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Improving Treatment Strategies for Acute Cerebrovascular Disease

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Abstract This article analyzes the effectiveness of dehydration therapy in reducing local cerebral edema during the treatment of cerebrovascular disease, specifically ischemic stroke. The analysis uses the NIH Stroke Scale (NIHSS) and Scandinavian Stroke Scale scores, along with hemodynamic indicators, to assess the severity and dynamics of neurological deficits.

Keywords Tashkent Medical Academy, Ischemic stroke, NIHSS and Scandinavian scales, Duplex scanning, MSKT

1. Introduction

Cerebrovascular diseases rank among the leading causes of death and disability. Globally, the annual incidence of primary strokes exceeds 15 million. This figure is approximately 750,000 in the US, 500,000 in Russia, 100,000 in Ukraine, and 40,000 in Uzbekistan. In Uzbekistan, the incidence of cerebral stroke is 0.9-1.5 per 1000 population. Globally, ischemic heart disease ranks first among causes of death, followed by stroke. Ischemic strokes are 5-6 times more frequent than hemorrhagic strokes: ischemic strokes account for 80-85% of all strokes, while hemorrhagic strokes account for 15-20%. Stroke is a process that develops over several minutes (sometimes several hours or days), ranging from simple functional changes in the brain to irreversible changes – necrosis [16].

Ischemic stroke is the most common type of acute cerebrovascular accident, based on local cerebral ischemia, with diverse underlying causes. The most frequent causes of ischemic stroke development are atherosclerotic lesions of extra- and intracranial cerebral vessels, arterial hypertension, and their combined occurrence, as well as diabetes mellitus and the relatively less common, but currently increasing, cerebral vasculitis [1,2,12,15]. Numerous studies show that stenotic or occlusive changes in cerebral vessels, especially extracranial ones, are crucial in the development of ischemic stroke in 84-90% of cases [1,2,4,14]. Stenotic and occlusive changes in extracranial vessels are 2-5 times more common than in intracranial vessels [14,17]. Atherosclerotic changes in cerebral blood vessels lead to reduced cerebral blood flow and, consequently, excitation of the circulatory center in the medulla oblongata, resulting in vasoconstriction and increased arterial blood pressure.

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2. Materials and Methods

Atherosclerosis contributes not only to the development of arterial hypertension but also to the exacerbation of existing hypertension [4,8,12,13,14]. The prevalence of arterial hypertension in patients with carotid artery stenosis is 72%. The combined occurrence of atherosclerotic lesions of the carotid artery and arterial hypertension further increases the risk of ischemic stroke [8,11,12].

The widespread use of neuroimaging techniques has enabled accurate identification of ischemic stroke subtypes. Of all ischemic strokes, 34% are atherothrombotic, 22% cardioembolic, 15% hemodynamic, 20% lacunar, and 9% are of the hemorheological microocclusion type [1].

In recent years, the significant role of cerebrovascular hemodynamic disturbances in the pathogenesis of cerebrovascular diseases has been emphasized. New pharmacological agents are being actively introduced into medical practice, their main effect aimed at treatment through normalization of brain tissue permeability, reducing the effects of ischemia and hypoxia, restoring microcirculation, and normalizing vascular tone. One such drug is Lidozinate. The drug quickly breaks down into lysine and aescin ions in the blood and readily crosses the blood-brain barrier. The main active substance determining the pharmacological

properties of the drug is aescin. Lidozinate has endotheliotropic, venotonic, anti-inflammatory, and analgesic effects [3].

Current research on ischemic stroke, despite the abundance of diagnostic methods assessing brain function and vascular status, often shows low treatment efficacy. This is due to the failure to consider the diversity of underlying processes, the etiology of this pathology, and the specific characteristics of vascular changes. Therefore, in cases of confirmed cerebral stroke, it is crucial to investigate the contributing factors, leading to a significant reduction in disability and mortality rates.

Exclusion Criteria:

- Patients with hyperdense lesions detected on CT scan.
- Patients with severe somatic pathologies (myocardial infarction, pneumonia, cancer, decompensated diabetes), severe uncontrolled arterial hypertension (not controlled by antihypertensive drugs), and those receiving concomitant therapy with mannitol and/or saluretics were excluded from the study.

