TRANSITOR ISCHEMIC ATTACK

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Abstract: A transient ischemic attack is a transient episode of neurological dysfunction caused by focal ischemia of the brain or spinal cord or retinal ischemia without acute infarction." That is, the definition of the concept was based only on morphological criteria. Symptoms of carotid TIA: — sudden monocular blindness (amaurosis) or decreased vision (amblyopia) on the side of the stenosed internal carotid artery; the duration of the episode is usually several minutes; — opto-pyramidal syndrome — monocular amaurosis or amblyopia is accompanied by weakness and sometimes numbness in the opposite extremities;

Key words: TIA, blood pressure, Cardioembolism, ischemia.

Ideas about transient ischemic attack (TIA) as an independent nosological form of acute vascular pathology of the brain began to form in the 50s of the twentieth century. TIA was defined as focal cerebral ischemia with symptoms lasting up to 24 hours. Studies of the last decade have shown that this arbitrarily chosen time threshold is too wide, since in 30-50% of patients with a typical TIA clinic, magnetic resonance imaging (MRI) reveals signs of focal brain damage. Since 2002, TIA has been interpreted as a short-term episode of neurological dysfunction caused by focal cerebral or retinal ischemia with clinical symptoms lasting more than an hour, without obvious signs of acute cerebral infarction. TIA is a transient episode of neurological dysfunction caused by focal ischemia of the brain or spinal cord or retinal ischemia without acute infarction" (2008). That is, the definition of the concept was based only on morphological criteria. The prevalence of TIA cannot be accurately accounted for due to many factors, primarily due to differences in the criteria used in epidemiological studies. In one of the examinations of patients with classical TIA, it was shown that MRI leads to a refutation of the diagnosis in about 30% of cases, increasing the incidence of ischemic stroke (AI) by about 7%. In Western European countries, the incidence of TIA is on average 50 per 100 thousand population; in Ukraine in 2009 — 92.9 per 100 thousand population. The frequency of TIA in the USA is from 200 to 500 thousand cases per year with a frequency of 2.3% in the population, which is about 5 million people. The prevalence varies depending on age. According to the Cardiovascular Health Study, the incidence of TIA among men aged 65-69 years was 2.7% (1.6% among women), in the category of 75-79 years — 3.6 and 4.1%, respectively. According to the study, the prevalence of TIA among adults 45-64 years was 0.4%, and among people who have undergone AI or TIA, — 7-40%. This indicator varies depending on the type of study (hospital or population), as well as on the very definition of TIA (time factor). Epidemiological studies in recent years have shown a high risk of stroke immediately after TIA. In 10-15% of patients, AI can develop within 48 hours after TIA, in 10% — within 3 months, in 20% — within a year. In this regard, TIA, like stroke, is recognized as an urgent condition (ICD-10: G45.0-G45.9). Etiology of TIA: — systemic cardiogenic

