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CURRENT TREATMENT STRATEGIES AND PATHOGENESIS OF STROKE

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Annotation. Every year, around 800,000 individuals experience new or recurrent strokes, with most of these being new cases. Approximately 87% are ischemic, 10% reflect intracranial hemorrhage (ICH), and 3% are SAH. Despite a general decrease in stroke incidence during the past 30 years, it is projected that by 2030, an additional 3.4 million US.The pathogenesis of stroke is highly complex, multifactorial, and involves multiple interconnected mechanisms. It includes neurovascular unit dysfunction, hemodynamic disturbances, excitotoxicity, calcium overload, mitochondrial dysfunction, oxidative stress, inflammatory responses, apoptosis, and autophagy. These processes interact dynamically and contribute to neuronal injury and infarct progression.Despite significant advances in understanding these mechanisms, the precise sequence of events and the extent of their interaction remain incompletely elucidated. In particular, the integrated role of metabolic factors—such as calcium–phosphorus homeostasis, lipid metabolism, and hormonal regulation—in the progression and severity of ischemic stroke has not yet been fully clarified.Therefore, stroke pathogenesis remains only partially understood, and further experimental and clinical research is required to better define its molecular mechanisms and to develop more effective, individualized therapeutic strategies.

Key words: Ischemic stroke, Stroke pathogenesis, Calcium–phosphorus homeostasis, mineral metabolism, hyperphosphatemia, hyperphosphatemia, vascular calcification, neurovascular dysfunction, atherosclerosis,dyslipidemia, cerebral hemodynamics.

INSULT PATOGENEZI VA DAVOLASHGA ZAMONAVIY YONDASHUVLAR

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Annotatsiya. Har yili 800 000 ga yaqin kishi yangi yoki qayta insultni boshdan kechiradi, bunda aksariyat holatlar ilk bor yuzaga kelgan xurujlar hissasiga to‘g‘ri keladi. Insultlarning taxminan 87 foizi ishemik, 10 foizi miya ichi qon quyilishi (Intracerebral Hemorrhage, ICH) va qariyb 3 foizi subaraxnoidal qon quyilishi (Subarachnoid Hemorrhage, SAH) dir. So‘nggi 30 yil ichida insult bilan kasallanish darajasi umumiy pasayganiga qaramay, 2030-yilga kelib bu ko‘rsatkich oshishi taxmin qilinmoqda. Insult patogenezi o‘ta murakkab va ko‘p omilli jarayon bo‘lib, o‘zaro bog‘liq bo‘lgan quyidagi mexanizmlarni o‘z ichiga oladi: neyrovaskulyar birlik disfunksiyasi, gemodinamik buzilishlar, eksaytotoksiklik, kalsiyning ortiqcha to‘planishi, mitoxondrial disfunksiya, oksidativ stress, yallig‘lanish reaksiyalari, apoptoz va autofagiya. Bu jarayonlar bir-biri bilan dinamik tarzda o‘zaro ta’sirlashib, neyronlarning shikastlanishiga va infarktning avj olishiga sabab bo‘ladi. Ushbu mexanizmlarni o‘rganishda erishilgan salmoqli yutuqlarga qaramay, hodisalarning aniq ketma-ketligi va ularning o‘zaro ta’sir darajasi hali yetarlicha o‘rganilmagan. Xususan, kalsiy-fosfor gomeostazi, lipidlar almashinuvi va gormonal boshqaruv kabi metabolik omillarning ishemik insult og‘irligining shakllanishi va rivojlanishidagi integrativ roli hozirgi kunga qadar to‘liq aniqlanmagan. Shunday qilib, insult patogenezi qisman o‘rganilganligicha qolmoqda, bu esa

kasallikning molekulyar mexanizmlarini aniqroq tushunish hamda samarali individuallashtirilgan davolash strategiyalarini ishlab chiqish uchun keyingi eksperimental va klinik tadqiqotlarni o‘tkazishni taqozo etadi.

