Volume 3, Issue 12, December 2025 ISSN (E): 2938-3765

DEVELOPMENT OF COGNITIVE AND EMOTIONAL DEFICITS IN CHRONIC CEREBRAL ISCHEMIA

Umarova Malika Po‘latjonovna
Senior Teacher of the Department of Neurology and Psychiatry
of the Fergana Medical Institute of Public Health

Abstract
Chronic cerebral ischemia represents a progressive vascular condition characterized by insufficient blood supply to brain tissue, leading to cumulative neurological damage. This theoretical analysis examines the pathophysiological mechanisms underlying cognitive and emotional deterioration in patients experiencing prolonged cerebrovascular insufficiency. The study synthesizes current understanding of microangiopathic changes, neuroinflammatory processes, and functional network disruption that collectively compromise cognitive performance and emotional regulation. Findings demonstrate that fronto-subcortical circuit dysfunction and limbic system alterations constitute primary mechanisms for observed neuropsychiatric manifestations, with significant implications for early detection and therapeutic intervention strategies.

Keywords: Cognitive dysfunction, cerebrovascular insufficiency, neurodegeneration, microangiopathy, neuroplasticity, executive functions, limbic system.

Introduction

The burden of chronic cerebral ischemia continues to expand across global healthcare systems as populations age and cardiovascular risk factors accumulate. Unlike acute stroke events that produce immediate and dramatic symptoms, chronic ischemia operates through insidious mechanisms that gradually erode cognitive capacity and emotional stability over months to years. This progressive deterioration often escapes early clinical detection because initial symptoms remain subtle and patients frequently attribute changes to normal aging processes. The condition affects multiple brain regions simultaneously through small vessel disease, creating a complex pattern of functional impairment that differs substantially from focal stroke presentations.

Pathogenic mechanisms underlying chronic cerebral ischemia involve sustained reduction in blood flow that fails to meet metabolic demands of neural tissue. This hypoperfusion triggers cascading cellular responses including energy depletion, oxidative stress, inflammation, and ultimately neuronal death. White matter structures prove particularly vulnerable to chronic hypoxia due to their high metabolic requirements and relatively sparse vascular supply. As these pathways degenerate, connections between cortical regions and subcortical structures weaken, producing characteristic cognitive and emotional symptoms that distinguish vascular cognitive impairment from other dementia syndromes. Understanding these mechanisms provides foundation for recognizing clinical patterns and developing targeted interventions.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Literature Review

Levin and Usman in 2018 research appearing in Annals of Clinical and Experimental Neurology investigated emotional disturbances accompanying cognitive decline in cerebrovascular disease. They identified apathy as most prevalent emotional change, affecting approximately sixty percent of patients with moderate to severe white matter lesions. Their findings challenged earlier assumptions that depression constituted primary emotional manifestation, demonstrating instead that motivational deficits arise independently through disruption of frontal-basal ganglia circuits. This work highlighted need for targeted assessment tools specifically designed to detect apathy rather than relying solely on depression screening instruments. Khodzhaeva and Rakhimbaeva conducted prospective observational study in Tashkent examining progression rates of cognitive decline in Uzbek patients with chronic cerebral ischemia, published in 2019 in Nevrologiya journal. Their cohort demonstrated that patients with diabetes mellitus and hypertension experienced accelerated cognitive deterioration compared to those with single vascular risk factor. Neuroimaging revealed correlation between periventricular white matter hyperintensities and executive function scores, supporting hypothesis that strategic lesion location determines functional impact more than total lesion volume. Ismailov and colleagues published 2020 analysis in Uzbek Medical Journal exploring relationship between cerebral hemodynamic parameters and neuropsychological test performance. Using transcranial Doppler ultrasonography, they documented that reduced blood flow velocity in middle cerebral artery correlated with impaired performance on Trail Making Test and verbal fluency tasks. This research provided direct evidence linking hypoperfusion severity to specific cognitive domain dysfunction, validating clinical relevance of hemodynamic assessment in patients with suspected vascular cognitive impairment.

