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Transient ischemic attack is a type of transient disorder of blood circulation in the head brain. This makes up 15% of all cases of circulatory disorders in the brain, which in turn causes hypoxia to begin in a certain part of the brain. The patient's condition will depend on which blood vessel is affected, but all clinical signs will disappear within 24 hours. It occurs mainly in people over 60 years of age. The cause of the origin of TIA is characterized by congestion of the origin of cholesterol plaques at the expense of vasoconstriction - in many cases. In large blood vessels with atherosclerosis, that is, in the carotid Basin, in the vertebral - basilar Basin and in the intracerebral arteries, thrombus can come and get more congestion. Slowing blood flow and causing the appearance of clots and blood clots that block smaller vessels in diameter at the expense of a violation in the intima floor in the vascular wall, and

THE SPECIFICS OF SLEEP DISORDERS IN TRANSIENT ISCHEMIC ATTACK

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ABSTRACT

The cause of TIA is characterized by the accumulation of cholesterol plaques due to vasoconstriction - in many cases. In large blood vessels with atherosclerosis, that is, in the carotid pool, in the vertebrobasilar pool and in the intracerebral arteries, a blood clot may form and stagnation may increase. TIA patients have a high incidence of various disorders associated with insomnia. Insomnia can also occur as a result of morphological changes in brain structures involved in the management of sleep and wakefulness, as well as as a result of affective disorders - anxiety and depression.

the accompanying occurrence of hypertension is the main cause of the origin of TIA (in 25-50% of cases). [1,3,5,7]. This leads to a gradual change in the state of the small vessels of the brain. Sometimes the TIA develops directly during a hypertensive crisis. In 20% of patients, circulatory disorders lead to the formation of blood clots that appear due to poor heart function. [1,2,6,7]. The cause can be arrhythmia, aortic stenosis, two-valve, three-valve defect, angina or heart attack. In many cases, there is a combination of several listed reasons. The minimum number of cases (5%) is associated with other pathologies that occur at a young age, namely inflammatory processes in the vessels, changes in the rheological properties of blood, diabetes mellitus, etc. Tia: stages: in its development, the TIA goes through three stages, and the initial stage can be almost invisible. At the initial,



first stage, there is a decrease in blood flow to the brain. [2,4,5,7]. At the same time, special compensation mechanisms will be involved. They cause vasodilation and the blood supply is restored. In the second stage, perfusion is so great that internal mechanisms cannot compensate for it. [2,3,6,8]. But there will still be enough oxygen to keep neurons alive and functioning. At the third stage, the permeability of nerve fibers, especially axons, begins at night with difficulty, hypoxia appears at the cellular level. Symptoms of functional impairment develop. This stage is called ischemic penumbra. Temporary amnesia is a type of Tia in which short-term memory loss develops over several hours. [4,6,7]. Transient blindness is the loss of vision in one of the eyes. At the same time, the patient has a dark veil in front of the eyes, and the mobility of the eyeball is limited. The main cause of the TIA crisis in the section is the embolism of the visual artery. Depending on how often attacks occur, they are divided into rare (up to 2 times a year), medium (up to 6 times a year) and frequent (every month). Mild attacks last about 10 minutes, while severe attacks last between 12 and 24 hours. Most clinical signs are due to the qantomir infestation condition. If the vertebrobasilar Basin is affected, then the Coordination of the patient's movements and speech is impaired. [5,6,7]. A person loses his vertical position, trembling gait appears, involuntary movements may appear or, conversely, lose the ability to move. Speech disorders. Worried about dizziness, headache, vomiting. Hypoxia in the carotid Basin is characterized by visual disturbances, paresis of the hands or feet, convulsions or loss of sensitivity. If short-