embolisms that occur in the presence of atrial fibrillation (MA), cardiac thrombi, dilated cardiomyopathy, myocardial infarction, artificial heart valves, bacterial and non-bacterial myocarditis; — atherosclerotic and atherothrombotic stenoses > 70% of extracranial sections of the main arteries of the head (MAG) and large arteries of the base of the brain; — arterio-arterial embolisms; — less frequent causes include vasculitis and arteritis in the structure of collagenoses, dissection of MAG as a result of trauma; — anomalies of MAG development (inflection, hypo- and aplasia), coarctation of the aorta; — extravasal compression of vertebral arteries in cervical osteochondrosis. The pathogenesis of TIA is based on reversible local cerebral ischemia, which develops with a decrease in cerebral perfusion of less than 18-22 ml per 100 g/min, which is the functional threshold of ischemia. A transient drop in blood flow in the area distal to the place of occlusion of the artery leads to the appearance of focal symptoms. With the restoration of blood flow, there is a regression of focal symptoms and the completion of a vascular episode. In case of a further drop in perfusion below the threshold of reversible changes (8-10 ml per 100 g/min), a brain infarction is formed. Just like AI, TIA can develop by various mechanisms, depending on the etiological factor: — more often by the mechanism of cardioembolism, in which embolized material is thrown from the heart; less often by the atherothrombotic mechanism, in which the formation of a blood clot in the area of atherosclerotic plaque leads to narrowing of the vessel lumen; — with arterio-arterial embolism a thrombus is detached from the surface of ulcerated atherosclerotic plaques in the area of stenosing lesions of the arteries; — hemodynamic TIA occur due to a sharp drop in systemic arterial pressure (BP) with stenoses of more than 70%, which leads to the development of cerebral hypoperfusion. A drop in blood pressure can develop for a number of reasons: cardiac arrhythmia, myocardial infarction, orthostatic hypotension, hypovolemia, a decrease in the minute volume of the heart, an overdose of hypotensive drugs, deep sleep, hyperventilation, physical exertion, cough. The mechanism of vasospasm is currently controversial. TIA in the carotid basin proceeds with hemispheric symptoms, in the vertebrobasilar — with stem-cerebellar. Symptoms of carotid TIA: — sudden monocular blindness (amaurosis) or decreased vision (amblyopia) on the side of the stenosed internal carotid artery; the duration of the episode is usually several minutes; — opto-pyramidal syndrome — monocular amaurosis or amblyopia is accompanied by weakness and sometimes numbness in opposite extremities; — brachiofacial paresis with predominantly cortical localization of TIA: mild paresis of the mandibular musculature and weakness and/or numbness of the hand contralaterally; — with a stenosing process in the left internal carotid artery in right—handed people — rough short-term speech impairment - elements of aphasia (cortical dysphasia); — much less often — short-term focal clonic convulsions in the contralateral extremitiesSymptoms of vertebrobasilar TIA: — systemic dizziness, positionally increasing, accompanied by vegetative disorders (nausea, vomiting, hyperhidrosis), ataxia; — diplopia; — mild dysarthria, elements of dysphagia, hypophonia; — photopsias (metamorphopsias) in both eyes; — transient amnesia; — nystagmus; — cerebellar ataxia. Factors provoking stem-cerebellar TIA are a sharp tilting or turning of the head. This is typical for patients with cervical osteochondrosis and vertebral artery stenosis. Such TIA can be accompanied by a sudden fall without turning off consciousness, convulsions and enuresis — these are the so-called drop-attaks, the occurrence of which is explained by transient ischemia of the reticular formation with subsequent shutdown of postural tone. Rarely, stem TIAs are provoked by stereotypical movements with raised hands due to non-

physiological blood flow from the vertebral artery to the subclavian artery with its stenosis. It is not typical for TIA in the vertebrobasilar pool when the symptoms are limited to only one symptom: — isolated dizziness; — short—term loss of consciousness; — falling; - transient darkening in the eyes. The paroxysmality of TIA makes it necessary to differentiate this pathology: — with convulsive epipripadki. EEG in all TIA (with the exception of cortical ischemia with focal seizures) does not reveal epiactivity; — short-term hypoglycemia conditions, especially characteristic of insulomas. The clinical picture may resemble TIA in the vertebrobasilar or carotid basin, especially in patients with dyscirculatory encephalopathy (DEP), but an emergency study of blood sugar levels and glucose correction lead to complete regression of symptoms; — dissection of the carotid or vertebral artery — an infrequent but serious reason for differentiation with TIA. In favor of this pathology — indications of a neck injury or an unusually sharp turn of the head. Unfortunately, this may be a consequence of manual therapy; — associated migraine, which may occur with a clinic almost identical to carotid TIA. Such patients (and these are mainly women of fertile age) should be examined Dopplerographically to study the state of the MAG. Currently, MRI is recognized as the most adequate method of instrumental diagnosis, which is performed on the first day of admission of a patient with TIA to a specialized hospital. Already in the first hours of hospitalization, differential diagnosis of TIA and AI is possible, which can determine the volume of therapy. 2. Magnetic resonance angiography is the most informative. 3. Ultrasound dopplerography of the extra- and intracranial segments of the MAG allows to determine the degree of their atherosclerotic lesion from initial manifestations to critical subtotal stenoses. 4. EEG is used in the diagnosis of cortical TIA occurring by the type of focal seizures. 5. ECG and EchoCG are especially valuable if non-cardiac causes of TIA are excluded. Transesophageal echocardiography is more informative than transthoracic in determining the pathology of the atrial septum (septal aneurysm, open oval window, etc.), atrial thrombi and valve diseases. 6. When detecting pathological changes on ECG and EchoCG, it is advisable to conduct Holter monitoring of patients. 7. Laboratory tests for TIA are the same as for AI: clinical and biochemical blood tests, coagulogram. These studies make it possible to exclude hypoglycemia and help identify the cause of thrombosis (true polycythemia). Young patients who do not have obvious vascular risk factors should undergo specialized tests of the coagulation system: — protein C, protein S, antithrombin III; — activated protein C, factor V (Leiden); — fibrinogen; — D-dimer; — anticardiolipin antibodies; — lupus anticoagulant; — homocysteine; — factor VII; — Willebrand factor; — inhibitor of plasminogen activator 1; — activity of endogenous tissue plasminogen activator. Diagnostic algorithm: — examination by a neurologist — in-depth study of complaints, anamnesis, somatic and neurological status; — angioeducational examination — palpation and auscultation of the common and internal carotid arteries, palpation of the temporal arteries; — neuroimaging on MRI to exclude AI, preferably on the first day of the disease; — ECG, EchoCG; — consultation of a therapist, cardiologist; — ultrasound examination of the extracranial parts of the MAG; — examination of peripheral arteries (rheovasography); — laboratory tests (see above). Emergency pathogenetic therapy of TIA at the hospital stage in the conditions of the neurovascular department should be carried out in the scope of AI therapy, especially on the first day of the disease, when in most cases both clinical and paraclinical (MRI) diagnostics do not yet provide convincing evidence in favor of one or another option. The duration of infusion therapy can reach several days, depending on the background pathology (DEP, CHD, etc.). Basic therapy

