerebral perfusion, salvage of the ischemic penumbra, and minimization of neurological injury [1].

Mechanical thrombectomy is currently the principal emergency treatment for acute ischemic stroke due to large vessel occlusion, and it is indicated for severe patients with intracranial large vessel occlusion confirmed by imaging within the therapeutic time window. This procedure directly removes the thrombus from the occluded vessel using interventional devices, achieving rapid vascular recanalization. It effectively compensates for the limitations of traditional thrombolytic therapy, significantly increases the success rate of recanalization, and substantially reduces the disability and mortality rates of patients with severe stroke. It has now been widely adopted in hospitals at all levels [2].

2. Definition, Classification, and Clinical Hazards of Post-procedural Hemorrhagic Transformation

Post-procedural hemorrhagic transformation after mechanical thrombectomy refers to secondary hemorrhagic injury occurring in the ischemic brain tissue after vascular recanalization. Clinically, it is mostly classified according to the European Cooperative Acute Stroke Study (ECASS) classification, mainly including petechial hemorrhage alone, small parenchymal hematoma, large parenchymal hematoma, and subarachnoid hemorrhage. The severity of hemorrhagic transformation varies considerably among different types: minor hemorrhage may present without obvious clinical symptoms, whereas severe hemorrhage can exacerbate brain tissue edema and neurological impairment, prolong the hospital stay, and substantially increase the risk of disability and long-term mortality. The occurrence of post-procedural hemorrhagic transformation directly offsets the therapeutic benefits of mechanical thrombectomy and is a key risk factor affecting patient prognosis. Therefore, systematic investigation of its risk factors and prevention and treatment strategies holds important clinical practical significance for optimizing treatment protocols and improving patient outcomes [3].

3. Risk Factors for Hemorrhagic Transformation after Mechanical Thrombectomy

3.1. Baseline Clinical Risk Factors of Patients

The patient's baseline clinical status is the fundamental factor determining the risk of post-procedural hemorrhagic transformation. Underlying conditions such as advanced age, chronic hypertension, diabetes mellitus, and hyperlipidemia continuously damage the cerebral vascular endothelium, reduce vessel wall stability, and aggravate degenerative changes in the cerebral microcirculation. Patients with atrial fibrillation are prone to thrombus formation and coagulation disorders, and patients with previous stroke have residual cerebrovascular injury, both of which significantly increase the risk of hemorrhage. A higher NIHSS score on admission indicates a larger extent of cerebral ischemic injury and more severe neurological deficits, and the probability of hemorrhagic transformation increases accordingly. In addition, abnormal indicators such as persistent hyperglycemia on admission, abnormal blood pressure fluctuations, renal impairment, coagulation imbalance, and severe anemia can disrupt the integrity of the blood-brain barrier, attenuate the brain tissue's tolerance to reperfusion injury, and ultimately induce post-procedural hemorrhage [4].

3.2. Imaging-Related Risk Factors

Preoperative imaging indicators can effectively predict the risk of postoperative hemorrhagic transformation and possess significant clinical predictive value. A larger preoperative cerebral infarct volume and a more limited ischemic penumbra suggest less reversible brain tissue and more severe ischemic injury, making reperfusion hemorrhage highly likely after vascular recanalization. Severe white matter lesions and multiple old cerebral microbleeds indicate chronic cerebrovascular injury and a marked increase in vascular fragility. Severe intracranial atherosclerosis and multi-segment vascular stenosis lead to a decline in cerebral blood flow compensatory regulation capacity; rapid blood flow reperfusion after thrombectomy can easily cause fragile vessels to rupture, representing important high-risk imaging features for postoperative hemorrhagic transformation.

