AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 2, Issue 1, 2024 ISSN (E): 2993-2149

Comparison of Pathomorphological Changes in the Brain and Other Organs in Women Who Died as a Result of Ischaemic Vertebrobasilar and Hemispheric Strokes

Malika Ilkhomovna Kamalova
Associate Professor, Department of Human Anatomy Samarkand State Medical University

Remzi Ibragimovich Suleymanov
Assistant of the Department of Human Anatomy Samarkand State Medical University

Abstract: The article presents the results of pathomorphological studies in women who died as a result of ischaemic stroke. The obtained data confirm the concept of pathogenetic heterogeneity of ischaemic strokes and the necessity of the earliest possible pathogenetically grounded treatment.

Keywords: ischaemic stroke, pathomorphological changes, pathogenesis.

Introduction. Every year, up to 6 million people die of stroke worldwide, and about 5 million people remain disabled and dependent on external assistance after suffering a stroke [9]. In our country, strokes are the second most important cause of death and the leading cause of disability, creating a serious burden on the health care system, the economy, and the entire society. Only 20% of stroke survivors return to active life [2,5].

The absolute majority of acute cerebral circulatory disorders are ischaemic strokes (IS) [4, 9], of which up to 20% develop in the vertebrobasilar basin (VBB) [6, 7, 10]. The VBB includes functionally and phylogenetically different parts of the brain - the cervical spinal cord, brain stem and cerebellum, part of the thalamus and hypothalamic region, occipital lobes, posterior and mediobasal parts of temporal lobes [6, 8]. Clinical manifestations of ischaemic strokes in the IBS depend on many factors, and their diagnosis is often difficult due to the atypical clinical picture and the rarity of classical symptoms [1, 6].

The choice of therapeutic tactics determining the outcome of the disease depends on timely and correct diagnosis. In this regard, it seems important to further improve early diagnosis of ischaemic strokes in order to optimise treatment tactics in the acute period of the disease.

The aim of the study was to compare pathomorphological changes in the brain and other organs in persons who died as a result of ischaemic vertebrobasilar and hemispheric strokes.

Material and methods: The results of pathomorphological studies of the frontal, parietal, temporal and occipital lobes of the brain, hypothalamus, corpus callosum, optic tubercle, caudate nucleus, cerebral pedicles, Varolian bridge, medulla oblongata and cerebellum, as well as the heart and large vessels of 30 deceased with a confirmed diagnosis of ischemic stroke were analysed. The deceased were hospitalised in the neurological department of the Tashkent Medical Academy 2021-2023. Material was taken 4-12 hours after death with staining of preparations according to Van Gee-zon. Analysis of pathomorphological changes in the main extra- and intracerebral arteries, myocardium, coronary arteries and heart valves, aorta, as well as

changes in different parts of the brain allowed to specify the pathogenetic variant of AI. Pathomorphological studies at the Republican Pathologoanotomic Scientific and Practical Centre of Tashkent city.

RESULTS: Patients aged 71-80 years prevailed among those who died as a result of ischaemic stroke - 15 (50 %). In the age group of 61-70 years there were 7 (23 %) patients, 51-60 years and over 80 years - 4 (13,3 %) observations each. Among those who died at the age of 51-70 years, males predominated (73 %). At the age of over 71 years, 13 (68 %) female patients prevailed.

Within the first 3 days from the onset of the disease 5 (17 %) patients died. In the interval from 4 to 10 days - 20 (67 %) patients, after 11-15 days - 4 (13 %) patients. One patient died on the 20th day after ischaemic stroke.

RESULTS: Autopsy revealed lesions in the following vascular basins: in the basin of the left anterior cerebral artery - 2 (7 %) observations, right middle cerebral artery - 12 (40 %), left middle cerebral artery - 13 (43 %), right posterior cerebral artery - 6 (20 %), right superior cerebellar artery - 3 (10 %) and right posterior cerebellar artery - 2 (7 %) observations. Simultaneous lesion of the left and right middle cerebral artery basins occurred in 5 (17 %) cases. The most frequent localisation of the lesion in our observations was the cerebral hemispheres. In 4 (13 %) observations the focus was located in the brain stem, in 3 (10 %) - in the cerebellum, in 2 (7 %) - in the pontine region, in 2 (7 %) - in the thalamus, in 4 (13 %) - in the subcortical nuclei. Simultaneous development of two or more foci was observed in 7 (23 %) cases.

