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# Management Tactics and Antiplatelet Therapy for Primary Prevention of Transient Ischemic Attack and Cerebral Infarction

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***Abstract:*** *Despite the significant successes achieved in the treatment and prevention of acute cerebral circulatory disorders (ONMC), strokes remain one of the main causes of mortality and the leading cause of disability in Uzbekistan. More than 60 thousand cases of stroke (acute cerebrovascular accident) are registered annually in Uzbekistan. At the same time, disability after stroke is 83.8%, and the percentage of hospital mortality is 17.3%.*

***Keywords:*** *TIA, ischemic stroke, NAID, prevention.*

In the regions of Uzbekistan, difficulties remain in organizing care for patients with transient ischemic attacks (TIA). Due to the short duration of the existence of symptoms in most cases, patients do not attach importance to them, do not seek medical help, so the registration of TIA remains incomplete. Meanwhile, timely recognition of TIA is extremely important, because it allows you to provide effective assistance aimed at preventing the development of gross motor, speech and other disorders depending on the location of the lesion, as well as to carry out secondary prevention of stroke. TIA, according to the clinical classification developed by E.V. Schmidt, adopted in the Russian Federation, is a variant of transient cerebrovascular accident (PNMC) [10]. TIA is traditionally defined as an acute cerebrovascular accident (ONMC) with a short-term (not exceeding 24 hours) violation of brain functions in the form of focal and/or cerebral symptoms, followed by complete regression of symptoms and the absence of signs of cerebral infarction according to neuroimaging data [2, 3, 5, 8]. Do not forget that TIA is a critical condition, which is clinically impossible to distinguish from a cerebral infarction before the symptoms pass (or before the expiration of 24 hours). It is known that the risk of ischemic stroke after TIA increases by 4-10% in the first two days, and by 10-20% in the next 3 months. In the presence of extracranial carotid artery stenosis exceeding 70% of the diameter, the risk of stroke in the first 2 years after TIA reaches 28%. Thus, TIA can be considered a predictor of stroke. Usually TIA precede atherothrombotic stroke (up to 50% of cases), less often – cardioembolic (in 10% of cases) or lacunar (in 20% of cases), other patients develop hemorrhagic stroke The most significant risk factors for TIA are arterial hypertension, hypercholesterolemia, diabetes mellitus, smoking, excessive alcohol consumption, overweight, sedentary lifestyle. The most common etiological factors of TIA are [1, 4, 8, 9, 12]: atherosclerosis of the carotid, vertebral arteries and large branches of the basin of these arteries, which leads to arterio-arterial embolism, and symptomatic carotid stenosis of more than 50% of the artery lumen, leading to the development of hemodynamic insufficiency; angiopathy, developmental abnormalities (inflection, doubling, hypo- or aplasia) of the carotid or vertebral arteries, coarctation of the aorta; cardiogenic embolism; coagulopathy; extravasal compression of the vertebral arteries by pathologically altered cervical vertebrae; dissection of the carotid or vertebral artery; use of sympathomimetics. In more rare cases, TIA may be caused by vasculitis, including specific blood diseases, antiphospholipid syndrome, venous thrombosis, migraine, oral contraceptives or other causes. The development of TIA is caused by an acute, but reversible (without the development of a