3.3. Procedure-Related Risk Factors

The surgical procedure and intraoperative monitoring indicators directly affect the incidence of postoperative hemorrhage. A longer time from symptom onset to recanalization, as well as a longer time from puncture to recanalization, results in more severe persistent cerebral ischemic injury and a substantially increased risk of reperfusion injury. A poor modified Thrombolysis in Cerebral Infarction (mTICI) reperfusion grade indicates incomplete recanalization and more severe residual microcirculatory ischemic injury. Prolonged operative time and multiple retrieval attempts can readily cause mechanical injury to the intracranial vascular endothelium and even lead to intraoperative vascular dissection. Marked intraoperative blood pressure fluctuations and non-standardized use of perioperative anticoagulant and antiplatelet agents can disrupt the body's coagulation balance and damage the vascular wall structure, ultimately inducing postoperative hemorrhagic transformation [5].

3.4. Risk Factors Related to Postoperative Adjuvant Therapy and Complications

Improper postoperative medication management and disease control are key precipitating factors for hemorrhagic transformation. Unreasonable timing and inadequate dose control of postoperative anticoagulant, antiplatelet, and thrombolytic agents can suppress coagulation function and increase the risk of intracranial hemorrhage. Uncontrolled postoperative blood pressure and blood glucose can persistently damage repairing cerebral vascular endothelial cells. Meanwhile, postoperative cerebral edema and abnormally elevated intracranial pressure can compress the cerebral microcirculation and aggravate tissue injury. Systemic complications such as pulmonary infection and electrolyte disturbances can disrupt the body's metabolic homeostasis and exacerbate ischemia-reperfusion injury, collectively inducing or aggravating postoperative hemorrhagic transformation [6].

3.5. Novel Potential Risk Factors

In recent years, clinical and basic research has continuously identified multiple novel risk factors. Abnormally elevated inflammatory cytokines and oxidative stress markers can mediate inflammatory and oxidative injury to the cerebrovascular endothelium and disrupt blood-brain barrier function. Blood-brain barrier injury biomarkers can reflect the degree of vascular structural damage at an early stage and accurately predict bleeding risk. Polymorphisms in genes related to coagulation function and vascular repair can lead to individual differences in bleeding risk among patients.

4. Research Progress on the Pathogenesis of Hemorrhagic Transformation after Mechanical Thrombectomy

4.1. Mechanisms of Blood-Brain Barrier Injury

Structural and functional disruption of the blood-brain barrier (BBB) is the pathological basis for hemorrhagic transformation after mechanical thrombectomy. Under normal conditions, the BBB strictly regulates the exchange of substances between brain tissue and peripheral blood, maintaining the stability of the intracranial microenvironment. Following acute cerebral ischemia, persistent cerebral hypoxia causes swelling of vascular endothelial cells and downregulation of tight junction protein expression, leading to an initial increase in BBB permeability. After mechanical thrombectomy achieves recanalization, the rapid restoration of blood flow further impacts the damaged vascular endothelium, resulting in a breach of barrier structural integrity. Once vascular permeability is abnormally increased, plasma components and red blood cells can extravasate through the damaged vessel wall into the brain tissue interstitium, resulting in varying degrees of hemorrhagic transformation [7]. Clinical studies have confirmed that the degree of BBB injury is positively correlated with the extent and severity of postoperative hemorrhage, serving as a critical antecedent factor for bleeding.

4.2. Mechanisms of Cerebral Ischemia-Reperfusion Injury

Cerebral ischemia-reperfusion injury is a major trigger for postoperative hemorrhagic transformation. When brain tissue is in a prolonged state of ischemia and hypoxia, cellular metabolism is disrupted, energy synthesis is insufficient, and neurons and vascular endothelial cells are in a state of reversible injury. After thrombectomy, the occluded vessel is recanalized, and blood rapidly returns to the ischemic area, triggering severe reperfusion injury. A large amount of oxygen free radicals is rapidly generated, exacerbating lipid peroxidation damage to cell membranes and destroying microvascular structures. Simultaneously, reperfusion-induced cerebral edema compresses distal microvessels, causing microcirculatory obstruction and leading to secondary local congestion and vascular rupture. The longer the ischemic duration, the poorer the brain tissue's tolerance to reperfusion and the higher the likelihood of microvascular rupture and hemorrhage, significantly increasing the probability of hemorrhagic transformation [8].

