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Clinical significance of cerebrospinal fluid dynamics in chronic ischemic processes of the brain

Nargiza Shukhratovna Muzafarova
Samarkand State Medical University, Uzbekistan

Farrukh Shukhratovich Chuliev
Samarkand State Medical University, Uzbekistan

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Annotation: Disorders of cerebrospinal fluid (CSF) dynamics play a critical role in the development of chronic cerebral ischemia, particularly in cases of dyscirculatory encephalopathy (DE). Chronic ischemia arises from impaired cerebral blood flow, which results in prolonged oxygen and nutrient deprivation within neural tissues. This deficit fosters progressive degenerative changes in the nervous system. Altered CSF circulation further exacerbates ischemic and hypoxic injury to brain structures. Numerous studies have shown that impaired CSF outflow combined with elevated intracranial pressure can intensify vascular dysfunction, leading to persistent cognitive impairments, emotional instability, and various neurological symptoms. Employing a comprehensive management strategy including the assessment and correction of CSF dynamic disturbances is therefore crucial for patients with chronic cerebral ischemia, as it contributes to improved clinical outcomes, enhanced quality of life, and a slower progression of the pathological process.

Keywords: cerebrospinal fluid dynamics, vascular insufficiency, cognitive functions.

INTRODUCTION

Recent analyses of scientific literature have demonstrated that, despite notable advances in understanding the molecular mechanisms underlying cerebrovascular diseases, the pathophysiology of dyscirculatory encephalopathy (DE) remains insufficiently explored. The lack of a standardized diagnostic algorithm and the absence of universally effective treatment strategies indicate that the key pathogenic mechanisms of DE are not fully understood. Therefore, investigating factors that contribute to the development of atrophic changes in cerebral tissue remains a pressing issue in modern neurology and clinical neuroscience.

Objective:

The primary aim of this study was to evaluate the significance of cerebrospinal fluid (CSF) dynamic disturbances in the pathogenesis of dyscirculatory encephalopathy (DE).

Materials and Methods

The study included 317 patients diagnosed with DE, comprising 118 men and 199 women, aged between 30 and 60 years (mean age 60 ± 2.0 years). The diagnosis of DE was established based on a combination of clinical and neurological examinations, supported by multiple objective diagnostic methods:

- Magnetic Resonance Imaging (MRI) of the brain,
- MR angiography of intra- and extracranial arteries,
- Echoencephalography (Echo-ES),
- Transcranial Doppler ultrasonography (TCD), and
- Lumbar puncture to measure CSF pressure and evaluate the residual oxidizing capacity of cerebrospinal fluid (ROC-CSF).

The findings from these methods suggested that disturbances in CSF dynamics, often arising from autoimmune or aseptic inflammatory processes, contribute to cerebral hypoperfusion. This hypoperfusion, in turn, promotes the activation of glycolytic (anaerobic) metabolic pathways in neural tissue.

Results

It is well-established that anaerobic glycolysis produces only two molecules of ATP, equivalent to approximately 61 kJ/mol of free energy. This level of energy production is critically insufficient to sustain normal neuronal function and support metabolic needs of brain cells.

Our study indicated that CSF dynamic disturbances, particularly those resulting from fibrotic adhesions within the brain and its meninges, serve as a primary factor leading to catabolic metabolic shifts in neural tissue. These metabolic disturbances exacerbate cerebral hypoperfusion, compromise energy homeostasis, and accelerate degenerative processes in the central nervous system.