and diagnostic monitoring: — correction of blood pressure — it is desirable to maintain the level of mild hypertension 160-180/90– 100 mmHg., to prevent a significant decrease in blood pressure; — regularly monitor blood glucose levels, with hyperglycemia above 10 mmol / l — consultation of an endocrinologist regarding insulin therapy; — constant monitoring of the water-electrolyte balance in order to avoid an increase in hematocrit and a decrease in rheological properties of blood; — electrocardiography — if arrhythmia is detected in some cases — daily monitoring and consultation of a cardiologist. To improve the rheological properties of blood and microcirculation, prescribe: — antiplatelet agents — aspirin at a dose of 325 mg / day for 48 hours with a transition to 100 mg / day; it is possible to combine with clopidogrel (75 mg) or dipyridamole (50 mg 2 times a day); — low molecular weight dextrans: reopoliglyukin, reomacrodex 200-400 ml / day; — pentoxifylline 200 mg intravenously 1-2 times a day; — anticoagulants: with diagnosed stenosis of MAG with frequent repeated TIA and with MA — low molecular weight heparins (fraxiparin, kleksan, etc.) under the control of the international normalized ratio (INR). Due to the delayed (in most cases) hospitalization of patients with TIA, neuroprotective therapy should be carried out simultaneously with both primary and secondary neuroprotectors in full, as with AI, since on the first day it is most often impossible to differentiate these clinical forms with MRI. It is recommended: — citicoline (ceraxone) intravenously at a dose of 500 mg 1-2 times a day, possibly in combination with actovegin 10-20% r-r 250 ml intravenously; — magnesium sulfate — 10-20 ml 25% r-ra on the physical intravenously; — antioxidants: mexidol 200-500 ml, or reamberin 400-800 ml, or cytoflavin 10 ml intravenously; — nootropics — cerebrolysin 10-20 ml or piracetam 5-6 g intravenously. After the final confirmation of the diagnosis of TIA, the dosages of the drugs may be revised. During the first days of the stay of patients with TIA in the neurovascular department, a number of issues should be resolved: — selection of patients for carotid endarterectomy (after Dopplerography and consultation with a vascular surgeon); — administration of tableted anticoagulants — warfarin to patients with MA after consultation with a cardiologist; — control of the risk of hyperinsulinemia during treatment with warfarin — determination of INR, which should remain within 2.0–3.0 (goal — 2.5); — administration of statins in case of hypercholesterolemia. According to the results of recent studies, carotid artery surgery is not recommended for significant asymptomatic stenosis (70-99%), with the exception of patients with a high risk of TIA and AI due to plaque instability; in all other cases, conservative therapy remains preferable. However, with repeated TIA in the ICA basin with a high degree of stenosis, carotid angioplasty with or without stent placement is indicated. The secondary prevention system is based on a high-risk strategy, which is determined primarily by significant and correctable risk factors for the development of TIA (AI) and the choice of therapeutic approaches in accordance with the nature of the acute cerebral circulatory disorder suffered, based on data from multicenter randomized clinical trials. Risk factors for TIA (II): arterial hypertension; coronary heart disease; type I and II diabetes mellitus; hypercholesterolemia; asymptomatic carotid stenosis more than 70%; smoking (more than 15 cigarettes a day). Patients who have undergone TIA on the background of cerebral atherosclerosis, arterial hypertension or cardiac pathology need non—drug methods of secondary prevention: - quitting smoking or reducing the number of cigarettes smoked; — refusal from alcohol abuse; — hypocholesterol diet; — reduction of excess body weight; — increase in physical activity to the level of 45-60 minutes of moderate intensity exercise or