4.3. Mechanisms of Inflammation and Oxidative Stress-Mediated Hemorrhagic Injury

The synergistic effect of inflammatory response and oxidative stress continuously aggravates intracranial hemorrhagic injury. Cerebral ischemia and reperfusion stimuli activate the innate immune response, promoting microglial activation and the massive release of inflammatory mediators such as tumor necrosis factor and interleukins. Inflammatory cytokines can induce vascular endothelial cell apoptosis, destabilize the microvascular wall, and exacerbate BBB damage. Concurrently, the excessively activated oxidative stress response disrupts the body's oxidative balance, leading to the accumulation of large quantities of reactive oxygen species, which persistently damage the vascular endothelium and brain tissue cells. The vicious cycle formed by inflammation and oxidative stress not only exacerbates acute-phase hemorrhagic injury but also expands the extent of brain tissue damage, worsening the degree of neurological deficit.

4.4. Mechanisms of Coagulation and Fibrinolytic System Imbalance

Dysfunction of the coagulation and fibrinolytic systems is an important intrinsic mechanism for postoperative hemorrhagic transformation. Acute severe cerebral ischemia activates the body's stress response, leading to substantial consumption of coagulation factors and decreased platelet aggregation function, resulting in a hypocoagulable state. Mechanical thrombectomy further stimulates the vascular endothelium, activating the fibrinolytic system and causing an abnormal increase in plasmin activity, leading to secondary hyperfibrinolysis. The antiplatelet and anticoagulant therapy commonly used postoperatively further interferes with the coagulation balance, resulting in insufficient hemostatic function. The combined effect of decreased coagulation and excessive fibrinolytic activation prevents effective hemostatic closure of damaged microvessels, ultimately causing red blood cell extravasation and inducing or aggravating postoperative hemorrhagic transformation, representing an important mechanism for clinical hemorrhagic complications [9].

5. Prevention and Treatment Strategies for Hemorrhagic Transformation after Mechanical Thrombectomy in Acute Ischemic Stroke

5.1. Precise Preoperative Assessment and Preventive Intervention

Precise preoperative screening and individualized intervention constitute the primary step in preventing postoperative hemorrhagic transformation. A multidimensional comprehensive assessment system should be established clinically, integrating baseline comorbidities, biochemical indicators, and imaging findings to comprehensively screen for high-risk populations. For high-risk patients with concomitant hypertension or diabetes, coagulation abnormalities, or multiple cerebral microbleeds, active symptomatic intervention should be initiated preoperatively to standardize the control of blood pressure and blood glucose levels and to correct coagulation disorders and electrolyte imbalances [10].

5.2. Standardized Intraoperative Management and Risk Control

Standardized intraoperative procedures and meticulous monitoring are key to reducing intraoperative vascular injury

and preventing postoperative hemorrhage. The operative specifications for mechanical thrombectomy should be strictly followed, with appropriate selection of thrombectomy devices based on vascular anatomical characteristics. The number of retrieval attempts should be minimized, the overall operative time shortened, and mechanical endothelial injury caused by repeated instrument manipulation avoided. Continuous intraoperative monitoring of vital signs is required to maintain blood pressure within a reasonable and stable range and to prevent drastic blood pressure fluctuations. The indications, dosage, and administration of intraoperative anticoagulants must be strictly standardized, with individualized medication based on the patient's thrombus burden and coagulation status, thereby effectively avoiding adverse events such as intraoperative vascular dissection and microcirculatory injury and reducing the risk of postoperative hemorrhage.