Extensive ischaemic stroke spreading to all brain regions receiving blood from the middle and anterior cerebral arteries (the entire internal carotid artery basin) occurred in 5 (17 %) observations. Large infarcts were detected in 21 (70 %) of the deceased, and medium infarcts in 4 (13 %) cases. Large and medium ischaemic strokes were more often multiple - from 2 to 3 foci. "White" ischaemic infarcts (without haemorrhagic transformation) were detected in 19 (63 %) of the deceased, "red" infarcts (with haemorrhagic transformation) - in 11 deceased (37 %).

Atherothrombotic ischaemic stroke was detected in 15 (50 %) of the deceased against the background of complicated atherosclerosis of precerebral arteries of large or medium calibre and combined complicated atherosclerosis of precerebral and cerebral arteries. Precerebral arteries (internal carotid, vertebral), large and middle cerebral arteries were affected by atheromatous plaques of eccentric character, mainly in the area of their proximal sections, as well as in the places of their division, tortuosity, and fusion. The narrowing of the vessel lumen varied from 25 to 75 %. Stenosing plaques in the arteries of the carotid basin were usually combined with widespread atherosclerosis of the arteries of the vertebro-basilar system. Cerebral arteries in 87 % of cases were affected in the area of the circle of villisia, atherosclerosis of cerebral arteries was ob-literative, reducing the arterial lumen up to 75 %. In trunk localisation of stroke, atherosclerotic lesions of the distal segments of the vertebral arteries were detected in all cases, more often in the area of the main artery formation. The degree of vertebral artery lumen narrowing varied from 50 to 80%. Such combined atherostenosis led to a sharp decrease in the possibilities of collateral circulation through the cerebral arterial circle, contributing to an increase in the size and number of ischaemic foci. A haemorrhagic component was frequently encountered in this variant of stroke.

Atheromatous plaques were complicated by the development of atherothrombosis. In such plaques there was damage to the endothelial cover or deep ulceration, on which thrombotic masses were layered, leading to complete closure of the vessel lumen or to its critical narrowing. Numerous lacunar infarcts along the course of small intracerebral arteries in the atherothrombotic variant of stroke could be a consequence of transient ischaemic attacks.

Ischaemic stroke of embolic genesis was detected in 13 (43 %) patients and was observed in case of complete or partial occlusion of a cerebral artery by an embolus. Most often such pathogenetic variant was found in the zone of blood supply of the middle cerebral artery. The size of the nidus

was, as a rule, medium or large, it was in such cases that the haemorrhagic component was more often attached. The morphological sign of cardioembolic stroke was the presence of a thromboembolus from the aorta or heart valve leaflet. The thromboembolus had no connection with the deendothelialised arterial wall, so only homogenisation, thickening of thromboembolic masses and appearance of haemosiderin were observed in dynamics. The appearance of endotheliocytes, fibrocytes, macrophages in the thickness of the thromboembolus, and later the covering of the thromboembolus with endotheliocytes was not characteristic for thromboembolism. Thromboembolism of aortic origin occurred in 9 (30%) cases. Cardiogenic emboli in heart valve lesions were detected in 4 (13 %) observations. In 3 (10 %) observations cardiogenic emboli were combined with thromboembolism of aortic origin. It should be noted that signs of ischaemic heart damage were revealed in 7 (23 %) of the deceased, large-focal (postinfarction) cardiosclerosis - in 4 (13 %), small-focal cardiosclerosis - in 26 (87 %). Signs of myocardial hypertrophy were detected in all cases.

The hemodynamic variant of ischemic stroke was diagnosed in 2 (7%) cases with vertebrobasilar localisation of stroke and was due to cerebral hypoperfusion with the development of cerebrovascular ischaemia. This variant developed against the background of atherosclerotic stenosis of extra- and intracranial arteries in the zone of adjacent blood supply. Morphological signs of the hemodynamic mechanism of MI were: free lumen of the corresponding cerebral artery except for the presence of atherosclerotic plaque, absence of arterial wall paresis, smooth shiny intact endocardium without wall thrombi, absence of such in the initial aortic section. These signs are relative, not absolute, because successful treatment, in principle, can "free" the vessel lumen from thrombotic masses.

In patients with ischaemic strokes in the VBB we also determined signs of chronic vertebrobasilar insufficiency in the form of lacunar infarcts of varying age - microcirculatory strokes associated with the lesion of perforating arteries. They showed signs of neuronal death and proliferation of glial elements, and atrophic changes in the cortex of the large hemispheres were additionally determined. Such changes were detected in 6 (20%) autopsy cases. None of these cases developed haemorrhagic transformation.

