COMPARATIVE STUDY OF RISK FACTORS FOR TIA AND ISCHEMIC STROKES.INITIAL DETERMINATION.

Rakhmatova Sanobar Nizamovna
Usmonov Surat Rustamovich
Bukhara State Medical Institute, Republic of Uzbekistan, Bukhara
https://doi.org/10.5281/zenodo.8150100

Abstract: the duration of neurological symptoms for more than 30 minutes increases the likelihood of developing a cerebral infarction. The pathogenesis of focal cerebral ischemia is characterized by the classical Virchow triad: it is manifested by a decrease in blood flow, damage to the vascular wall and increased blood clotting.The occurrence of molecular and biochemical changes in the skull substance is caused by acute focal ischemia of the skull

Key words: TIA, stroke, cardioembolism, atherosclerosis.

Etiological factors and Tia pathogenesis are mainly associated with three main mechanisms of local cranial hemisphere ischemia: cerebral embolism (heterogeneous sources of embolism), stenooclusion of brachiocephalic or intracranial arteries, which results in temporary decompensation of collateral blood flow resulting in hemodynamic changes.

Regression of neurological symptoms is associated with MK (55ml/100g/min) recovery after reversible local ischemia of brain tissue, which develops with a decrease in volumetric blood flow to 19 ml/100g/min. Further depletion of MK from the critical threshold results in cranial infarction, which is largely determined by MRI data from the brain using diffusing images and gradient T2*weight images. The duration of neurological symptoms for more than 30 minutes increases the likelihood of developing a cranial infarction [1]. The pathogenesis of focal cerebral ischemia is characterized by the classic Virchov triad: manifested by a decrease in blood flow, damage to the vascular wall and increased blood clotting. The occurrence of molecular biochemical changes in cranial matter is caused by acute focal cranial ischemia, which can result from tissue disruption to cell death. The nature of the changes depends on the magnitude of the decrease in cerebral blood flow, the duration of this decrease, as well as the sensitivity of the brain substance to ischemia. Typically, the volume of cerebral blood flow is 55-100ml of blood per 50g of brain matter per minute. An average decrease in blood flow (=40 ml/100 g/min) is accompanied by changes in selective gene expression and protein synthesis processes. A significant decrease in blood flow (up to 100ml per 30g/min) is accompanied by the activation of anaerobic glycolysis and the development of milk acidosis. With a decrease in the volume of cerebral blood flow by 100ml per 20g/min, glutamate excitotoxicity occurs and an increase in intracellular calcium triggers the structural injury mechanisms of these membranes and other intracellular formations. As a result of significant ischemia (up to 100 ml per 10g/min), anoxic depolarization of membranes occurs, cell death usually occurs within 6-8 minutes[2, 3, 4, 5]. In focal cerebral ischemia, the rate of decreased cerebral blood flow rate varies. In the cranial substance, the area caused by less than 10-12 ml/100g per minute of blood flow is called the"necrosis" zone/"nuclear infarction zone". Changes in brain tissue are accompanied by a pronounced energy deficit, loss of ion gradient, depolarization of membranes that cause irreversible damage to cells (in the form of cell