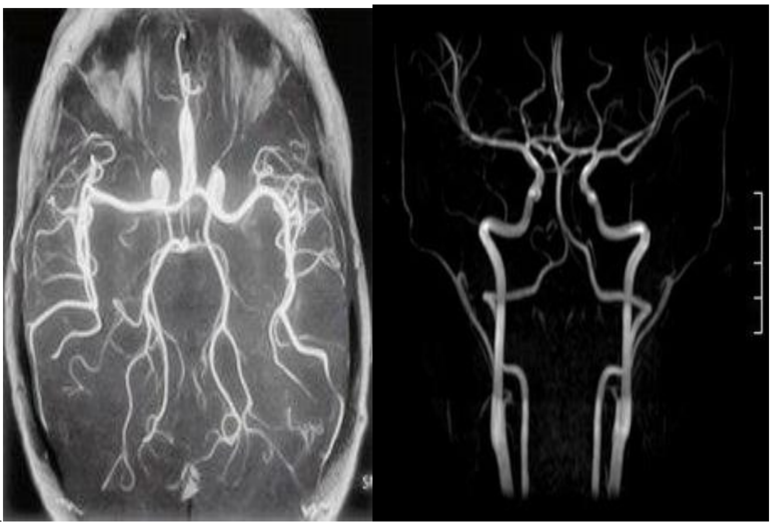
Assessment Methods

To quantify the severity of CSF dynamic disturbances, the following techniques were employed:

1. Dynamic Echo-ES to monitor cerebrospinal fluid circulation,
2. Lumbar puncture for measuring CSF pressure and determining ROC-CSF,
3. MR angiography of intra- and brachiocephalic arteries to identify the vascular causes of hypoperfusion that trigger anaerobic glycolysis.

These methods provided an integrated assessment of both the cerebrovascular and cerebrospinal fluid dynamics, enabling a detailed understanding of the pathological mechanisms underlying DE.

To assess the severity of cerebrospinal fluid (CSF) dynamic disturbances in the brain, dynamic Echo-encephalography (Echo-ES), lumbar puncture with measurement of CSF pressure, and evaluation of the residual oxidizing capacity of cerebrospinal fluid (ROC-CSF) were employed. The underlying causes of cerebral hypoperfusion, which contribute to the activation of anaerobic glycolysis, were investigated using MR angiography of the intracranial and brachiocephalic arteries (Figure 1).



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To determine the extent of cerebrospinal fluid (CSF) dynamic disturbances in the brain, dynamic Echo-encephalography (Echo-ES), lumbar puncture with CSF pressure measurement, and assessment of the residual oxidizing capacity of cerebrospinal fluid (ROC-CSF) were utilized. The factors responsible for cerebral hypoperfusion, which promote the onset of anaerobic glycolysis, were examined through MR angiography of the intracranial and brachiocephalic arteries (Figure 1).

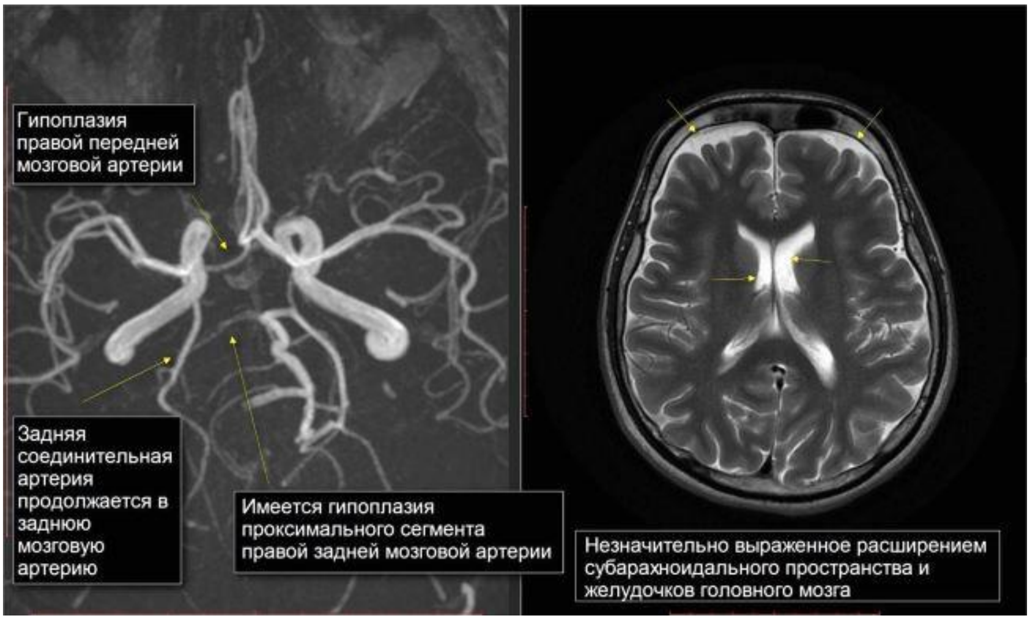

Figure 2 – English Version (High-paraphrase, Expanded)