Study Design:

The study included 50 patients (n=50) who were randomly divided into two groups:

- Group 1 (Control): 25 patients received standard baseline therapy and standard treatment for ischemic stroke.
- Group 2 (Experimental): 25 patients received baseline therapy, standard treatment, and intravenous Lidozinate (10 ml in 100 ml of physiological saline) for 10 days to combat cerebral edema.

Assessments:

All patients (n=50) underwent clinical neurological (NIHSS and Scandinavian scales), angiographic (duplex scanning of neck vessels), and neuroimaging (brain CT scan on day 1) assessments on days 1, 2, and 10 of hospitalization.

3. Results and Discussions

The mean age of patients in Group I (N=25) was 52.5 ± 1.4 years, with 72.0% (n=18) being male and 28.0% (n=7) female. The mean age of patients in Group II (n=25) was 50.4 ± 1.5 years, with 76.0% (n=19) being male and 24.0% (n=6) female (Table 1).

In both groups of patients, the overall clinical neurological status was primarily assessed, paying particular attention to the neurological symptoms that developed and their severity (Table 2).

Table 1. Number and Mean Age of Patients by Group

	Total (n=50)	I–Group (n=25)	II – Group (n=25)
Males	37 (74,0%)	18 (72,0%)	19 (76,0%)
Famels	13 (26,0%)	7 (28,0%)	6 (24,0%)
Mean Age	51,5±1,4	52,5±2,15	50,5±1,8

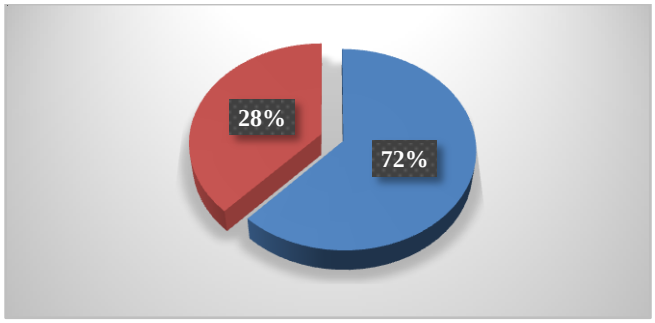

Figure 1. Gender Distribution of Patients - Group 1 (n=25)

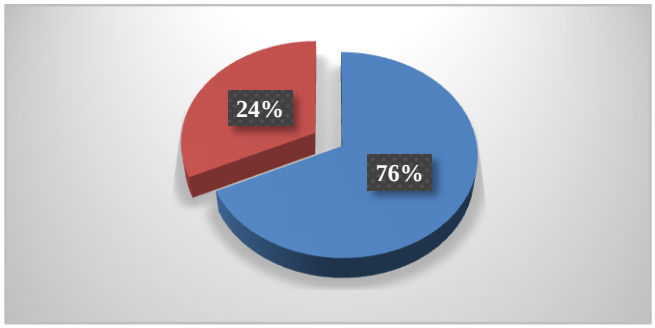

Figure 2. Gender Distribution of Patients - Group 2 (n=25)

Table 2. Distribution of Patients by Main Subjective Clinical-Neurological Symptoms

Symptoms of the Disease	I group n=25		II group n=25	
	abs	%	abs	%
Headache	44±0,7	(91.7)	27±0,9	(96,4)
Tinnitus (ringing in the ears)	33±0,9	(64.5)	25±1,1	(89,3)
Dizziness	37±1,1	(77,1)	24±1,2	(85,7)
Memory Impairment	23±0,8	(47.9)	19±0,8	(67,8)
Visual Impairment	46±0,6	(95.8)	27±0,9	(96.4)

4. Conclusions

1. Therefore, the study results demonstrate that the use of Lidozinate normalizes brain tissue permeability, reducing the impact of ischemia and hypoxia. This plays a crucial role in both early and late neuronal damage processes in acute ischemic stroke. Neurological scales showed a significant improvement in the clinical presentation of the disease, confirmed by a reduction in the severity of neurological deficit according to clinical indicators. The use of Lidozinate effectively improves intracranial blood flow due to improved venous circulation, which in turn leads to improved cerebral arterial perfusion.
2. Atherosclerosis and pathological deformation of the carotid arteries are considered the main causes of ischemic stroke development. Over time, if reconstructive surgery of the carotid arteries is not performed in a timely manner, the degree of stenosis in the neck vessels increases, leading to an increased risk of ischemic stroke.