membrane destruction, mitochondrial and DNA destruction). In most cases, necrosis in the nuclear zone of the infarct is formed within 5 minutes after arterial occlusion[2, 3, 4, 5]. In the" Penumbra " zone, brain tissue is not functionally active (there is not enough energy substrate for adequate functioning), but it is vital (the structural integrity of neurons and glial cells is preserved). The ischemic penumbra zone includes independently regenerating areas and areas that develop into brain infarction without successful reperfusion therapy. The main role in the transformation of the Penumbra zone into the center of necrosis is played by the duration of ischemia and a decrease in the level of MK. The level of irreversible damage to the brain tissue is also determined by indicators of the oxygen transport properties of the blood (anemia, violation of hemoglobin oxygenation and its chemical properties), electrolyte balance, osmotic pressure, temperature, glucose content and the state of collateral blood flow [6, 7]. The principle of "time-brain" reflects the rapid death rate of brain tissue cells under the influence of negative factors. The concept of ischemic penumbra emphasizes the need for emergency medical care for patients with acute circulatory disorders of the brain[2, 3, 4, 5]. In a wide ischemic area, which occupies at least 50% of the basin of the spinal blood supply, a pronounced swelling in the disease can be manifested with mass-Effect and vnutricherepnoy hypertension, accompanied by a shift in brain structures with the development of the temporal-tectorial wedge. The cause of most strokes is cardioembolic or thrombotic occlusion of the internal carotid artery or M1 spinal cord. Stroke is a complex severe course of this complication, with hemispheres occurring in 36-78% of patients with ischemic stroke [8,9]. A brain tumor that occurs with a pronounced mass-Effect effect and the development of a neurological defect can develop both within 24-36 hours from the moment of the onset of a stroke, and gradually over several days - weeks [2, 10]. The cascade of local inflammatory reactions, which includes immune cells and anti-inflammatory cytokines, leads to significant brain damage, hematoencephalic barrheal disorders and determines the initiation of the vasogenic component of brain edema. Cranial edema begins on the first day in 25% of patients with malignant stroke[11, 12]. The mortality rate in patients with malignant stroke reaches 78%. Venous stroke thrombosis of the cerebral vessels and hard curtain sinuses is one of the rare causes of stroke and is often undetectable [13]. The frequency of occurrence of venous stroke cases ranges from 0.22 to 1.57 to 100,000 (frequency ratio in women and men is 3/1 [14, 15, 16]. Genderg differences may be associated with increased risk of cerebral vascular thrombosis and hard curtain sinuses associated with pregnancy, postpartum, as well as contraceptives [17]. Thrombosis of the cerebral vessels or hard curtain sinuses prevents the outflow of blood from the brain tissue, which leads to an increase in Venous and capillary pressure, followed by a violation of the hematoencephalic barric barrier, vasogenic edema and the transfer of plasma to the interstitial space. Increased cranial pressure can lead to venous blood clots as a result of ruptured venules and capillaries[18]. For cerebral vascular thrombosis and hard curtain sinuses. One of the lessons that leads to thrombosis of the sinuses of the cranial veins and hard cerebral membranes is prothrombic conditions common risk factors: prothrombotic conditions (both genetic and acquired); taking oral contraceptives; pregnancy and the postpartum period; the presence of malignant neoplasms; infection; head injury [19]. TIA and stroke are the main cause of population disability (3.2 per 1000 inhabitants) [20]. According to the National Stroke register, 31% of stroke patients need the help of tshzgas to take care of themselves, while 20% cannot walk on their own. Only 8% of surviving patients can return to their previous work [21]. According to the epidemiological

study of stroke by the regional-population register method, in 2010, stroke incidence amounted to 1000 cases per 3.27 population, mortality — 0.96 per 1000 population. The ratio of ischemic strokes to hemorrhagic strokes is 5:1, with a median stroke age of 66.7 years (63.7 years for men and 69.4 years for women). In patients under 67 years of age, the absolute number of strokes is higher in men and higher in women in adulthood [22]. By 2016, the number of stroke cases decreased from 1,000 residents to 2.85 residents (compared to 30% by 2009), while the death rate was 0.4 per 1,000 residents (decreased to 22%). The risk of re-stroke is 0.79 per 1000 inhabitants. The proportion of ischemic stroke among relapses was 87.5%, with etiology uncertain strokes at 4.6%. The prevalence of relapsed stroke among all strokes is 25.5% [22]. Population testing results showed that atherotrombotic II incidence was 16%, cardioembolic II was 29%, lacunar II was 16%, and the risk of re-stroke in the first 30 days of disease i for rare causes was higher in atherotrombotic stroke compared to other pathogenetic variants of II [22]. Stroke incidence in the OMA basin accounts for 3% of all stroke[22]. Approximately 10,000 people have a stroke in the OMA Basin each year. Cerebellar nsults make up 0.5% of all strokes. etiology unknown stroke is 36%. Every year, cerebral strokes are observed in 1,500 people. Traditionally, stroke was often found to be abundant in people of older age groups, we can now come and observe that stroke is rejuvenated[14]. This is due to the prevalence and improvement of neurovisualization methods, the spread of risk factors for cardiovascular disease, and the increase in illegal drug use. Stroke incidence in patients under 45 years of age ranges from 11.3 to 100,000 per 3.4 population per year [15]. The causes and risk factors for stroke in young people differ significantly from those in older patients. Young patients are more likely to be diagnosed with congenital and acquired heart disease, blood diseases, vasculopathy, hereditary diseases, drug use. Arterial hypertension, smoking, diabetes mellitus and hypercholesterolemia are more common in elderly patients, although these risk factors are also present at a young age [16, 17]. The incidence of stroke in women during pregnancy and postpartum, and artificial intelligence during pregnancy and postpartum is 100,000 per 11-34 births per year. This is a high indicator of the annual incidence of artificial intelligence compared to the frequency of cases in women of similar reproductive age (10.7 cases per year 100000) [18, 19, 20]. The maximum frequency II occurs in the postpartum period - in 50% of cases, about 40% of prenatal II cases develop, and during childbirth-10% of ii cases. In order to improve the quality of prevention of ischemic stroke during pregnancy and childbirth, in addition to the "general" risk factors for ischemic stroke, it is necessary to take into account the "additional" risk factors of artificial intelligence that appear during these periods: age over 35 years, migraine with Aura, arterial hypertension. pregnant women, postnatal infectious complications [21, 22] The pathogenetic subtype of atherothrombotic ischemic stroke is diagnosed in patients with over 50% stenosis of the Ipsilateral affected hemisphere or occlusion of one of the main arteries of the head. Criteria 1. The clinical picture corresponds to damage to the cerebral cortex (aphasia, motor disorders, etc.) or cerebellum. 2. In Anamnesis-Tia in the affected arterial Basin, noise during auscultation in the carotid arteries, a decrease in their pulsation. 3. The presence of protein in the Anamnesis. 4. According to CT or MRI, the infarction of the cranial cortex, myacha, and subcortical hemisphere exceeds 1.5 cm in diameter. 5. According to duplex scanning or digital subtractive angiography, more than 50% of stenosis or Ipsilateral occlusion of the intra - or extracranial artery to the affected hemisphere. 6. Potential sources of cardiogenic embolism are excluded. 7. If changes in

