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SOME QUESTIONS OF ETIOLOGY AND PATHOGENESIS OF LACUNAR STROKES

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Lacunar stroke is a marker of cerebral small vessel disease and accounts for up to 25% of ischemic stroke cases. Lacunar stroke is defined as a subcortical infarction less than 20 mm in diameter caused by blockage of the intracranial artery perforator (Wardlaw J.M., Smith E.E., Biessels G.J. (2013).

Acute focal symptoms require urgent neuroimaging. Non-contrast computed tomography of the head is preferred in acute situations as it is readily available, fast and informative to rule out life-threatening conditions such as intracerebral hemorrhage. MRI is an excellent neuroimaging method in acute and subacute states to detect LR. (Norrving B.2015). Ultrasound examination of the carotid arteries is a non-invasive and highly informative method for diagnosing atherosclerotic lesions of the extracranial carotid artery.

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 LR who did not show morphological sources of cerebral embolism - 79 patients (79%);

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

Group I patients were divided into two subgroups:

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

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

Study results. The main clinical manifestations of LR 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 LR were rare, in group I - in 5 patients (6.3%), in II - in 1 patient (4.8%). The LR, 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 LR 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 LR, 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 ($p < 0.05$).

Hemodynamically significant atherosclerotic lesion of brachiocephalic arteries (stenosis > 60%) was verified in 31 patients with LR, in the I-B subgroup of patients with atherosclerotic microangiopathy - in 13 patients (37.1%), in the group of patients with embolic gene LR - in 18 (85.7%) ($p > 0.05$). It should be noted that in the I-B 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.

Conclusion. Thus, today it is known that the morphological substrate of LR 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%.