walking from 30 to 60 minutes several times a week. For the purpose of secondary prevention of TIA (II), it is necessary to maintain blood glucose at a level not higher than 7 mmol / l (oral hypoglycemic agents or the appointment of insulin). Most patients with TIA are prescribed low doses of aspirin (75-100 mg / day), and this therapy should be continued for life.) Acetylsalicylic Acid (ASA) Aspirin (acetylsalicylic acid) at a dose of 75-100 mg should be used for the prevention of recurrent stroke (P-1) ASA + slow-release dipyridamole (agrenox) A fixed combination of ASA and slow-release dipyridamole (50 mg / 200 mg twice a day) is prescribed as the drug of first choice for the prevention of recurrent stroke (P-1) Clopidogrel is somewhat more effective than ASA in preventing recurrent stroke (P-1). It can also be prescribed in case of intolerance to ASA and dipyridamole (P-4) and in high-risk patients (P-3). Patients who have started therapy with thienopyridine derivatives should receive clopidogrel instead of ticlopidine due to the serious side effects of the latter (P-3) Clopidogrel + ASA Patients who have undergone TIA (II) with concomitant unstable angina should receive a combination of clopidogrel 75 mg and ASA 75 mg twice a day (P-3), however at the same time, the risk of bleeding may increase For secondary prevention of stroke in patients with hypertension who have suffered an acute cerebral catastrophe — TIA (II), combined hypotensive therapy with the use of a diuretic and one or two representatives of the main modern groups of antihypertensive agents (beta-adrenoblocker + ACE inhibitor, beta-adrenoblocker + Ca2+ antagonist, etc.) is recommended. These recommendations should be treated with caution in cases of high degree of carotid artery stenosis, especially if it is bilateral, since an excessive decrease in blood pressure can lead to hypoperfusion in the areas of adjacent blood circulation. Hypotensive therapy in this group of patients should be carried out under constant medical supervision. From the modern point of view, early administration of anticoagulants in acute cerebral ischemia is not very desirable due to the high risk of hemorrhagic complications. Unfortunately, currently only a small part of patients who have undergone TIA receive adequate therapy in the structure of secondary prevention. Improving organizational measures for the dispensary management of patients who have undergone TIA seems to be a promising direction in solving this urgent problem. Prevention work is an interdisciplinary problem, so its success is determined by the interaction of an outpatient therapist, a family doctor, a general practitioner, a neurologist, a neurosurgeon, an optometrist, etc.

References:

1. Vertkin A.L. Neuroprotective therapy in the acute period of stroke: a step forward. neurole. magazine. — 2007. — № 4(14). — Pp. 53-58.
2. Kremetz K.G. www. stroke. ahajournals. org.
3. Likhachev S.A., Astapenko A.V., Belyavsky I.N. Transient ischemic attacks: etiology, pathogenesis, classification, clinic, diagnosis // Moscow news. — 2003. — № 10.
4. Mishchenko T.S. New European guidelines for the treatment and prevention of stroke and transient ischemic attacks: how much is new in them? // Health of Ukraine. - 2008. — No. 12/1. — P. 15.
5. Mishchenko T.S. // News of medicine and pharmacy. Thematic issue "Neurology". — 2010. — No. 339.