cerebral arteries during duplex scanning or digital subtractive angiography are minimal or absent, the atherothrombotic pathogenetic subtype of artificial intelligence cannot be diagnosed. A subtype of cardioembolic pathogenetic ischemic stroke is diagnosed in patients with cerebral artery occlusion due to cardiogenic embolism. The clinical picture of TIA/ischemic stroke is determined by the area of blood supply to the affected vessel. Circulatory disorders in the carotid system develop more often than in the vertebral–basilar system (in 80-85% of cases) (in 15-20% of cases). Clinical manifestations of impaired blood flow through the internal sleep artery are mainly characterized by the development of collateral circulation and the level of occlusion. The internal carotid is characterized by damage to the extracranial part of the artery, moderate neurological symptoms, and often manifests itself in the form of a TIA or a small stroke. At the same time, mosaic clinical manifestations can be noted. Ophthalmoplegic syndrome is characterized by the development of Denny–Brown Syndrome when the eye artery exits or occludes in the proximal part of this area, manifested by blindness on the focal side (due to retinal and optic nerve ischemia) and Central hemiplegia or hemiparesis, sometimes in combination with hemigipesthesia on the opposite side. In the occlusion of the intracranial part of the internal thigh artery, neurological defects are characterized by the manifestation of contralateral hemiparesis, hemigipesthesia and psychological functions.

References:

1. Smith, Wade S.; Johnston, S. Claiborne; Hemphill, III, J. Claude (2019). "Cerebrovascular Diseases". In Jameson, J. Larry; Fauci, Anthony S.; Kasper, Dennis L.; Hauser, Stephen L.; Longo, Dan L.; Loscalzo, Joseph (eds.). Harrison's Principles of Internal Medicine (20th ed.)

2Gusev E.I., Skvortsova V.I. Cerebral ischemia, M.: Medicine, 2001. — 328 p.

3. Саломова, Н. К. (2022). ФАКТОРЫ РИСКА ЦЕРЕБРОВОСКУЛЯРНЫХ ЗАБОЛЕВАНИЕ И ПОЛЕЗНОЕ СВОЙСТВО УНАБИ ПРИ ПРОФИЛАКТИКИ. Oriental renaissance: Innovative, educational, natural and social sciences, 2(2), 811-817.

4. Рахматова, С. Н., & Саломова, Н. К. (2021). Қайта Такрорланувчи Ишемик Ва Геморрагик Инсультли Беморларни Эрта Реабилитация Қилишни Оптималлаштириш. Журнал Неврологии И Нейрохирургических Исследований, 2(4).

5. Рахматова, Д. И., & Саломова, Н. К. НОВЫЙ ДЕНЬ В МЕДИЦИНЕ. НОВЫЙ ДЕНЬ В МЕДИЦИНЕ Учредители: Бухарский государственный медицинский институт, ООО" Новый день в медицине", (1), 225-229.

6. Саломова, Н. К. (2021). Особенности течения и клинико-патогенетическая характеристика первичных и повторных инсультов. Central Asian Journal of Medical and Natural Science, 249-253.

7. Саломова, Н. Қ. (2023). ҚАЙТА ИШЕМИК ИНСУЛЬТЛАРНИНГ КЛИНИК ПОТОГЕНИТИК ХУСУСИЯТЛАРИНИ АНИҚЛАШ. Innovations in Technology and Science Education, 2(8), 1255-1264.