term memory suffers, a person begins to ask the same questions, cognitive disorders appear. Changes do not last long, at most a few hours. After that, all functions will be fully restored. Often, no symptoms are observed during hospitalization of the patient or referral to a neurologist. [1,7]. However, a complete examination is required. Vascular angiography may be prescribed to detect blockage of small arteries. In hypertensive microangiopathy, autoregulation of blood flow to the brain is impaired. In this case, harchyl disorders of general hemodynamics (increase or decrease in systemic blood pressure; decreased cardiac myocardial pumping activity and b.) leads to a deterioration of perfusion in brain tissue and an increase in hypoxia, while this condition, in turn, triggers a cascade of pathobiochemical reactions, mainly the death of nerve cells through the mechanism of apoptosis, an increase in AG leads to structural changes in the walls of the trunk arteries in the extracranial areas of the head. Initially, on the inner floor of the arteries, adaptive changes (to an increase in arterial pressure) develop in the form of foci and circular muscle-elastic thickenings. Then, with slowness, pathological structural changes are formed, which are characterized by the death of myocytes and elastic structures and sclerosing of the wall of arteries. [1,3,5,7]. These processes lead to deformation of the trunk arteries of the head (BMA), the formation of pathological curvatures, sprains and severe stenoses, lack of blood circulation in the brain and the progression of TIA. AT-plaques develop at the very first BMA atherosclerosis, and then move to the level of the extracerebral and large intracerebral arteries. Atherosclerotic angiopathy at the level of



the intracerebral vessels is manifested in the form of structural precalibrovka of the vessels. [1,9]. AT-plaques develop at the very first level of BMA, then move to the level of the extracerebral and large intracerebral arteries. Atherosclerotic angiopathy at the level of the intracerebral vessels is manifested in the form of structural precalibrovka of the vessels. Based on modern views, the occurrence and development of AT is a chronic multi-factor pathological process that is complex and not fully studied to the end, the main zvenos of which is the excessive accumulation of atherogenic low-density lipoproteins in the blood and subendothelial space, as well as inflammatory and autoimmune changes in the vessel wall depending on this process, in addition, Based on the accepted rules of the classical lipid theory, the main event in the development of it is considered to be the accumulation of pzlps in the blood serum. PZLPS acquire an atherogenic property after perokis oxidation caused by activated neutrophils in the blood. [8,9,11]. Modified (oxidized) lipoproteins (mpzlp) move from the blood to the vascular intima and accumulate in the subendothelial space. The mpzlp in the vessel wall becomes an active influencer (activators) for monocytes, which in turn move from the blood to the subendothelial space and turn into macrophages. [7,9,12]. Macrophages absorb mpzlp and turn into foam cells filled with cholesterol esters. These cells form the basis of the lipid band, which is the initial phase of atherosclerotic lesions of the vessel. The modified PZLP, which infiltrates intima, is considered to be powerful stimulants of inflammation of the vascular wall. Activated macrophages and foamy cells,

injured endothelial cells produce pre-inflammatory cytokines (interleukins-il-3 (tumor necrosis factor-O'no, u-interferon), adgesia molecules, growth factors (thrombocytic origin factor, main growth factor of fibroblasts and b.), their development is not clearly manifested in the wall of the arteries in the norm. At the stage of development of chronic inflammation in the vascular wall, high-activity lysosomal enzymes are secreted, which lead to injury to the intima and Medea. [8,9,12]. The response reaction that is carried out using the growth factor is the hypertrophy of smooth muscle cells and the thickening of the layer of smooth muscle of the intima, an increase in connective tissue elements in the vessel wall, which leads to re-modeling of the vessel wall (an increase in the thickness of the intima-media complex). An increase in the thickness of the Intima-media complex by more than 0.9 mm is considered the initial stage of atherosclerotic lesions of the arteries. The growth of connective tissue in a certain area and the formation of fibrosis (atherosclerotic) pilach is a reaction of the vessel wall to local inflammation. When hyperlipidemia of the fibrosis plaques is observed, it begins to grow slowly and goes to narrow the vascular cavity. The inflammatory process in the vessel wall is accompanied by the development of endothelial dysfunction, manifested by injuries and all complications of endothelial cells that create conditions for the formation of thrombi inside the vessel (violation of vasodilation, activation of platelets, displacement of hemostasis towards hypercoagulation). Increased levels of pre-inflammatory cytokines, the development of a chronic inflammatory process in the vascular wall, the activation