5.3. Refined Postoperative Preventive Management

Systematic postoperative management can effectively block the precipitating factors of hemorrhagic transformation. A stratified blood pressure and blood glucose control model should be established postoperatively, with individualized regulatory targets formulated according to the patient's recanalization status to maintain homeostasis. The initiation timing and dosage of postoperative anticoagulant and antiplatelet agents should be standardized to avoid coagulation dysfunction caused by medication misuse. Early postoperative cranial imaging reexamination and continuous neurological monitoring should be performed to promptly detect prodromal symptoms of hemorrhage such as headache and decreased consciousness [11].

5.4. Targeted Treatment Strategies for Postoperative Hemorrhagic Transformation

Graded and precise treatment should be implemented for hemorrhagic transformation of different types and severity. For patients with mild petechial hemorrhage, conservative treatment is the mainstay, including hemostasis, dehydration to reduce intracranial pressure, neurotrophic support, and improvement of cerebral circulation, along with correction of coagulation abnormalities to stabilize the patient's condition. For patients with moderate intraparenchymal hematoma, close monitoring of clinical changes is required, with optimization of dehydration and hemostasis protocols and strict control of complications. For patients with severe massive hemorrhage and significant intracranial hypertension, interventional or surgical treatment may be performed according to indications to evacuate the hematoma and reduce intracranial pressure, while comprehensive critical care combined with vital sign monitoring is carried out to reduce the risk of disability and mortality [12].

5.5. Advances in Novel Preventive and Therapeutic Approaches

With the deepening of clinical research, various novel preventive and therapeutic approaches are gradually being applied in clinical practice. Blood–brain barrier protective agents can effectively attenuate vascular endothelial injury and reduce the incidence of hemorrhagic transformation. Antioxidant and anti-inflammatory preparations can inhibit oxidative stress and inflammatory responses, thereby alleviating reperfusion injury. Individualized precision anticoagulation regimens allow dynamic adjustment of medication based on the patient's genetic profile and coagulation parameters, achieving targeted prevention and control. In addition, novel interventions such as neuroprotective therapy and hyperbaric oxygen therapy can ameliorate cerebral ischemia and hypoxia, and promote the repair of damaged neural tissue [13]. These emerging techniques and pharmacological agents provide new directions for the prevention and treatment of postoperative hemorrhagic transformation and hold broad prospects for clinical application.

6. Problems and Prospects

At present, there remain numerous deficiencies in the clinical diagnosis, treatment and research of hemorrhagic transformation after mechanical thrombectomy for acute ischemic stroke. The current screening system for risk factors of postoperative hemorrhagic transformation is relatively simplistic and lacks overall precision, making it difficult to

achieve early and accurate identification of high-risk patients. The molecular pathogenesis of the disease has not been fully elucidated, and the specific interaction mechanisms among various risk factors remain unclear. There is no unified and standardized individualized prevention and treatment protocol in clinical practice, and therapeutic decisions largely depend on the physician's clinical experience.

In the future, it is necessary to actively conduct large-sample, multicenter prospective clinical studies that integrate clinical, imaging and molecular indicators to optimize and construct a standardized risk prediction model for hemorrhagic transformation. Further in-depth investigations into the microscopic molecular pathogenesis of hemorrhagic transformation are needed to identify novel biomarkers for early warning and targeted therapy. The stratified, individualized precision prevention and treatment system should be continuously refined, and clinical diagnostic and therapeutic standards should be unified. The clinical translation and application of novel neuroprotective agents and anti-inflammatory/antioxidant interventions should be accelerated, and perioperative management of thrombectomy should be continuously optimized, so as to effectively reduce the incidence of postoperative hemorrhagic transformation and improve the long-term outcomes of stroke patients after thrombectomy [14].

