SOME QUESTIONS OF ETIOLOGY AND PATHOGENESIS OF LACUNAR STROKES

[1]Majidova Yokuthon Nabievna, [2]Mirzaeva Nilufarkhon Bakhtiyor Kizi
[1]Professor. Doctor of Medical Sciences. Head of the Department of Neurology, Pediatric Neurology and Honey. genetics of the Tashkent Pediatric Medical Institute.
[2]Neuropathologist of RSCEMC
Republican Medical Emergency Science Center.
Tashkent. Uzbekistan
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Abstract. *Lacunar stroke is a marker of cerebral small vessel disease [15] and accounts for up to 25% of ischemic stroke cases. The word lacunar comes from the Latin "lacuna," meaning "islet," and is used to describe a small focus of encephalomalacia containing liquors, which is the end result of thinning necrosis. Lacunar stroke is defined as a subcortical infarction less than 20 mm in diameter caused by blockage of the intracranial artery perforator. [15].*

We examined 100 patients with lacunar ischemic stroke, admitted to the Republican Scientific Center for Emergency Medical Care from 2015 to 2023. Among the examined patients there were 44 men and 65 women aged 35 to 84 years, the average age was 61,2±12,2 years.

Keywords: *Lacunar stroke, small vessels, islet, focus of encephalomalacia, subcortical infarction.*

The frequency of lacunar stroke varies depending on the studied population from 25 to 50 cases per 100,000 people [11], which is 15-25% of cases of ischemic

Stroke [11, 10, 16]. These numbers, however, decline over time, probably due to better control of vascular risk factors such as hypertension [1]. Lacunar stroke has common risk factors with other stroke subtypes, namely hypertension, diabetes, older age, smoking, and hyperlipidemia [2, 17].

Atherosclerosis. There are several atherosclerotic mechanisms that can lead to lacunar stroke. The most important mechanism that has been pathologically proven is branch atheromatosis [3]. Atherosclerotic plaques of the maternal artery can affect the mouth of perforating branches, leading to occlusion and infarction of the distal parenchyma. This mechanism has been widely described in patients with narrowing of the lumen of the maternal artery, in which case lacunar stroke is classified as associated with intracranial atherosclerosis, as opposed to small vessel disease. On the other hand, some atherosclerotic plaques can cause material embolism of perforated arteries without significant luminal stenosis. This has been described, in particular, in patients with lacunar bridge stroke against the background of atherosclerotic involvement of the branches of the main artery, the common or internal carotid artery, as well as the aortic arch [5, 6, 18].

Cardioembolism. There are experimental data obtained in animal models showing that small emboli can penetrate perforated arteries and cause lacunar stroke [7]. However, current evidence suggests that the development of lacunar stroke against the background of atrial fibrillation or post-infarction cardiosclerosis is a very rare manifestation of cardioembolism [9, 13].

Computed tomography rarely detects a lacunar ischemic stroke within the first 24 hours because of its small size. Lacunar strokes, if seen on computed tomography, are poorly expressed hypodensitization unless there is a hemorrhagic component in acute stroke. Increased large artery density in non-contrast head computed tomography indicates the presence of a thrombus within the artery lumen or calcification of the vessel. Early signs of infarction in non-contrast computed tomography include loss of gray-white differentiation and focal hypoattenuation of the brain parenchyma. However, these details are difficult to distinguish by fine subcortical structures.

You can also do MSCT angiography of the vessels of the head and neck. This may indicate a filling defect associated with a thrombus blocking said vessel. It may also indicate narrowing of the arteries and extensive vascular damage, such as carotid arteries, which may be the source of embolus, or features of the middle cerebral artery characteristic of atherosclerotic plaque. The method of imaging extra- and intracranial arteries is essential for the diagnosis of possible major artery occlusion, as it helps to determine the need for intraarterial thrombolysis or thromboextraction.