of hemostasis lead to subsequent disorders of microcirculation, deterioration of perfusion, deepening of ischemia, impaired metabolism and death of brain cells. [11,12,13]. And this process is considered the pathogenetic basis of Tia. The role of autoimmune processes in the development of structural changes in the vascular wall is great. Peroxis modification of pzlps leads to the formation of autoantigenic properties in them and the production of autoantitelos (at), 85% of which are mainly composed of immunoglobulin. the multiple development of circulating ATS against mpzlp leads to the formation of the mpzlp-antitelo immune complex, which has high atherogenicity properties and enhances the expression of cytokines il-3 I Ono a, which participate in the development of vascular wall inflammation. The atherogenic effect is explained by the fact that phagocytosis of pzlp immune complexes by macrophages leads to the accumulation of cholesterol in these cells and their transformation into foamy cells, which produce pre-inflammatory cytokines. In addition, in the most early stages of atherogenesis, immune complexes with their place have a cytotoxic effect, calling apoptosis and (or) cell necrosis, the expression of IL-3 cytokine provokes the production of leukocytoadgitive molecules through endothelial cells, creating favorable conditions for the formation of thrombi. In general, according to the autoimmune theory of at pathogenesis, the formation of mainly at vascular lesions is carried out according to the following scheme: the formation of the mpzlp-antitelo autoimmune complex ->foam cell ->atherosclerotic plaque, atherosclerosis itself is considered as an inflammatory

jararion of an immune nature, which is chronic. [11,12,13].

The APO V-100 receptor absorption blockade of unsaturated fatty acids, which are not exchanged by the cells of the vessel wall according to the concept of unsaturated fatty acid deficiency, becomes the impetus for the launch of atherogenesis. In the norm, Poly-ORs are swallowed through the apov-100-receptor endocytosis pathway in the form of polyene esters of cholesterol. The result of Apo V-100 receptor endocytosis blockade is an essential poly-or deficiency in vascular wall cells and an overabundance of mpzlp that enter the vascular wall in the blood and are covered by macrophages and turn into foam cells. Necrotic death of these calls for local inflammation in the vessel wall. Inflammation through the synthesis of proteins in the acute phase blocks the absorption of essential poly-or by cells, forming a closed ring. [5,7]. AT at all structures of the vessel wall in the atherogenic lipids, the inflammation received cytokines, peroxide oxidation products of lipids, autoimmune reactions and at the very first and most severe degree of intima are injured by exposure to infectious agents.

Currently, the role of chlamydia, viruses and bacteria is being studied as a factor that creates conditions for the penetration of atherogenic mpzlp into the vessel wall, the development of inflammation and the formation of it-plaques.

Based on the mechanism of destabilization and destructiveness of the formed at-pilacs, the main place now is fibrosis focused on aseptic inflammation of the capsule, that is, its infiltration by activated macrophages, the production of aggressive free radicals, the reactogenic form of



oxygen and proteases. Monocytes inflammation associated with the production of Ono by macrophages is also important, its intensity is at the maximum depth of the pilacs. Under the influence of aggressive molecules of oxygen and proteases, as well as activated cells (macrophages, endotheliocytes, lymphocytes, etc.) inflammation by the pre-cytokins due to its production, the integrity of the fibrosis lining of the AT-pilacs is disturbed, the detritus in the lipid composition opens, which leads to the formation of thrombi at the site of the injured layer, entering into direct contact with platelets of the lipid core of the pilaccha. Thrombus fragments and decomposing AT-pilacs provide the basis for the observation of embolism in the distal branches of the arteries affected by atherosclerosis[6,9,10,13].

Emotional disorders lead to sleep disorders, which in turn are inextricably linked. According to the indicators of the International Classification of insomnia sleep disorders (ICDS- 2), sleep disorders are more common in women than in men ($r < 0.05$), especially on days 2-3 of the disease, the predominance in women is more pronounced (64, 3% in women, 38, 5% in men, $r < 0, 05$).