7. Conclusion

The occurrence of hemorrhagic transformation after mechanical thrombectomy results from the combined effects of multiple factors, including the patient's baseline status, imaging characteristics, procedural factors and postoperative management. Its pathogenesis is closely related to blood–brain barrier disruption, reperfusion injury, inflammatory and oxidative stress dysregulation, and coagulation–fibrinolysis imbalance. Currently, clinical diagnosis and treatment still face problems such as insufficient precision of risk screening, incomplete elucidation of pathogenesis, lack of unified prevention and treatment standards, and delayed translation of novel technologies. In the future, it will be essential to rely on large-sample multicenter studies to refine risk prediction models, to thoroughly explore molecular mechanisms and novel therapeutic targets, and to continuously optimize the whole-course perioperative management protocol, thereby establishing an individualized precision prevention and treatment system. These efforts will further reduce the incidence of postoperative hemorrhagic transformation and improve the long-term prognosis of patients with acute ischemic stroke after mechanical thrombectomy.

Disclosure statement

The author declares no conflict of interest.

References

[1] Xie ZH, Xiao Y, Fang X, et al., 2026, Analysis of risk factors for adverse outcomes after mechanical thrombectomy in acute ischemic stroke and construction of a prediction model. Zhejiang Medical Journal, 48(13): 1399-1404.

[2] Huang JM, Yin ZY, Chen MY, et al., 2026, Application of systematic postural management in mechanical thrombectomy for acute ischemic stroke. Chinese Journal of Neurosurgical Disease Research, 20(7): 114-118.

[3] Chang Y, Zhang CF, Fu QZ, 2026, Research progress on inflammation and hemorrhagic transformation after mechanical thrombectomy for acute ischemic stroke. West China Medical Journal, 41(5): 827-832.

[4] Wu QY, Han J, 2026, Application effect of problemoriented nursing intervention in patients with acute ischemic stroke undergoing endovascular mechanical thrombectomy. Chinese Community Doctors, 42(14): 115-117.

[5] Sun X, Li HX, Zhu LT, et al., 2026, Efficacy of intravenous thrombolysis and mechanical thrombectomy in patients with acute ischemic stroke on the background of chronic diseases. Journal of Chronic Diseases, 27(2): 244-247.

[6] Hu YD, Xu W, Zhu ZJ, 2025, Clinical effect of mechanical thrombectomy in patients with acute ischemic stroke and its impact on vascular recanalization efficiency. Guide of China Medicine, 23(33): 21-23.

[7] Yang ZC, Zhang L, Zhao YK, et al., 2025, Research progress on prediction models for hemorrhagic transformation after mechanical thrombectomy in acute ischemic stroke. Heilongjiang Medical Journal, 49(21): 2683-2685.

[8] Huo S, Zhou WK, 2025, Predictive value of intracranial hyperdense signs after mechanical thrombectomy for hemorrhagic transformation and clinical prognosis in acute ischemic stroke. Contemporary Medicine Forum, 23(28): 19-21.

[9] Yan WX, 2025, Feasibility of stent retriever mechanical thrombectomy in elderly patients with acute ischemic stroke. Chinese Journal of Dialysis and Artificial Organs, 36(3): 37-40.

[10] Ban GL, Li CX, Chen J, et al., 2025, Construction of a risk prediction model for hemorrhagic transformation after mechanical thrombectomy in patients with acute ischemic stroke. Bachu Medical Journal, 8(3): 81-87.

[11] Gao LZ, 2025, Efficacy of mechanical thrombectomy combined with intraarterial thrombolysis in the treatment of acute ischemic stroke. Medical Innovation of China, 22(22): 94-98.

[12] Zhang WY, Li ZC, Zan X, et al., 2025, Risk factors for hemorrhagic transformation after mechanical thrombectomy in acute ischemic stroke caused by anterior circulation large vessel occlusion. Journal of Stroke and Neurological Diseases, 42(6): 523-528.

[13] Niu SY, Hou SG, Dong JQ, et al., 2025, Observation on the therapeutic efficacy of mechanical thrombectomy for acute ischemic stroke. Journal of Jinzhou Medical University, 46(3): 60-64.

[14] Tao H, Zhong WT, Wu JH, et al., 2025, Influence of age on the therapeutic effect of mechanical thrombectomy in patients with acute ischemic stroke. Chinese Journal of Gerontology, 45(11): 2585-2589.