Kalit so‘zlar: Ishemik insult, insult patogenezi, kalsiy-fosfor gomeostazi, mineral almashinuvi, giperfosfatemiya, qon tomirlar kalsifikatsiyasi, neyrovaskulyar disfunksiya, ateroskleroz, dislipidemiya, serebral gemodinamika.

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Introduction. Stroke is a leading cause of adult-onset disability. Over half of stroke survivors beyond 6 months are dependent on others for at least one activity of daily living. Annually, there are over 12 million incident cases of stroke, of which 65% are ischemic strokes. Patients with stroke with symptomatic steno-occlusive disease have a risk of recurrent stroke of at least 10–15% within 5 years. Globally, stroke accounts for over seven million deaths each year [1]. Modifiable risk factors include blood pressure management, blood glucose and cholesterol control, physical activity, weight management, and environmental exposure such as air pollution[2, 3].Acute ischemic stroke is a heterogeneous disease with various stroke subtypes, including atherothrombotic infarct involving large vessel occlusions, cardioembolic infarct, lacunar infarct involving penetrating arteries and perforators, subcortical infarct without clear etiology, and infarct of unusual etiology such as hypercoagulable states, vasculitis, genetic disorders, and dissection.

Increasing evidence suggests that the brain is exquisitely sensitive to even short-duration ischemia and that multiple mechanisms are involved in the tissue damage that results from cerebral ischemia. Ischemic stroke initiates a cascade of events, including ATP depletion, ionic dysregulation, the increased release of glutamate and the excess production of free radicals, as well as edema and inflammation; all these events eventually contribute to cell death[4]. In contrast, in intracerebral hemorrhage, oppression and destruction of brain tissue by hematoma is the primary cause of brain injury, but inflammation, coagulation response and the toxicity of the released hemoglobin play a pivotal role as well[5]. Cell death after ischemic stroke has been attributed in the past mainly to necrosis or apoptosis, but recent reports show the involvement of other newly described forms of cell death(Figure 1).