Thiasi has different manifestations of sleep disorders in men and women as can be seen from this table, women have more presomic disorders ($r < 0.05$), such as difficulty falling asleep compared to men (41.6% and 29.2%, respectively), in addition to emotional stress and different localization (in joints, chest, abdomen, etc.) intrasomic disorders such as bad dreaming (34.7% and 16.7%) and frequent (more than 2 times per night) waking up (37.5% and 20.8%) associated with feeling pain are more common in women. Postsomic disorders such as premature waking up in the morning and postsomic asthenia (feeling "insatiable" from sleep, drowsiness and morning fatigue) have also been observed more in women than in men ($r < 0.05$). But at the same time, it was found that some intrasomic (superficial sleep) and postsomic disorders (sleep inversion – insomnia in the evening and drowsiness during the day) are almost identical in members of different sexes. Gender-dependent differences in the frequency of Pre-, intra-and postsomic disorders are manifested in such a way that the disease acquires more pronounced and statistical significance on 2-3 days ($r < 0.05$).

Table 1.

Violation of sleep	Checked groups					
	TIA (n=78)		TIA 1 day (n =39)		TIA II day (n = 39)	
	m(n=42)	w (n=36)	m(n=21)	w(n=18)	m(n=21)	w (n=18)
Difficulty falling asleep	42, 6	28, 2*	37, 7	23, 2	44, 2	31, 7*
Late awakenings	36, 5	21, 8*	15, 7	10, 1	51, 4	22, 1*
Unpleasant dreams	33, 7	15, 7*	25, 7	23, 2	41, 5	16, 4*
Superficial sleep	17, 7	11, 5	6, 7	11, 1	12, 8	23, 8



Post-medical asthenia	36, 1	18, 8*	26, 7	22, 2	42, 8	17, 9*
Getting up early in the morning	33, 3	13, 5*	23, 3	12, 1	40, 5	12, 8*

According to the center's survey, that is, according to Federal somnology, the limit meaning of sleep for patients with TIA or minor strokes was found to occur with approximately the same frequency in men (55.6%) and women (53.3%), whereas on the 2 day of the disease it clearly prevailed in men (64.1% in men, 35.8% in women, The quality of sleep in women is characterized by the fact that compared to men, the disease occurs both on the 1st day (18.3±0.5 and 20.9±1, 0 points, $r<0.05$, respectively), and on the 2nd day (16.0±0.7 and 18.4±0, 3 points, $R<0.01$). As the disease progressed, the quality of sleep in both sexes decreased ($r<0.01$, $r<0.05$). Thus, patients with Thiasia are characterized by the occurrence of various

Insomniac disorders with a high frequency. Insomnia can also occur as a result of morphological changes in brain structures involved in sleep and wakefulness management, as well as as a result of affective disorders - anxiety and depression. The predominance of Insomniac disorders in women with checked Tia is explained by the fact that anxiety-depressive disorders in relation to men are clearly manifested in them and occur with a high frequency.

AG-arterial hypertension

PZLP - low density lipoproteins

AT-atherosclerosis

mpzlp-modified low density lipoproteins

TIA-transitory ischemic snack

At-autoantitelos

References:

1. Рахматова С.Н., Ходжаева Д.Т., Хайдарова Д.К. Особенности клиники и течения неврологических синдромов у мужчин и у женщин, страдающих дисциркуляторной энцефалопатией // Клиническая неврология-Санкт-Петербург.2013.С-239-241.(14.00.00;24.12.2009;№100)
2. .Рахматова С.Н., Б.Г.Гафуров., Клинические особенности дисциркуляторной энцефалопатии у лиц мужского и женского пола. // Неврология.-Ташкент, 2011. № 4. С.- 2-4.(14.00.00;№4)
3. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Корреляции между когнитивными и локомоторными нарушениями у мужчин и женщин при хронической церебральной ишемии.// Неврология.-Ташкент, 2018.№4 С- 10-12. (14.00.00; № 4)
4. Рахmatova S.N., Gafurov B.G., Aliqulova N.A. Correlations between cognitive and locomotors disorders in men and women with chronic cerebral ischemia. // European Science Review. Volume 2. Medical science. 9-10. 48-51 (14.00.00; №19)
5. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Когнитив бузилишларнинг эркак ва аёлларда қиёсий баҳоланиши.// Неврология.-Ташкент, 2019г. №3. С 29-31.(14.00.00;№4)
6. Raхmatova S.N. Nazarova J.A, (2021). Clinical and statistical characteristics of patients with cerebral stroke. Uzbek Medical Journal, 2(1).