MRI is an excellent neuroimaging method in acute and subacute states to detect LS. In the acute stage, the diffusion-weighted MRI image (DWI) has the greatest diagnostic accuracy. MRI-DVI helps to distinguish acute from chronic infarction [8]. Under high light conditions, in T1-weighted images, lacunes appear as focal regions of reduced signal intensity and as focal regions of increased intensity in T2-weighted images. Chronic lesions are isointensive to liquor in all sequences.

Ultrasound examination of the carotid arteries is a non-invasive and highly informative method for diagnosing atherosclerotic lesions of the extracranial carotid artery. Stroke risk is higher in patients with severe carotid artery stenosis.

Careful examination of the patient for embolism, including echocardiography and vascular imaging, gives a very low result in cases of lacunar strokes [14]. However, in some clinical cases, this may be necessary, for example, in young patients who have not previously experienced health problems.

It is also necessary to perform laboratory and instrumental tests such as ECG, blood glucose, complete blood count, including platelets, troponin, prothrombin time and international normalized ratio, activated partial thromboplastin time, blood lipid spectrum. These tests are necessary in identifying major risk factors for stroke.

Material and methods:

We examined 100 patients with lacunar ischemic stroke, admitted to the Republican Scientific Center for Emergency Medical Care from 2015 to 2023. Among the examined patients there were 44 men and 65 women aged 35 to 84 years, the average age was 61,2±12,2 years. Patients were divided into 2 groups:

Group I - patients with LI who did not show morphological sources of cerebral embolism - 79 patients (79%);

Group II - patients with LI who had revealed morphological sources of cerebral embolism - 21 patients (21%).

Group I patients were divided into two subgroups:

Ia - patients with LI with a long history of hypertension - 44 patients;

Ib - patients with LI with gross atherosclerotic damage to cerebral arteries - 35 patients.

Group II - patients with embolic genesis of LS.

All patients had:

1. Assessment of risk factors for stroke;
2. MSCT of the brain;
3. brain MRI;
4. Transthoracic echocardiography;
5. Color duplex scanning of the extra- and intracranial arteries;
6. Transcranial Doppler;
7. Laboratory diagnostics of biochemical parameters and blood coagulation system;
8. NIHSS assessment of neurological deficits.

Study results.

The main clinical manifestations of LS were the following symptom complexes:

1. Pyramidal disturbances;
2. Sensitive disorders;
3. Speech disorders by type of different types of aphasias;
4. Cerebellar-discoordinatory disorders;

The most common symptom was pyramidal disorders in the form of unilateral hemiparesis: in group I - in 48 patients (60.7%), in II - in 15 patients (71.4%), there were no statistically significant differences in the detection of isolated hemiparesis between the groups ($p > 0.05$).

Purely "sensitive" disorders in LS were rare, in group I - in 5 patients (6.3%), in II - in 1 patient (4.8%). The LS, which manifested only speech disorders, was detected in group I - in 6 patients (7.6%), in II - in 2 patients (9.5%). Cerebellar-discoordant manifestations were diagnosed in group I - in 8 patients (10.1%), in II - in 2 patients (9.5%). There were also no statistically significant differences between the groups.

The severity of neurological deficits in the acute LS period was assessed on the NIHSS scale. At the onset of the disease, the severity of neurological deficiency was higher in the II group of patients with LS, who identified potential sources of cerebral embolism (8,8±2,9 points) compared to the I group (4,7±2,2 points), while there were statistically significant differences between the groups ($p < 0.05$).

The severity of neurological deficiency on day 10 from the start of the LS clinic in group II compared to the first day of the disease was significantly lower and amounted to 4,6±2,2 ($p < 0.01$). In group I, the severity of neurological symptoms on day 10 compared to the first day of stroke also significantly decreased - 3,1±1,9 ($p < 0.01$). However, the severity of neurological deficiency in the group of patients with verified sources of cerebral embolism remained higher on day 10 than in patients with LS due to cerebral microangiopathy, the inter-group difference had a level of statistically significant differences ($p < 0.01$).