Comparison of pathomorphological changes in different terms showed that the maximum changes were observed on the 2nd - 3rd day from the onset of the disease. During this period the focus of complete collisional necrosis was clearly delimited from the unaffected tissues, within which all structural elements of the nervous tissue - nerve cells and fibres, neuroglia, vessels - were destroyed. Ischemic damage of neurons, cytolysis, loss of neurons with preservation of other structural elements of brain substance, tigrolysis (chromatolysis) and hyperchromatosis in the remaining neurons, disintegration of myelin in nerve fibres of white matter were also observed. Tigrolysis was accompanied by other cell changes - swelling and displacement of the nucleus to the periphery, appearance of lipofuscin or vacuoles in the cytoplasm, cell shriveling and atrophy.

In the perifocal zone surrounding the zone of ischemic focus, dysge-mic disorders were constantly revealed: signs of venous stasis and spasm of arterioles, which was accompanied by reduction of their lumen and reduction of blood supply to cerebral structures, plasma saturation of their walls, perivascular edema, single small hemorrhages and focal changes of brain tissue in the form of edema, dystrophic changes of neurons. Sharp pericellular and perivascular oedema of the brain substance was detected around the unaffected areas, which contributed to deterioration of cerebral hemodynamics and led to an increase in the level of ischemic brain damage, as a result of which by the 2-3rd day the brain substance acquired a holey (honeycomb-like) character. In the first day from the onset of the disease, changes were determined, which we interpreted as reversible.

In addition, all deceased patients showed signs of chronic ischaemic encephalopathy due to slowly progressive diffuse insufficiency of blood supply to the brain tissue caused by atherosclerotic stenosis and occlusion of intracerebral and extracranial vessels. Reduced blood

flow in atherosclerosis of the main arteries was combined with changes in the microvascular bed. Microvessels showed wall thickening due to fibrosis, areas with cell proliferation of capillary walls and larger microvessels, as well as microvascular formations

with several (3-5) lumen (convolutes) as a compensatory reaction of the microvascular channel to the disconnection of its parts from the blood flow. The morphological equivalent of the manifestations of hypoxic dyscirculatory encephalopathy was also perivascular and pericellular edema, venous fullness, perivascular accumulations of leukocytes, pronounced dystrophic changes of neurons; leukostasis, swelling and desquamation of endothelium in small calibre brain vessels. Thus, the pathomorphological changes revealed in the deceased at different terms after ischaemic stroke confirm the concept of pathogenetic heterogeneity of ischaemic strokes. And the dynamics of their development testifies to the necessity of the earliest possible pathogenetically grounded treatment aimed at preventing the progression of perifocal damage and brain oedema.

Conclusions:

1. Ischaemic strokes are characterised by a wide variety of macroscopic and microscopic changes due to pathogenesis, localisation of the lesion and duration of the disease. Extensive and large ischaemic cerebral infarcts are pathogenetically associated with obturating atherothrombosis of one or several arterial branches, cardiac thromboembolism or arterio-arterial thromboembolism, medium-sized ones more often developed by haemodynamic mechanism, and the development of lacunar infarcts was associated with the lesion of perforating cerebral arteries.
2. The magnitude and localisation of cerebral infarction were influenced by the severity of hemodynamically significant atherostenosis or atherothrombosis, the rate of vessel stenosis or occlusion development, and the degree of collateral circulation development.
3. The decisive role in the genesis of hemispheric infarcts belongs to atherosclerosis of the main vessels of the head and heart pathology.
4. Stenotic lesions of the precerebral and intracerebral segments of vertebral arteries have a more important pathogenetic significance in strokes in the VBB.
5. The maximum severity of pathomorphological changes in ischaemic strokes is observed on the 2nd - 3rd day from the onset of the disease in the form of irreversible damage of all structural elements of nervous tissue in the area of the main ischaemic focus with the presence of perifocal zone characterized by pronounced perivascular and pericellular edema and reduction of the microcirculatory channel.

Literature:

1. Ilkhomovna, K. M., Eriyigitovich, I. S., & Kadyrovich, K. N. (2020). Morphological Features of microvascular Tissue of the Brain at hemorrhagic stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 2(10), 53-59.
2. Kadyrovich, K. N., Erkinovich, S. K., & Ilhomovna, K. M. (2021). Microscopic Examination Of Postcapillary Cerebral Venues In Hemorrhagic Stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 3(08), 69-73.
3. Камалова, М. И., & Хайдаров, Н. К. (2020). Prevention and risk factors for brain infarction (literature review). Журнал неврологии и нейрохирургических исследований, 1(2).
4. Ismoilov, O. I., Murodkosimov, S. M., Kamalova, M. I., Turaev, A. Y., & Mahmudova, S. K. (2021). The Spread Of SARS-Cov-2 Coronavirus In Uzbekistan And Current Response Measures. The American Journal of Medical Sciences and Pharmaceutical Research, 3(03), 45-50.