6. Merkulova G.P. Primary and secondary prevention of stroke // Emergency Medicine: Selected wedge. lectures. — Kharkiv, 2008. — pp. 236-249.
7. Pokrovsky A.V. Surgical treatment for atherosclerotic stenosis of the internal carotid artery as prevention of stroke // Journal. neurol. and a psychiatrist named after S.S. Korsakov. Adj. Stroke. — 2007. — Special Issue. — pp. 32-33.
8. Cherniy V.I., Ostrovaya T.V., Andronova I.A. On the expediency of the combined use of neuroprotectors in acute cerebral insufficiency of various etiologies // Ukrainian Neurological Journal — 2008. — No. 1. — pp. 48-56.
9. Yavorskaya V., Flomin Yu., Dyologa N., Grebenyuk A. Transient ischemic attacks and stroke, what we already know and what we have to learn // Likes of Ukraine. - 2004. — No. 9. — pp. 72-79.10. Salomova, N. K. (2022). Risk factors for recurrent stroke. Polish journal of science N, 52, 33-35.
11. Kakhorovna, S. N. (2022). Features of neurorehabilitation itself depending on the pathogenetic course of repeated strokes, localization of the stroke focus and the structure of neurological deficit.
12. Qahharovna, S. N. (2023). Thromboocclusive Lesions of the Bronchocephalic Arteries: Treatment Options and Phytotherapy Options. AMERICAN JOURNAL OF SCIENCE AND LEARNING FOR DEVELOPMENT, 2(2), 41-46.
13. Salomova, N. K. (2022). Risk factors for recurrent stroke. Polish journal of science N, 52, 33-35.
14. Salomova, N. K. (2023). KAITA ISCHEMIC INSULTLARNING CLINIC POTOGENITIC HUSUSIYATLARINI ANIKLASH. Innovations in Technology and Science Education, 2(8), 1255-1264.
15. Salomova, N. K. (2021). Features of the course and clinical and pathogenetic characteristics of primary and recurrent strokes. Central Asian Journal of Medical and Natural Science, 249-253.
16. Rakhmatova, S. N., & Salomova, N. K. (2021). Kaita Takrorlanuvchi Ischemic Va Hemorrhagic Stroke Bemorlarni Erta Rehabilitation Kilishni Optimallashtirish. Journal Of Neurology And Neurosurgical Research, 2(4).
17. Salomova, N. K. (2022). RISK FACTORS FOR CEREBROVASCULAR DISEASE AND THE BENEFICIAL PROPERTY OF UNABI IN PREVENTION. Oriental renaissance: Innovative, educational, natural and social sciences, 2(2), 811-817.
18. Akhrorova, S. B. (2021). Prevalence and neurological features of diabetic polyneuropathy in type 1 diabetes. Journal of Neurology and Neurosurgical Research, 2(2).
19. Akhrorova, Sh. B. (2021). Diabetic polyneuropathy in type I diabetes mellitus. JOURNAL OF NEUROLOGY AND NEUROSURGICAL RESEARCH, (SPECIAL 1).20. Ay H., Koroshetz W. et al. Transient ischemic attack with infarction: Aunigie Syndrome? // Ann. Neurol. — 2005. — Vol. 57, № 5. — P. 679-686.
21. Wu C.M., Melaughlin K., Lorenzeitti D.L. et al. Early risk of stroke after transient ischemic attack // Arch. Intern. Med. — 2007. — Vol. 167(22). — P. 2417-2422. Получено 16.02.
22. Rakhmatova, D. I. (2022). The effectiveness of neurotrophic therapy of serotonin in ischemic stroke. Journal of Neurology and Neurosurgical Research, 3(1).
23. Rakhmatova, D. I. (2020). Opportunities of acupuncture in treatment of facial nerve neuropathy. European Journal of Molecular and Clinical Medicine, 7(7), 567-572.

24. Gaffarova, V. F. (2021). Clinic-eeg correlation somatogenous of conditioned febrile seizures in children. International Journal of Human Computing Studies, 3(1), 114-116.
25. Furqatovna, G. V. (2023). Evaluate the Neuropsychological, Clinical-Neurological and Neurophysiological Characteristics of Febrile and Afebrile Seizures. AMERICAN JOURNAL OF SCIENCE AND LEARNING FOR DEVELOPMENT, 2(2), 187-192.
26. Zavkiddinovna, D. H. (2023). Features of the Course of Cognitive Dysfunction in Patients with Type II Diabetes Mellitus. AMERICAN JOURNAL OF SCIENCE AND LEARNING FOR DEVELOPMENT, 2(3), 53-55.
27. Davronova, H. (2023). COGNITIVE DISORDERS IN TYPE 2 DIABETES MELLITUS. International Bulletin of Applied Science and Technology, 3(5), 901-906.