Table 1. Main sonographic characteristics of atherosclerotic plaque depending on the nature of carotid lesion.

№	Characteristics of ASCP	Number of patients	%
1.	Homogeneous hypoechogenic plaque	1	1
2.	Homogeneous hyperechoic plaque	2	2

3.	Heterogeneous, predominantly hypoechogenic plaque	2	2
4.	Heterogeneous, predominantly hyperechogenic plaque	45	45
5.	Calcined plaque	3	3
6.	ASCP with ulceration	15	15
7.	Unclassifiable plaque	0	0
8.	It is not possible to determine the nature of ASCP due to vessel occlusion	5	5
9.	No plaques, only hemodynamically significant bending	7	7
10.	Thickening of intima-medium complex	21	21
	Total	**100**	**100%**

No stable plaque
Stable plaque

Hemodynamically significant atherosclerotic lesion of brachiocephalic arteries (stenosis > 60%) was verified in 31 patients with LS, in the IB subgroup of patients with atherosclerotic microangiopathy - in 13 patients (37.1%), in the group of patients with embolic gene LS - in 18 (85.7%), and the differences did not reach the level of statistical significance ($p > 0.05$) It should be noted that in the IB subgroup, 5 patients (14.3%) had carotid occlusion, the remaining 22 patients had carotid artery stenosis on the ipsilateral side of the affected basin and the remaining 8 patients on the contralateral side of the affected basin. In group II, the stenosing lesion of the carotid arteries in all patients was located ipsilateral to the affected hemisphere. In the subgroup of patients with LS due to hypertensive microangiopathy, no gross wall-occlusive lesions were observed, hemodynamically insignificant extracranial artery stenoses were diagnosed **(Table 1).**

Table 2. Main sonographic characteristics of atherosclerotic plaque in patients with LS.

№	Characteristics of ASCP	Number of patients	%
1.	Homogeneous hypoechogenic plaque	1	1
2.	Homogeneous hyperechoic plaque	2	2
3.	Heterogeneous, predominantly hypoechogenic plaque	2	2
4.	Heterogeneous, predominantly hyperechogenic plaque	45	45
5.	Calcined plaque	3	3
6.	ASCP with ulceration	15	15
7.	Unclassifiable plaque	0	0
8.	It is not possible to determine the nature of ASCP due to vessel occlusion	5	5
9.	No plaques, only hemodynamically significant bending	7	7
10.	Thickening of intima-medium complex	21	21
	Total	**100**	**100**

No stable plaque
Stable plaque

When analyzing the morphological structure of atherosclerotic plaque, attention was paid to its "stability/instability." Such Doppler features of non-stable plaque as homogeneous hypoechoic plaque are identified in 1 case; heterogeneous, predominantly hypoechogenic plaque occurred in 2 patients, calcified plaque was detected in 3 patients; atherosclerotic plaque with tire ulceration was diagnosed in 15 patients **(Table 2).**

Potential sources of cardioembolism were identified in 11 patients. The following sources of cardioembolism were identified: left atrial auricle (with atrial fibrillation), local pathology of myocardial wall movement, left ventricular aneurysm, damage to the mitral or aortic valves of the heart. All sources of cadiogenic embolism were verified in patients of group II.

Conclusion. Thus, today it is known that the morphological substrate of LI is microangiopathy of the perforated arteries. In our study, the proportion of patients in whom lacunar infarction developed due to "small vessel disease" was 78.9%. The main causes of perforated arteries damage are lipogialinosis (replacement of muscle and elastic membranes with collagen and generalized subintimal collagen deposition) against the background of hypertension [4] and microateromatosis of perforated arteries due to generalized atherosclerotic process [12]. The correct determination of the etiopathogenetic cause of the development of LI is fundamental, since it will allow you to choose the correct further tactics in the prevention of repeated stroke.

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