REFERENCES

[1] Akmetov V.V., Lemenov V.A., et al. Features of the atherosclerotic process in patients with transient ischemic attacks. Collection of theses. IX All-Russian Congress of Neurologists. Yaroslavl 2006. -P. 368.

[2] Barkhatov D.Yu., Jibladze D.N., Barkhatova V.P. The relationship between clinical and biological disorders in atherosclerotic lesions of the carotid arteries. // Journal of Neurology and Psychiatry. -2006; 106: 4. -P. 10-14.

[3] Belkin A.A., Alasheev A.M., Inyushkin S.N. Transcranial Dopplerography in intensive care. Methodological guide for physicians. – Yekaterinburg, 2006. - P. 40-60.

[4] Vereshchagin N.V., Piradov M.A. Stroke: assessment of the problem. // Neurological Journal, 1999, -No. 5, -P. 4-7.

[5] Gaydar B.V., Semenutyn V.B., Parfenov V.E., Svistov D.V. Transcranial Dopplerography in neurosurgery. Monograph. - St. Petersburg, 2008. - P. 57-80.

[6] Gafurov B.G. Clinical and epidemiological characteristics of cerebral strokes in Uzbekistan // Materials of the Republican scientific-practical conference "Current problems of neurology and psychiatry", Andijan, 2004. - P. 82-83.

[7] Gafurov B.G. Problems and prospects for the development of the neurological service in the Republic of Uzbekistan // Neurology. -2002. - No. 4. – P. 22-24.

[8] Gusev E.I., Skvortsova V.I. Ischemia of the brain. -M.: Medicine, 2001. -328 p.

[9] Zenkov L.R. Functional diagnostics of nervous diseases. Ultrasound diagnostics of vascular diseases of the nervous system (Nikitin Yu.M.) M.: Medicine, 2004. P. 384-435.

[10] Rakhimjanov A.R., Askerov Sh.A. Rheumatic diseases of the nervous system. – Tashkent, 1978.

[11] Skvortsova V.I. Ischemic stroke: pathogenesis of ischemia, therapeutic approaches // Neurological Journal, 2001, -No. 3, - P. 4-9.

[12] Skvortsova V.I., Sokolov K.V., Shamalov N.A. Arterial hypertension and cerebrovascular disorders. // Journal of Neurology and Psychiatry. -2006; 106: 11. -P. 57-64.

[13] Chazov I.E., Mychka V.B., Mamyrbayev K.M., et al. Cerebrovascular complications in metabolic syndrome: possible approaches to reducing risk. // Journal. Therapeutic Archive. -2004. -Vol. 76. -No. 6. - P. 74–80.

[14] Shnayder N.A., Vinogradova T.E. Prevention of atherothrombotic stroke. Methodological guide. – Krasnoyarsk: KrasGMA, 2003. – P. 4-20.

[15] Yakhno N.N., Vilensky B.S. Stroke as a medico-social problem. // Russian Medical Journal. -2005. -Vol. 13. -No. 12. -P. 807-815.

[16] Yakhno N.N., Shtulman D.R. Diseases of the nervous system. Moscow 2003. –P. 228-238. Benninger D.H., Georgiadis D., Kremer C., Studer A., Nedeltchev K., and Baumgartner R.W. Mechanism of Ischemic Infarct in Spontaneous Carotid Dissection // Stroke. 2004. Vol. 35: no. 1. pp. 482 – 485.

[17] Goldstein L.B., Bertels C., Davis J.N. Interrater reliability of the NIHSS stroke scale. Arch Neurol 1989; 46: 660-662.

[18] Scandinavian Stroke Study Group: Multicenter trial of hemodilution in ischemic stroke. Background and study protocol. Stroke 1985; 16: 885-890.