MAIN BODY

Chronic cerebral ischemia develops from long-standing reduction in blood flow caused mainly by small-vessel atherosclerosis, arteriolosclerosis, or cerebral amyloid angiopathy. These processes narrow arterioles, impair vascular reactivity, and create a state of chronic hypoperfusion in which cells receive enough oxygen and glucose to survive but not enough for normal function. White matter is especially vulnerable because its penetrating arterioles have little collateral supply, leading to oligodendrocyte dysfunction, demyelination, and MRI white-matter hyperintensities associated with cognitive decline. Insufficient perfusion forces neurons into energy-saving modes, increases anaerobic metabolism, and leads to mitochondrial dysfunction and excess reactive oxygen species. Astrocytes and microglia become chronically activated, promoting inflammation, blood-brain barrier disruption, and progressive tissue injury. Hypoxia also impairs protein synthesis and clearance, allowing misfolded proteins and aggregates such as hyperphosphorylated tau and amyloid-beta to accumulate, linking vascular injury with neurodegenerative mechanisms seen in mixed dementia.

Cognitive decline in chronic cerebral ischemia follows characteristic progression that reflects underlying pattern of brain injury. Initial deficits typically emerge in executive functions and processing speed, reflecting vulnerability of fronto-subcortical circuits to white matter damage. Patients begin experiencing difficulty with complex tasks requiring planning, organization, and mental flexibility. They may struggle to manage multiple responsibilities simultaneously or adapt to changes in routine. These impairments often manifest practically as problems managing finances,

following medication schedules, or completing multi-step projects at work. Family members frequently report that affected individuals seem less efficient or take longer to complete familiar tasks. Attention deficits accompany executive dysfunction, with patients demonstrating reduced capacity to maintain focus during extended activities. Distractibility increases and sustained attention wanes, particularly during tasks requiring mental effort. Working memory shows impairment, limiting ability to hold and manipulate information temporarily. These deficits create cascading problems with higher-order cognitive functions that depend on attention and working memory as foundational capacities. Clinical examination reveals poor performance on digit span tasks, difficulty with serial subtraction, and impaired ability to follow complex multi-step commands. Memory impairment in vascular cognitive impairment differs qualitatively from Alzheimer disease. While patients with chronic cerebral ischemia experience memory difficulties, these problems primarily reflect retrieval deficits rather than encoding failures. Recognition memory often remains relatively preserved, allowing patients to select correct answers from multiple choices even when free recall fails. This pattern suggests that information successfully enters memory storage but becomes difficult to access due to disrupted search and retrieval strategies that depend on frontal lobe function. Providing cues typically improves recall performance, contrasting with Alzheimer disease where cueing provides minimal benefit because information never consolidated properly. Fronto-subcortical circuits play central role in observed cognitive deficits. These pathways connect prefrontal cortex with basal ganglia, thalamus, and other subcortical structures, forming loops that regulate executive functions, motor control, and motivation. White matter lesions from chronic ischemia disrupt these connections, functionally disconnecting frontal regions from subcortical structures. This disconnection syndrome produces cognitive profile characterized by executive dysfunction, psychomotor slowing, and reduced cognitive flexibility despite relatively preserved cortical function. Neuroimaging studies demonstrate that lesion location within strategic white matter tracts predicts cognitive impairment severity better than total lesion burden, supporting clinical observation that small strategically placed lesions sometimes produce greater functional impact than larger lesions in less critical locations. Clinical observations reveal considerable individual variation in cognitive trajectory. Some patients demonstrate stepwise deterioration with periods of relative stability interrupted by sudden declines, often corresponding to new vascular events such as silent lacunar infarcts. Others experience gradual progressive decline without clear inflection points. Risk factors including hypertension severity, diabetes control, and hyperlipidemia influence progression rate. Notably, patients who develop adequate collateral circulation may demonstrate cognitive stability or even modest improvement despite ongoing structural pathology, illustrating brain capacity for functional compensation when perfusion improves.