5. Shomurodov, K., Khaidarov, N., & Kamalova, M. (2021). The formation and eruption of baby teeth in children. Збгрник наукових праць SCIENTIA.
6. Khodjieva D. T., Khaydarova D. K., Khaydarov N. K. Complex evaluation of clinical and instrumental data for justification of optive treatment activites in patients with resistant forms of epilepsy //American Journal of Research. USA. – 2018. – №. 11-12. – С. 186-193.
7. Kamalova M. I., Khaidarov N. K., Islamov S. E. Pathomorphological Features of hemorrhagic brain strokes //Journal of Biomedicine and Practice. – 2020. – С. 101-105.
8. Khodjieva D. T. et al. Optimization of the diagnosis and treatment of early neurological complications in cardio embolic stroke //European Journal of Molecular & Clinical Medicine. – 2020. – Т. 7. – №. 07. – С. 2020.
9. Ходжаева Д. Т., Хайдарова Д. К., Хайдаров Н. К. Характеристика поражений проводящих путей при умеренно-когне-тивных расстройствах на фоне хронической ишемии мозга //Евразийский Союз Ученых. – 2015. – №. 7-3 (16). – С. 97-98.
10. Ilkhomovna K. M., Kadyrovich K. N., Eriyigitovich I. S. Clinical and demographicquality of life for patients with ischemic stroke in Uzbekistan //ACADEMICIA: An International Multidisciplinary Research Journal. – 2020. – Т. 10. – №. 10. – С. 883-889.
11. Ходжиева Д. Т., Хайдаров Н. К., Хайдарова Д. К. Коррекция астеноневротического синдрома энергокорректоромцитофлавином //Неврология.–Ташкент. – 2013. – №. 3. – С. 16-19.
12. Kamalova M., Khaidarov N. Assessment of quality of life in ischaemic stroke patients //Збірник наукових праць Scientia. – 2021.
13. Ходжиева Д. Т., Хайдарова Д. К., Хайдаров Н. К. Биофизические основы возникновения сирингомиелии: современные воззрения //Журнал неврологии и нейрохирургических исследований. – 2020. – Т. 1. – №. 3.
14. Ходжаева Д. Т., Хайдарова Д. К., Хайдаров Н. К. Совершенствование диагностической тактики постинсультных когнитивных расстройств //ORCID ID: 0000-0002-9309-3933 Масъул котиб Маматова Нодира Мухторовна–тиббиёт фанлари доктори, Тошкент давлат стоматология институти Фармакология кафедраси мудири. ORCID ID: 0000-0003-2396-4905. – 2019. – С. 80.
15. Kadirovna K. D., Kadirovich X. N., Tadjiyevna K. D. Clinical basis for the development of neuroprotective therapy in acute ischemic stroke //International Journal of Health Sciences. – 2022. – №. I. – С. 4177-4183.
16. Ходжиева Д. Т., Хайдаров Н. К. Аспект происхождения неврологических расстройств при болезни covid 19 //журнал неврологии и нейрохирургических исследований. – 2021. – №. Special 1.
17. lhomovna K. M., Kadyrovich K. N., Eryigitovich I. S. Patient Demographics in Haemorrhagic and Ischaemic Stroke //Central Asian Journal of Medical and Natural Science. – 2021. – Т. 2. – №. 4. – С. 40-42.
18. Kamalova M. I., Khaidarov N. K., Islamov S. E. Pathomorphological Changes in Cerebral Vasculature in Ischaemic Stroke //JournalNX. – 2021. – Т. 7. – №. 03. – С. 60-66.
19. Камалова М. И., Исламов Ш. Э., Хайдаров Н. К. Современные представления о факторах риска и профилактики инфаркта головного мозга (обзор литературы) //журнал неврологии и нейрохирургических исследований. – 2020. – Т. 1. – №. 3.
20. Камалова М. И., Хайдаров Н. К. Предупреждение и факторы риска инфаркта головного мозга (литературный обзор) //журнал неврологии и нейрохирургических исследований. – 2020. – Т. 1. – №. 2

21. Хайдаров Н. К. Встречаемость заболеваемости инсультом в бухарской области //Актуальные проблемы экспериментальной и клинической медицины. – 2019. – С. 92-92.
22. Хайдаров Н. К., Тешаев Ш. Ж., Камалова М. И. Risk factors and mechanisms of oncology in women (literature review) //журнал неврологии и нейрохирургических исследований. – 2023. – т. 4. – №.