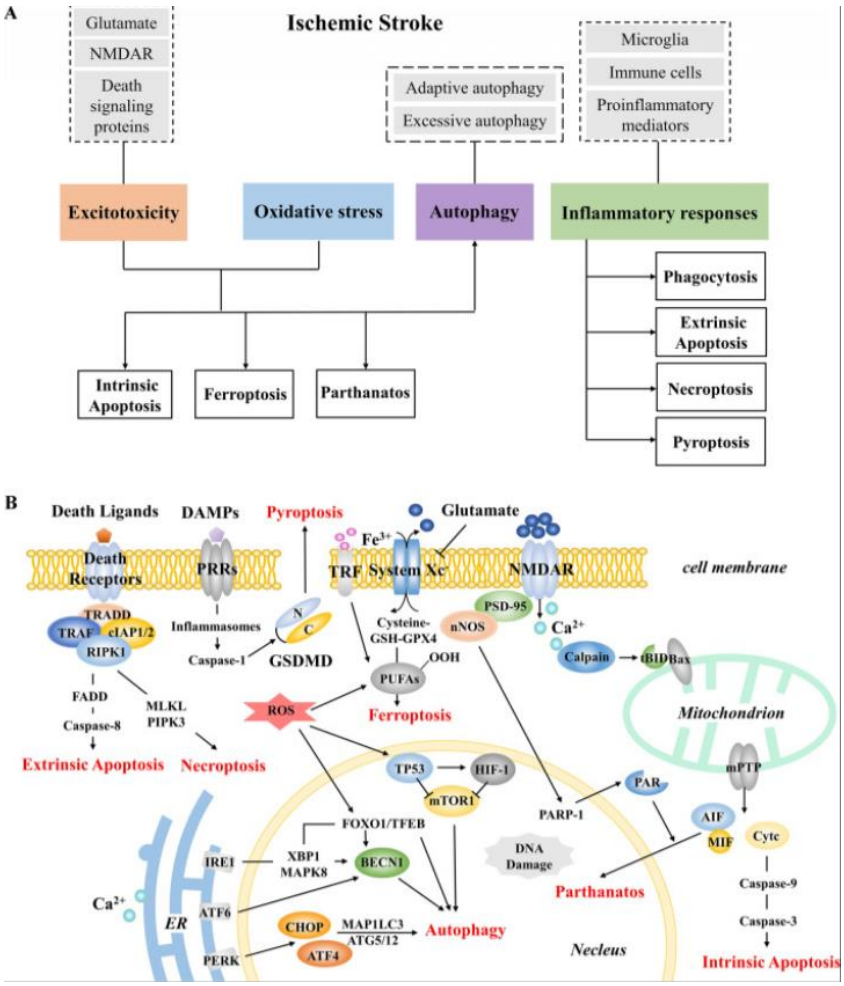

Fig 1.Overall several death mechanisms in ischemic stroke.

The modified Rankin scale (mRS) is a functional assessment scale measuring the degree of disability or dependence in patients with stroke. It is a 7-point scale ranging from 0 (no symptoms) to 6 (death)[6] (Table 1). The National Institutes of Health Stroke Scale (NIHSS) is a 15-item neurological examination used to assess stroke severity and changes in clinical status. The NIHSS score ranges from 0 to 42, scoring areas including level of consciousness, eye movements (gaze deviation), visual fields, facial weakness, motor strength, limb ataxia and coordination, sensory loss, language function (dysphasia/aphasia), speech clarity (dysarthria), and neglect/inattention (hemispatial neglect) (Table 2) [7]. NIHSS scores of ≥ 10 are 73% sensitive and 74% specific for predicting the presence of large vessel occlusion [8].

mRS	Daily function
0	No symptoms
1	No significant disability
2	Slight disability, independent but unable to carry out all previous ADL
3	Moderate disability, can walk independently but requires help with ADL
4	Moderately severe disability, not independent with ADL
5	Severe disability, full-time nursing care
6	Death

ADL activities of daily living

Table1. Modified Rankin scale (mRS)

Stroke severity based on NIHSS score

NIHSS score	Stroke severity
0	No stroke symptoms
1–4	Minor stroke
5–15	Moderate stroke
16–20	Moderate to severe stroke
21–42	Severe stroke

Table2. Stroke severity score NIHSS

Cerebral collateral circulation represents a complex compensatory vascular network that maintains cerebral perfusion during arterial occlusion. Multiple anatomical pathways—including the anterior and posterior communicating arteries, leptomeningeal collaterals, external carotid artery connections, dural meningeal arteries, ACA–PCA limbic loop pathways, and anterior spinal artery anastomoses—contribute to alternative blood flow supply. The quality of collateral circulation significantly influences tissue viability and clinical outcomes[9]. Well-developed collaterals are associated with preserved distal perfusion and improved prognosis, whereas poor collateral status correlates with larger infarct size and worse neurological outcome. Therefore, assessment of collateral circulation is crucial for prognostication and therapeutic decision-making in ischemic stroke (Figure2).