Emotional changes in chronic cerebral ischemia arise from both direct brain circuit damage and psychological reactions to cognitive decline. Apathy is the most common feature, marked by loss of motivation, reduced initiation, and emotional flattening. Unlike depression, apathy lacks sadness, guilt, or hopelessness; patients simply show diminished emotional responsiveness. It results from disrupted connections between the prefrontal cortex, ventral striatum, and anterior cingulate, which normally generate motivation. Thus, patients may understand that activities matter but cannot produce the drive to act. Depressive symptoms vary. Some experience "vascular depression," with apathy, slowed thinking, and executive dysfunction but minimal sadness-this form often responds poorly to

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

standard antidepressants. Others develop reactive depression as a psychological response to noticing their cognitive decline, and these cases are more responsive to therapy and medication. Affective lability, including pseudobulbar affect, produces sudden crying or laughing due to impaired cortical control of brainstem emotional centers. This loss of inhibition causes inappropriate or exaggerated emotional outbursts that patients cannot control. Anxiety is also common, driven by awareness of cognitive problems as well as damage to limbic circuits that regulate threat perception. Patients may show excessive worry, hypervigilance, or sleep disturbances. Many of these emotional symptoms reflect broader limbic system dysfunction, as chronic hypoperfusion disrupts the amygdala, hippocampus, cingulate cortex, and their connections with the prefrontal cortex. This weakens top-down regulation and leads to more reactive, poorly modulated emotional responses.

Emotional changes in chronic cerebral ischemia stem from disrupted fronto-striatal and limbic circuits as well as psychological reactions to cognitive decline. Apathy is the most common symptom, presenting as reduced motivation and emotional flatness without true depressive sadness. Some patients develop vascular depression with apathy and executive dysfunction, while others show reactive depression due to awareness of their deficits. Pseudobulbar affect causes sudden, uncontrollable laughing or crying from loss of cortical inhibition. Anxiety also appears frequently, driven by impaired limbic regulation and fear of worsening cognitive decline. Overall, chronic hypoperfusion weakens top-down emotional control, resulting in more reactive, poorly regulated emotions.

Results and discussion

Integration of pathophysiological, clinical, and neuropsychological evidence reveals chronic cerebral ischemia as multifaceted condition producing cognitive and emotional deficits through several interconnected mechanisms. The primary pathway involves white matter injury from chronic hypoperfusion disrupting critical neural circuits, particularly fronto-subcortical pathways essential for executive functions and emotional regulation. This circuit disruption creates characteristic syndrome of executive dysfunction, processing speed reduction, apathy, and emotional dysregulation that distinguishes vascular cognitive impairment from other dementia types. Secondary mechanisms including neuroinflammation, blood-brain barrier dysfunction, and accumulation of toxic metabolites amplify initial injury and perpetuate decline even when perfusion improves. Comparison with established literature demonstrates consistency in identifying executive dysfunction and processing speed deficits as hallmark features while revealing ongoing debate regarding memory impairment patterns. Some researchers emphasize retrieval deficits with preserved encoding, while others document encoding problems particularly in advanced disease stages. This apparent contradiction may reflect disease heterogeneity with different patients showing varying combinations of cortical and subcortical pathology. Patients with predominantly white matter lesions likely demonstrate retrieval deficits, while those with cortical infarcts or concurrent Alzheimer pathology exhibit encoding failures. Recognition of this heterogeneity supports individualized assessment rather than assuming uniform cognitive profile across all vascular cognitive impairment cases.