8. Salomova, N. K. (2022). Risk factors for recurrent stroke. Polish journal of science N, 52, 33-35.

9. 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.
10. Саломова, Н. Қ. (2023). ҚАЙТА ИШЕМИК ИНСУЛЬТЛАРНИНГ КЛИНИК ПОТОГЕНИТИК ХУСУСИЯТЛАРИНИ АНИҚЛАШ. Innovations in Technology and Science Education, 2(8), 1255-1264.
11. Salomova Nilufar Kakhorovna//FEATURES OF NEUROREHABILITATION ITSELF DEPENDING ON THE PATHOGENETIC COURSE OF REPEATED STROKES, LOCALIZATION OF THE STROKE FOCUS AND THE STRUCTURE OF NEUROLOGICAL DEFICIT//European Journal of Research Development and Sustainability (EJRDS 11. 8-12. 2022/11
12. Саломова, Н. К. (2021). Особенности течения и клинико-патогенетическая характеристика первичных и повторных инсультов. Central Asian Journal of Medical and Natural Science, 249-253.
6. Henneritsi M.G., Boguslavsky J., Sacco R.L.; translated from English; under the general editorship of chl.-corr. RAM Skvortsova V.I. Stroke: Clinical guidelines.– 2nd ed. –Moscow: MEDpress-info, 2008. – 224 p.: ill.
7. Stroke. A guide for doctors. Edited by L.V. Stakhovskaya, S.V. Kotov. MIA Publishing House, 2014. — 400 p.: ill.
8. Tul J.F., Gusev E.I., Vascular diseases of the brain / translated from English. Under the editorship of akad. RAMS E.I. Guseva, Prof. A.B. Geht. A guide for doctors: 6th ed. – Moscow: GEOTAR-Media, 2007. — 608 p.
9. Evolving Paradigms in Neuroimaging of the Ischemic Penumbra Chelsea S. Kidwell, MD; Jeffry R. Alger, PhD; Jeffrey L. Saver, MD, Stroke.2004;35[suppl I]:2662-2665.
10. H.S. Markus Cerebral perfusion and stroke. J Neurol Neurosurg Psychiatry 2004;75:353–361.
11. Liebeskind DS, Jüttler E, Shapovalov Y, Yegin A, Landen J, Jauch EC Cerebral Edema Associated With Large Hemispheric Infarction. Stroke. 2019;50(9):2619. Epub 2019 Aug 20.
13. Wijdicks E., Diringer M. Middle cerebral artery territory infarction and early brain swelling: progression and effect of age on outcome. Mayo Clin. Proc 1998;.73(9): 829-836.
14. Schwab S, Aschoff A, Spranger M, Albert F, Hacke W The value of intracranial pressure monitoring in acute hemispheric stroke. Neurology. 1996;47(2):393.;
15. Nikitin A.S., Krylov V.V., Burov S.A., Petrikov S.S., Asratyan S.A., Kamchatnov P.R., Kemezh Yu.V., Belkov M.V., Zavalishin E.E. Dislocation syndrome in patients with malignant massive ischemic stroke. Journal of Neurology and Psychiatry named after S.S.Korsakov 2015; 3 Special Issue "Stroke": 20-26.
16. Qureshi A.I., Suarez J., Yahia A.M. et al. Timing of neurological deterioration in massive middle cerebral artery infarction: a multicenter review. Crit. Care Med 2003; 31: 272-277.
17. Saposnik G, Barinagarrementeria F, Brown RD Jr, Bushnell CD, Cucchiara B, Cushman M, deVeber G, Ferro JM, Tsai FY Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2011;42(4):1158. Epub 2011 Feb 3.
18. Ferro JM, Correia M, Pontes C, Baptista MV, Pita F Cerebral vein and dural sinus thrombosis in Portugal: 1980-1998. Cerebrovasc Dis. 2001;11(3):177

19. Coutinho JM, Ferro JM, Canhão P, Barinagarrementeria F, CantúC, Bousser MG, Stam J Cerebral venous and sinus thrombosis in women. Stroke. 2009;40(7):2356. Epub 2009 May 28.
20. Stam J Thrombosis of the cerebral veins and sinuses. N Engl J Med. 2005;352(17):1791.
21. Gotoh M, Ohmoto T, Kuyama H Experimental study of venous circulatory disturbance by dural sinus occlusion. Acta Neurochir (Wien). 1993;124(2-4):120.
22Medical and demographic indicators of the Russian Federation in 2012` 2013: Stat. guide/The Ministry of Health of Russia. – M., 2013. – 180 p.