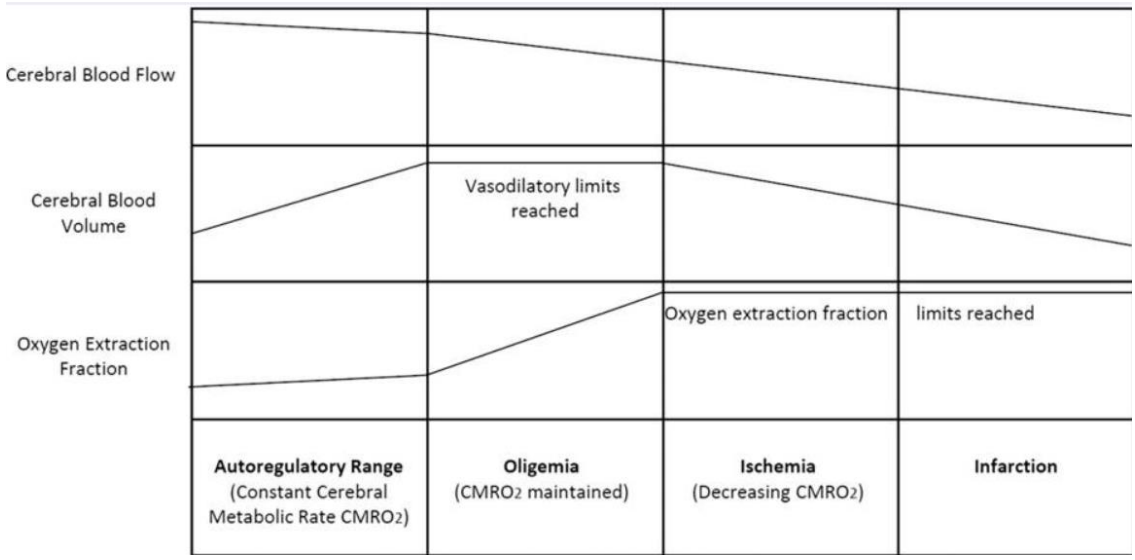

Fig.4. Stages of cerebral hypoperfusion.

Several clinical factors affect the extent of cerebral hypoperfusion in the setting of ischemia. They include extent of hemodynamic impairment and cardiac status, cerebral autoregulatory impairment and extent of compensatory cerebral collateral circulation, presence of metabolic syndrome of hypertension, hyperlipidemia, and insulin resistance, and brain–body interactions affecting blood oxygen carrying capacity and delivery (including coagulation status, anemia, other hematologic disorders, systemic infection and sepsis, renal and hepatic disorders)[2].

The 2026 International Stroke Conference highlighted major advancements in cerebrovascular science and reinforced the importance of global collaboration in improving stroke care. The release of the updated acute ischemic stroke guidelines reflects significant progress in expanding treatment options, including mobile stroke units, broader thrombolysis use, extended eligibility for mechanical thrombectomy, and the inclusion of pediatric stroke care. The conference emphasized equitable healthcare systems, innovative recovery strategies, hypertension management, and the growing role of artificial intelligence in stroke medicine. Overall, ISC26 underscored the continuing evolution of evidence-based stroke management and the critical role of international cooperation in advancing research and clinical outcomes worldwide.

Cerebrovascular mean flow velocities

	Normal MFV (cm/s)
Artery	
ICA siphon	< 70
MCA M1–M2	< 80
ACA A1	< 80
BA	< 50
VA	< 60
PCA	< 50
Arterial stenosis (50%)	
ICA siphon, ACA (A1)	> 90
MCA M1–M2	> 100
BA-VA-PCA	> 70

Fig.5. MFV mean flow velocity, ICA internal carotid artery, MCA middle cerebral artery, ACA anterior cerebral artery, BA basilar artery, VA vertebral artery, PCA posterior cerebral artery

Tissue plasminogen activator (tPA), or alteplase, is the gold standard thrombolytic agent given within 4.5 h of stroke symptom onset [1]. Alteplase binds to fibrin, a mesh-like protein in clots and acts within the endogenous fibrinolytic cascade to convert plasminogen to plasmin, the enzyme that dissolves fibrin resulting in clot dissolution. Tissue plasminogen activator (tPA), or alteplase, is the gold standard thrombolytic agent given within 4.5 h of stroke symptom onset . Alteplase binds to fibrin, a mesh-like protein in clots and acts within the endogenous fibrinolytic cascade to convert plasminogen to plasmin, the enzyme that dissolves fibrin resulting in clot dissolution.

Conclusion. Given the complex and multifactorial nature of ischemic stroke, integrating the broader metabolic and vascular framework may enhance understanding of underlying mechanisms. Further experimental and clinical studies are required to clarify its prognostic significance and to support the development of targeted, individualized therapeutic strategies.

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