In patients with stage III dyscirculatory encephalopathy (DE), brain MRI revealed marked expansion of the subarachnoid spaces over the convexity, enlargement of perivascular channels, and dilation of the ventricular base cisterns. On Echo-encephalography (Echo-ES), multiple high-amplitude echo signals with bifurcated peaks were observed, accompanied by pronounced widening of the base of the midline M-echo. Notably, pulsations of the echo signals were absent.

The systolic cerebral blood flow velocity, measured via transcranial Doppler (TCD), averaged over 200 cm/s. Lumbar cerebrospinal fluid (CSF) pressure measurements averaged 370 ± 20 mm H_2O, while the residual oxidizing capacity of CSF (ROC-CSF) was 27 ± 4.0 mg %.

These findings from clinical-neurological assessments and objective imaging techniques collectively indicate that disturbances in CSF dynamics are present to varying degrees across all stages of DE and constitute a major contributing factor to the development of cerebral hypometabolism.

Pathophysiological Mechanisms

The observed CSF dynamic disturbances are often secondary to autoimmune or aseptic inflammatory processes, which lead to cerebral hypoperfusion and trigger glycolytic (anaerobic) metabolic pathways. Anaerobic glycolysis produces only two molecules of ATP per glucose, equivalent to approximately 61 kJ/mol of free energy, which is insufficient to meet the energy demands of neurons. Consequently, neurons cannot sustain normal cellular functions under prolonged energy deficit.

During this intracellular energy depression, a subset of neurons undergo apoptosis, while others survive by relying on exothermic energy released from the breakdown of macromolecular complexes (protein–mucopolysaccharide–lipid matrix). This represents a catabolic (destructive) metabolic process. However, the energy derived from catabolism remains inadequate for maintaining normal neuronal activity, and thus, these catabolic processes progress gradually over time.

Oxidative Stress and Neurodegeneration

Persistent intracellular energy deficits are accompanied by the release of highly reactive free radicals (HRFRs), which possess strong destructive potential. Under the influence of HRFRs, pre-existing degenerative and atrophic changes in the brain are exacerbated, leading to reduction in brain volume and mass observed in DE. Proteins dissociated from mucopolysaccharide complexes during catabolic metabolism act as autoantigens, triggering chronic autoimmune inflammation within brain parenchyma, ventricles, and meninges. This inflammatory process results in adhesion formation (fibrosis), which further disrupts CSF dynamics and contributes to cerebral hypoperfusion. The subsequent depression of intracellular energy synthesis perpetuates a vicious cycle of metabolic insufficiency, neurodegeneration, and inflammation.

Conclusions

1. CSF dynamic disturbances arising from fibrotic adhesions within the brain parenchyma and meninges represent a primary factor driving catabolic metabolism in neural tissue.
2. Such disturbances are consistently observed across all stages of DE and are a key contributor to cerebral hypometabolism.

These mechanisms highlight the central role of CSF circulation abnormalities in the pathophysiology of DE, linking structural brain changes, hypoperfusion, and metabolic insufficiency in a complex interrelated process.