heart attack) critical decrease in blood supply to a part of the brain in a certain arterial basin. The key point in the pathogenesis of TIA is precisely reversible local cerebral ischemia, which develops with a decrease in cerebral perfusion below 18-22 ml per 100 g/min. (with a norm of 50-60 ml per 100 g/min.), which is the functional threshold of ischemia. A transient drop in blood flow in the area distal to the site of arterial occlusion and the development of ischemia in the foci of brain tissue corresponding to the affected arteries leads to the appearance of reversible focal symptoms. In the case of restoration of cerebral perfusion, a regression of focal symptoms and the completion of the TIA episode is clinically observed. In case of a further drop in perfusion below the threshold of irreversible changes (8-10 ml per 100 g / min.), a brain infarction develops. In general, the mechanisms of TIA development are similar to the pathogenesis of ischemic stroke. The clinical outcome of ischemic type ONMC (TIA or cerebral infarction) mainly depends on the localization of the lesion and the rate of development of the pathological process, the state of collateral circulation and rheological properties of blood [2, 3, 6, 11]. Clinically, TIA is manifested by transient symptoms of cerebral ischemia, depending on the localization of the pathological process, such as: loss of vision in one or both eyes, paresis in the arm, leg or facial muscles, impaired sensitivity in the arm, leg and facial muscles, impaired balance and coordination of movements, speech impairment, loss of consciousness, memory impairment, psychomotor arousal, violation of behavior. The average duration of a TIA episode is 8-14 min., most TIAs are resolved within 1 hour or earlier. According to V.A. Parfenova, the duration of TIA symptoms less than 1 hour was noted in 43.5%, from 1 to 3 hours – in 45.7%, more than 3 hours – in 10.9% of patients. TIA occur in the carotid basin almost 4 times more often than in the vertebrobasilar [7]. In most cases, the diagnosis of TIA is determined retrospectively, because at the time of examination of the patient by a neurologist, his focal symptoms regress spontaneously [13]. In this regard, it is necessary to carefully collect anamnesis and knowledge of the clinical manifestations of TIA by doctors of various profiles. TIA can be repeated often or develop once. Verification of the diagnosis of TIA is based on: assessment of the clinical picture, determination of the etiology of TIA using echocardiography (EchoCG) or duplex scanning of the vessels of the neck and brain, if necessary, angiographic examination, exclusion of cerebral infarction using neuroimaging methods (CT or MRI), which must be carried out even with complete regression of symptoms. The main objectives of the diagnosis of TIA are the exclusion of cerebral infarction in the first 3 hours from the development of TIA, the exclusion of other diseases that have a clinical picture similar to TIA, the determination of the etiology of TIA (differentiation of embolic and non-embolic TIA, TIA with carotid and vertebral stenosis) in order to timely begin targeted prevention of cerebral infarction. Computed tomography (CT) or magnetic resonance imaging (MRI) of the head is indicated for all patients who, based on the clinical picture, have a suspicion of TIA. If the patient is hospitalized within 1-6 hours after TIA, CT or MRI should be used to exclude a cerebral infarction, as well as a subdural hematoma or brain tumor. Duplex scanning makes it possible to diagnose various pathological vascular processes based on direct echographic signs, including atherosclerosis, vasculitis, angiopathies, vascular anomalies, aneurysms, etc. The advantages of DS include the possibility of detecting early preclinical signs of the disease, assessing changes in hemodynamics in real time with the detection of not only organic, but also functional disorders of blood flow. Magnetic resonance angiography (MRA) or CT angiography is indicated when DS does not give a reliable result. Carotid angiography is a standard diagnostic procedure before performing carotid endarterectomy. It is also indicated for patients with TIA in the event that DS and MRI (CT angiography) give contradictory results, or if their implementation is impossible. EchoCG is indicated when the cardioembolic mechanism of TIA is suspected in cases where the data of anamnesis and / or objective examination indicate the possibility of cardiological pathology when the patient's age exceeds 45 years, as well as when the results of a study of the vessels of the neck, brain and blood tests did not reveal the cause of TIA. In addition, the examination plan for TIA, as a rule, should include an ECG, a general blood test, determination of plasma electrolytes, creatinine, hematocrit, blood viscosity, prothrombin time, international normalized ratio (INR), serum osmolarity, fibrinogen, platelet and erythrocyte aggregation, glucose and blood lipids and antiphospholipid antibodies. The basic principles of management of patients with TIA before the end of the episode of PNMC do not differ from the tactics of management of a patient with a brain