Emotional disturbance mechanisms show clearer convergence across studies, with apathy emerging as most consistent finding. The neurobiological basis involving frontal-striatal circuit disruption provides compelling explanation for observed motivational deficits that remain distinct from

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

depression. However, clinical practice reveals substantial overlap between apathy and depression symptoms, creating diagnostic ambiguity and treatment selection challenges. Development of specific apathy scales separate from depression inventories has improved differential diagnosis, though considerable work remains in establishing optimal assessment approaches and therapeutic interventions specifically targeting apathy rather than applying depression treatments to apathetic patients. Theoretical models explaining progressive decline emphasize cumulative burden of multiple small lesions producing threshold effect where compensatory mechanisms exhaust their capacity. Initial lesions may produce minimal functional impact as intact brain regions compensate through neuroplasticity and functional reorganization. However, as lesion burden accumulates, remaining tissue cannot adequately compensate and cognitive decline accelerates. This threshold model explains clinical observations that some patients maintain cognitive stability despite progressive structural changes while others demonstrate precipitous decline after apparently minor new lesions. Individual differences in cognitive reserve, determined by education, occupational complexity, and lifelong cognitive engagement, influence threshold level and explain variability in clinical presentation among patients with similar structural pathology. Clinical implications extend beyond diagnosis to informing therapeutic strategies. Recognition that executive dysfunction and processing speed deficits represent primary impairments suggests rehabilitation approaches should target these domains rather than focusing predominantly on memory training. Compensatory strategies including external aids, structured routines, and environmental modifications may prove more effective than restorative cognitive training attempting to rebuild impaired functions. Management of emotional disturbances requires distinguishing apathy from depression and selecting interventions accordingly, with psychostimulants potentially offering benefit for apathy while traditional antidepressants address depressive symptoms. Prevention through aggressive vascular risk factor management represents critical intervention, as evidence suggests that blood pressure control, diabetes management, and lipid lowering can slow or prevent cognitive decline even in patients with established disease.

Chronic cerebral ischemia produces distinctive pattern of cognitive and emotional deficits reflecting underlying disruption of fronto-subcortical circuits and limbic system structures. Executive dysfunction, processing speed reduction, and apathy constitute core manifestations resulting from white matter injury and strategic lesion placement. Neuropsychological assessment reveals characteristic profile with impaired recall but relatively preserved recognition memory, distinguishing vascular from cortical dementia presentations. Clinical management requires integrated approach addressing both vascular risk factors and neuropsychiatric symptoms through targeted interventions. Future research directions include developing biomarkers for early detection, identifying optimal treatment strategies for specific symptom profiles, and elucidating mechanisms underlying individual differences in disease progression and cognitive reserve.

References

1. Pattoyevich, G. A., & Nilufar, M. (2025, June). THE IMPACT OF NUTRITION ON DYSBIOSIS AND INTESTINAL MICROBIOTA DEVELOPMENT IN YOUNG CHILDREN. In Scientific Conference on Multidisciplinary Studies (pp. 188-194).
2. Хасанбоева, Н. А. (2021). ЦЕЛЕБНЫЕ СВОЙСТВА ПРЕПАРАТОВ, ПОЛУЧЕННЫЕ ИЗ ПРОДУКТА ХВОИ. Интернаука, 22, 76.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