7. Suslina Z.A., Varakin Yu.Ya., Vereshchagin N.V. Vascular diseases of the brain. M., MEDpress-inform, 2006. 254 p.
8. Sacco R. L., Diener H.K., Yusuf S. et al. Aspirin and prolonged-acting dipyridamole in comparison with Clopidogrel in repeated stroke // N English J Med. 2008. -359:1238-1251.
9. Рахматова С.Н., Саломова Н.К.//Частота и встречаемость повторного инсульта в узбекистане/ Тиббиетда янги кун. 3.35.2021,. 204,205,206,207,208.
10. Саломова Н.К. //Особенности течения и клиничко-патогенетическая характеристика первичных и повторных инсультов// central asian journal of medical and natural sciences 249-253p
11. Рахматова С.Н., Структура когнитивных нарушений у амбулаторных пациентов с артериальной гипертензией без инсульта и транзиторных ишемических атак в анамнези. // Медицинская наука: Достижения и перспективы.№ 4.2015г.стр.79-82.
12. Рахматова С.Н.,Пулатов С.С.,Ахророва Ш.Б. Клинические особенности геморрагического инсульта пожилым возрасти.// Самарканд. Проблемы биологии и медицины.№2(69) 2012г. Стр-79-81.
13. Рахматова С.Н., Встречаемость депрессии в первичном звене здравоохранения. Истина побеждает. 67-международной (Всероссийской) итоговой научной студенческой конференции Южно-Уральского государственного медицинского университета с международным участием. 2013г (№4) стр- 45-46.
14. Рахматова С.Н.,Рузиев Ш.С. Гендер особенности расстройств корковых функций. // Психоэмоциональные нарушения во врачебной практике: Диагностика, Клиника лечение и профилактика. 2013год. 19 декабрь. Новосибирск 2013. С- 123-125.
15. Рахматова С.Н., Особенности клиники и течения дисциркуляторной энцефалопатии у мужчин и женщин. // Психическое здоровье. Научно-практический журнал. 2014 №3 С-14-15.
16. Рахматова С.Н., Особенности когнитивного дефицита у больных с хронической ишемией мозга в зависимости от пола.//Электронный инновационный вестник. Международный периодический журнал научных трудов.
17. Raxmatova S.N., Gafurov B.G., Aliqulova N.A. Correlation between cognitive and locomotor Abnormalities in males and females in chronic Cerebral ischemia XXIII world congress of neurology. 2016. Киотта. XXIII Всемирный конгресс неврологов.
18. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Особенности клиники и течения дисциркуляторной энцефалопатии у мужчин и женщин//.Тошкент-2018. Ўқув услубий кулланма (14.00.13)
19. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Дисциркулятор энцефалопатияда жинсга боғлиқ когнитив ва эмоционал бузилишларни дифференциал давоси. // Тошкент-2018. Ўқув услубий кулланма.
20. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Аёл ва эркекларда дисциркулятор энцефалопатиянинг когнитив бузилишларида клиника патогенетик ва оптимизациялаштирилган давони узига хослиги. // Методик тавсиянома.
21. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова // Веноз дисциркуляцияси бор беморларда когнитив ва статико-локомотор вазифаларини ўзига хослиги // Тошкент-2018. Ўқув услубий кулланма.



22. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Особенности церебральной гемодинамики при хронической ишемиии мозга // Тошкент-2019. Ўқув услубий кулланма.
23. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Когнитивные нарушения при хронической ишемии мозга с позиции гендерных различий. // Тошкент-2018. Ўқув услубий кулланма.
24. РахматоваС.Н., Б.Г.Гафуров., Н.А.Аликулова., Ж.А.Назарова Клинико-патогенетические особенности венозной дисциркуляции у лиц с хронической ишемией мозга .// Тошкент-2019. Ўқув услубий кулланма.