REFERENCE

1. Khakimova S.Z., Mamurova I.N., Samiev A.S. Clinical role of neurobrucellosis among patients with chronic radiculopathy. Academy 2019. 29-228. .(in Russ).
2. Khakimova S. Z., Khamdamova B. K., Kodirov U. O. Comparative correlation of markers of

inflammatory metamorphism in peripheral blood in dorsopathies of various origins // Uzbek Journal of Science Reports. – 2022. – T. 2. – No. 2. – pp. 12-18. .(in Russ).
3. Khamdamova B.K., Khakimova S.Z., Kodirov U.A. Features of the neurovascular state of the spine in dorsopathies in patients with diabetes mellitus // Journal of Biomedicine and Practice. – 2022. – vol. 7. – No. 6. .(in Russ)
4. Samiev A.S., Khakimava S.Z., Soibnazarov O.E. Rehabilitation of patients who have undergone spinal surgery. Journal of Biomedicine and Practice. 2022, vol. 7, issue 1, pp.139-144. .(in Russ).
5. Samiev A.S., Mavlyanova Z.F. British Medical Journal Volume-3, No. 2 39 Optimization of rehabilitation measures for lumbar spondylogenic radiculopathies.
6. Samiev A.S., Mavlyanova Z.F. Comprehensive rehabilitation of patients with lumbar spondylogenic radiculopathies. SCIENCE AND EDUCATION ISSN 2181-0842 VOLIUME 4, ISSU 2. 2023. Pp.453-461. .(in Russ)
7. Aldabergenova A.B, Biryuchkov MYu. Magnetic resonance imaging in the diagnosis of osteochondrosis of the lumbar spine. Journal of Theoretical and Clinical Medicine. 2000;3:107-108 (in Uzbek)
8. Drivotinov B.V, Polyakova T.D, Pankova M.D. Physical rehabilitation in neurological manifestations of osteochondrosis of the spine: textbook. allowance. Minsk: BGUFK, 2005 (In Russ.)
9. Khakimova S.Z, Akhmadeeva L.R. Markers of endothelial dysfunction in distal vessels of patients with chronic pain syndrome in dorsopathies of various origins. Uzbek journal of case reports.2022;l. 2:26-30 (In Russ.)
10. Khakimova S.Z, Atokhodjaeva D.A. Features of Pain Syndrome of Patients with Brucellosis if Damaged Nervous System. Medico-legal update. 2020;20:3.
11. Mulleman D, Mammou S, Griffoul I, Watier H, Goupille P. Pathophysiology of disk-related sciatica. I. – Evidence supporting a chemical component. Joint Bone Spine. 2006;73:151–158.
12. Novoseltsev SV. Pathogenetic mechanisms of the formation of lumbar spondylogenic neurological syndromes in patients with herniated lumbar discs. Manual Therapy 2010;3:77-82 (In Russ.)
13. Asadullaev M.M. Acute pain syndrome in vertebroneurology and its correction. Scientific and practical journal. Neurology.2005;1(25):5-8 (In Russ.)
14. Samibaev R, Samiev A, Mamurova I. Clinical and electroneuromyographic diagnosis and rehabilitation of vertebrogenic lumbosacral radiculopathy. Youth and medical science in the XXI century. 2017;179-179 (In Russ.)
15. Skoromets AA, Skoromets AP, Skoromets TA. Neurological status and its interpretation. Educational guide for doctors. MEDpress-inform, 2013;43 (In Russ.)
16. Tikhanova EP, Sergeeva IV. Clinical manifestations of neurobrucellosis. Modern problems of science and education. 2013;4 (In Russ.)
17. Tretyakov AV, Tretyakov VP. Clinical and neuroimaging comparisons of pain syndrome in lumbosacral dorsopathy. Neurological Bulletin. 2010;7(3):55-59 (In Russ.)
18. Filatova ES., Erdes ShF. Polyneuropathy in rheumatoid arthritis: significance in the pathogenesis of pain syndrome. Russian medical journal. 2017:7;470-473 (In Russ.)

19. Bektashev R.B., Bektashev O.R., Ergashev M.B. "Neurometabolic therapy in the complex treatment of epilepsy" Medical Journal of Uzbekistan. 2015. No. 2 p. 79-80. (In Russ.)
20. Vershchagin N.V. "Pathology of the vertebrobasilar system and cerebrovascular accidents." – M. Medicine. 1980 – 312 p. (In Russ.)
21. Vershchagin N.V., Morgunov V.A., Gulyevskaya T.S." Pathology of the brain in atherosclerosis and arterial hypertension." – M. Medicine., 1997-287p. (In Russ.)