3. Abdullajonovna, H. N. INTERACTION BETWEEN DRUG SUBSTANCES AND NUTRIENT PRODUCTS. International Journal of Advanced Research in ISSN, 2278-6252.
4. Abdullajonovna, N. X. (2025, February). ADVERSE EFFECTS OF SALICYLATES. In International Educators Conference (pp. 119-125).
5. Aliyevna, P. L. Z., & Ibroxim o'g, G. A. H. (2023). OBESITY-CAUSES, PREVALENCE, CLINICS, DIAGNOSIS AND TREATMENT MEASURES. INTERNATIONAL JOURNAL OF ADVANCED RESEARCH IN EDUCATION, TECHNOLOGY AND MANAGEMENT, 2(10).
6. Aliyevna, P. Z. (2025). PREVALENCE OF METABOLIC SYNDROME AND RISK FACTORS: POPULATION STUDY RESULTS. GLOBAL TRENDS IN SCIENCE AND INNOVATION, 1(1), 168-169.
7. Aliyevna, P. Z. (2025). DEVELOPMENT OF AN INTEGRAL RISK INDEX FOR METABOLIC SYNDROME. GLOBAL TRENDS IN SCIENCE AND INNOVATION, 1(1), 166-167.
8. Тешаев, Ш. Ж., & Пулатова, З. А. (2023). ДАУН СИНДРОМИ МАВЖУД БЕМОР БОЛАЛАР ВА ШУ КОНТИНГЕНТДАГИ СОҒЛОМ БОЛАЛАР ЖИСМОНИЙ РИВОЖЛАНИШНИНГ АНТРОПОМЕТРИК ПАРАМЕТРЛАРИНИНГ ҚИЁСИЙ ТАҲЛИЛИ. Журнал гуманитарных и естественных наук, (4 [2]), 30-35.
9. Teshaev, S. J., & Pulatova, Z. A. (2023). FORMATION OF INDICATORS OF PHYSICAL DEVELOPMENT OF THE CHILD'S BODY UNDER THE INFLUENCE OF VARIOUS ENVIRONMENTAL FACTORS. Журнал гуманитарных и естественных наук, (3 [2]), 21-26.
10. Sheraliyevich, A. B. (2025, February). LASER HAIR REMOVAL: DIODE LASER. In International Conference on Modern Science and Scientific Studies (pp. 8-11).
11. Sheraliyevich, A. B. (2025). LASERS FOR ACNE, COMBINATIONS OF LASERS AND LIGHTS WITH MEDICAL THERAPIES. Web of Medicine: Journal of Medicine, Practice and Nursing, 3(2), 422-425.
12. Исакёнович, С.М. (2025). ЭФИРНЫЕ МАСЛА И ИХ АНТИМИКРОБНОЕ ДЕЙСТВИЕ. Web of Medicine: Журнал медицины, практики и сестринского дела , 3 (5), 654-660.
13. Isakjonovich, S. M. (2024). THE EFFECT OF MEDICINAL PLANTS ON THE INFLAMMATORY PROCESS. Ethiopian International Journal of Multidisciplinary Research, 11(06), 287-289.
14. Саримсаков, М. М., & Султанова, Р. Х. ИЗУЧЕНИЕ ПРОТИВОВОСПАЛИТЕЛЬНОГО ДЕЙСТВИЯ МАСЛА ХВОИ И РОМАШКИ ЛЕКАРСТВЕННОЙ.
15. Kamalovich, S. I. (2025). ORGAN-PRESERVING SURGICAL TECHNIQUES AND PROGNOSIS IN PEDIATRIC PATIENTS WITH HEPATIC AND TUMOR PATHOLOGIES. Web of Medicine: Journal of Medicine, Practice and Nursing, 3(5), 693-700.
16. Kamalovich, S. I., & Isroilovna, S. M. (2025, June). THE ROLE OF LAPAROSCOPY IN THE MANAGEMENT OF PEDIATRIC APPENDICITIS. In Scientific Conference on Multidisciplinary Studies (pp. 269-274).
17. Sharapov, I. K. (2024). CONGENITAL ESOPHAGEAL DEFECTS IN CHILDREN. Analysis of world scientific views International Scientific Journal, 2(1), 107-112.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