infarction. If the symptoms associated with TIA persist for several hours and the patient has time to seek help, then he is subject to hospitalization in a specialized department for patients with ONMC for emergency indications in order to conduct differential diagnosis with ischemic stroke. This also applies to patients who have undergone TIA for the first time in their lives, if no more than 48 hours have passed since the regression of neurological symptoms. In the case of a longer time interval (more than 48 hours have passed since the end of the TIA), the patient is examined and treated on an outpatient basis. The examination includes MRI of the head, as well as ECG, DS and EchoCG. Repeated TIA, which developed during the next 12 hours of the outpatient examination period, forces a change in management tactics and serves as an indication for emergency hospitalization. In addition, it is necessary to hospitalize patients with symptomatic carotid stenosis of more than 50% of the artery lumen detected at the stage of outpatient examination; known heart disease, which may be a source of cardiogenic embolism; signs of hypercoagulation with a history of pulmonary embolism or any other arteries (ocular, coronary, mesenteric, peripheral); venous thrombosis, coagulogram changes. The main diagnostic tasks during hospital stay, as mentioned above, are the exclusion of cerebral infarction, differential diagnosis and the establishment of the etiology of TIA. After the completion of the TIA, the main efforts should be directed to the primary prevention of repeated ONMC. Prevention and dispensary supervision, medical indications and contraindications to the use of primary prevention methods, regular blood pressure screening and appropriate therapy for hypertension are recommended (lifestyle modification and drug therapy [14, 15, 17]. Elevated blood pressure makes the greatest independent contribution to the population cardiovascular risk, and blood pressure control is central to any successful strategy to reduce the risk of stroke. It was found that with an increase in diastolic blood pressure for every 10 mm Hg, the risk of stroke increases by 1.95 times. The optimal level of systolic blood pressure is 140 mm Hg. and diastolic blood pressure – 90 mmHg. For patients with hypertension, diabetes mellitus or kidney disease, the target values of blood pressure are < 130/80 mmHg. The use of drugs of various classes, including diuretics, beta-blockers, angiotensin converting enzyme inhibitors (ACE inhibitors), calcium antagonists and angiotensin II receptor blockers (ARBs), causes a similar reduction risk of stroke and cardiac events. Prevention and treatment of diabetes mellitus (DM), as this disease independently increases the risk of ischemic stroke by 1.8 - 6 times. Correction of blood glucose levels is recommended to be carried out by lifestyle changes and the appointment of individual pharmacotherapy [15, 16, 18, 19]. Impaired glucose tolerance is also an independent risk factor for stroke. However, normalization of blood glucose levels, intensive glycemic control (target level of glycated hemoglobin < 7.0%) does not lead to an additional reduction in the risk of stroke, although it is accompanied by a decrease in the number of cases of myocardial infarction (MI) and deaths. To reduce the risk of the first stroke in adult patients with diabetes with hyperlipidemia, the use of statins or fibrates [15, 16, 18, 19]. Patients with diabetes mellitus (DM) are characterized by the progressive course of atherosclerosis and the predominance of proatherogenic risk factors, primarily such as hypertension and lipid metabolism disorders. Correction of cholesterol (HC) (lifestyle changes, lipid-lowering drugs). In patients with coronary pathology, it is recommended to give preference to statin treatment [15, 16, 18, 19]. A direct link between an increased level of cholesterol and an increased risk of ischemic stroke has been proven. An inverse relationship was also revealed between the values of high-density lipoprotein cholesterol and the risk of ischemic stroke. The use of statins in people with coronary pathology is accompanied by a decrease in the relative risk of ischemic stroke by 19 - 32%. The use of lipid-lowering therapy with other drugs (fibrates, niacin and zetimibe) for stroke prevention has not been proven. And it is also recommended to give up smoking, alcohol abuse, rational nutrition in the presence of diseases of the cardiovascular system, adherence to a diet with a restriction of table salt and unsaturated fats, enriched with fiber-rich fruits and vegetables [15, 16, 18, 19]. It is recommended for people with an increased body mass index, diet and increased physical activity to reduce weight [15, 16, 18, 19]. With a sedentary lifestyle, an increase in physical activity associated with the implementation of light-intensity exercise programs: healthy adults of all ages should spend 2 — 2.5 hours a week on regular physical activity or moderate-intensity aerobic training or 1.5 - 2 hours on more intense physical exercises [15, 16, 18, 19]. Insufficient physical activity is associated with increased the risk of general mortality, cardiovascular mortality, cardiovascular morbidity and

stroke. According to world studies, the risk of stroke or death among physically active men and women is on average 25-30% lower compared to the least active people. The protective effect of physical exertion may be due to a decrease in blood pressure and a beneficial effect on other risk factors for cardiovascular diseases, including diabetes and overweight.

**Literature**

1. Vereshchagin N.V., Morgunov V.A., Gulevskaya T.S. Pathology of the brain in atherosclerosis and arterial hypertension. – M.: Medicine, 1997. 288 p.
2. Stroke: diagnosis, treatment, prevention / Edited by Z.A. Suslina, M.A. Piradova. – M.: MEDpress-inform, 2008. 288 p.
3. Hennerizzi M.G., Boguslavsky J., Sacco R.L. Stroke wedge. manual; translated from English under the editorship of V.I. Skvortsova. – M.: MEDpress-inform, 2008. 224 p.
4. Likhachev S.A., Astapenko A.V., Belyavsky N.N. Transient ischemic attacks: etiology, pathogenesis, classification, clinic, diagnosis // Medical news. 2003. No. 10. pp. 31-37.
5. Neurology: National Guidelines / Edited by E.I. Gusev, A.N. Konovalov, V.I. Skvortsova, A.B. Geht. – M.: GEOTAR-Media, 2010. 1040 p.
6. Emergency conditions in neurology / Edited by Z.A. Suslina, M.A. Piradova. – M.: NC of Neurology, 2009. 387 p.
7. Parfenov V.A., Ragimov S.K. Prognosis for transient ischemic attacks based on the results of one-year follow-up // Neurole. journal. 2011. No. 2. pp.23-26.
8. Parfenov V.A. Transient ischemic attacks // RMZH. 2001. No. 25. pp. 1174-1177.
9. Practical cardioneurology / Edited by Z.A. Suslina, A.V. Fonyakina. – M.: IMA-PRESS, 2010. 304 p.
10. Schmidt E. V., Maksudov G. A. Classification of vascular lesions of the brain and spinal cord // Journal of Neuropathology. and psychiatrist. 1971. No. 1. pp. 3-12.
11. Adams and Victor's principles of neurology / Ed. A.H. Ropper, R.H. Brown. –The McGraw-Hill Companies, Inc., 2005.
12. European Handbook of Neurological Management / Ed. N. E. Gilhus, M. P. Barnes, M. Brainin. – Blackwell Publishing Ltd., 2011.
13. Guidelines for management of ischaemic stroke and transient ischaemic attack. European Stroke Organisation (ESO) Executive Committee. ESO Writing Committee // Cerebrovasc Dis. 2008. Vol. 25. № 5. P. 457–507.
14. 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.
15. 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.
16. Salomova, N. K. (2022). Risk factors for recurrent stroke. Polish journal of science N, 52, 33-35.
17. Salomova, N. K. (2023). KAITA ISCHEMIC INSULTLARNING CLINIC POTOGENITIC HUSUSIYATLARINI ANIKLASH. Innovations in Technology and Science Education, 2(8), 1255-1264.
18. 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.

19. 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).
20. 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.
21. Akhrorova, S. B. (2021). Prevalence and neurological features of diabetic polyneuropathy in type 1 diabetes. Journal of Neurology and Neurosurgical Research, 2(2).
22. 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.
23. 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.
24. Rakhmatova, D. I. (2022). The effectiveness of neurotrophic therapy of serotonin in ischemic stroke. Journal of Neurology and Neurosurgical Research, 3(1).
25. Rakhmatova, D. I. (2020). Opportunities of acupuncture in treatment of facial nerve neuropathy. European Journal of Molecular and Clinical Medicine, 7(7), 567-572.
26. Gaffarova, V. F. (2021). Clinic-eeg correlation somatogenous of conditioned febrile seizures in children. International Journal of Human Computing Studies, 3(1), 114-116.
27. 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.
28. 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.
29. Davronova, H. (2023). COGNITIVE DISORDERS IN TYPE 2 DIABETES MELLITUS. International Bulletin of Applied Science and Technology, 3(5), 901-906.