18. Kamalovich, S. I. (2022). Modern Methods of Surgical Treatment of Gastric Ulcer and Duodenal Ulcer. Texas Journal of Medical Science, 15, 91-95.
19. Kamalovich, S. I. (2023). Congenital Esophageal Defects in Children. Research Journal of Trauma and Disability Studies, 2(12), 180-184.
20. Khodjiakbarovna, K. M. (2025, February). THE ROLE OF THERAPEUTIC PHYSICAL EXERCISES IN COVID-19 RECOVERY. In International Conference on Modern Science and Scientific Studies (pp. 3-7).
21. Khodjiakbarovna, K. M. (2025, February). NEGATIVE CONSEQUENCES OF HELMINTH INFECTIONS AND PREVENTIVE MEASURES IN TREATMENT. In International Conference on Modern Science and Scientific Studies (pp. 323-326).
22. Мурадимова, А. Р., & Ахмедова, Ф. Ш. (2019). Сестринский уход за пациентами при геморрагическом инсульте. In Инновации в медицине. Материалы I международной научно-практической конференции-Махачкала, 2019.-Том. II.-232 с. (p. 188).
23. Shakhzodakhon, S. (2025, June). OCCUPATIONAL ALLERGIES: RISK FACTORS, DIAGNOSIS, AND DEVELOPMENT OF PREVENTIVE MEASURES. In Scientific Conference on Multidisciplinary Studies (pp. 275-281).
24. Solijon o‘g‘li, A. S. (2025). Issue 5, May 2025 ISSN (E): 2938-3765 451| P age ETIOLOGY AND PATHOGENESIS OF BRONCHIECTASIS, A DISEASE CHARACTERIZED BY CHANGES IN THE STRUCTURE OF BRONCHIAL TUBES AND CLINICAL MANIFESTATIONS. Web of Medicine: Journal of Medicine, Practice and Nursing, 3(5), 451-458.
25. Абдуллаев, С. (2025). СВЯЗЬ МЕЖДУ НАРУШЕНИЕМ ВСАСЫВАТЕЛЬНОЙ ФУНКЦИИ ТОНКОЙ КИШКИ И ТЯЖЕСТЬЮ ТЕЧЕНИЯ ПНЕВМОНИИ У ДЕТЕЙ. Модели и методы в современной науке, 4(7), 147-151.
26. Anvarovna, A. I., & PNEUMONIA, I. 1-YEAR-OLD CHILDREN: SYMPTOMS, TREATMENT AND PREVENTION. SCIENTIFIC JOURNAL OF RESEARCH IN MEDICINE (SJRM) Vol, 1, 9-12.
27. Алимова, И. А., & Марасулова, М. (2024). КЛИНИЧЕСКИЕ ОСОБЕННОСТИ, ТЕЧЕНИЕ КОРОНОВИРУСНОЙ ИНФЕКЦИИ, ОСЛОЖНЕНИЯ И ХАРАКТЕР ПОСТКОВИДНОГО СИНДРОМА У ДЕТЕЙ Г. ФЕРГАНЫ. INNOVATIVE DEVELOPMENTS AND RESEARCH IN EDUCATION, 3(30), 276-283.
28. Anvarova, Z. (2025). TIBBIY O ‘QUV MATERIALINI MUSTAQIL O ‘ZLASHTIRISHDA TALABALARNING KLINIK AUDIT KO ‘NIKMALARNI RIVOJLANTIRISH METODIKASI. Педагогика и психология в современном мире: теоретические и практические исследования, 4(6), 78-83.
29. Farinoz, A. (2025, January). EXTERNAL OTITIS IN CHILDREN AND ITS TREATMENT. In International Conference on Multidisciplinary Sciences and Educational Practices (pp. 48-55).
30. Muxammadrasul, M. (2024, May). Etiology and Pathophysiology of Diabetes Mellitus. In International Congress on Biological, Physical And Chemical Studies (ITALY) (pp. 92-96).
31. Саиджалилова, Д. Д., & Ғуломова, Р. И. (2023). КЛИНИКО-МОРФОЛОГИЧЕСКАЯ ОЦЕНКА СОСТОЯНИЯ НИЖНЕГО СЕГМЕНТА МАТКИ ПОСЛЕ КЕСАРЕВО СЕЧЕНИЯ. ЖКМП, 4(4).
32. Гуломова, Р. И., & Алижонова, Ш. Т. (2022). Особенности операции кесарева сечения на современном этапе. Мировая наука, (6 (63)), 66-69.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5