The American Journal of Medical Sciences and Pharmaceutical Research (ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

Journal Website: https://theamericanjournals.com/index.php/tajmspr

Research Article

VERTEBRAL BASILAR INSUFFICIENCY: PROBLEMS AND PROSPECTS FOR SOLUTIONS

Submission Date: May 20, 2023, **Accepted Date:** May 25, 2023,
Published Date: May 30, 2023 |
Crossref doi: https://doi.org/10.37547/TAJMSPR/Volume05Issue05-11

Kamalova M. I.
Associate Professor At The Department Of Human Anatomy Samarkand State Medical University, Uzbekistan

Suleymanov R.I.
Assistant Of The Department Of Human Anatomy Samarkand State Medical University, Uzbekistan

Kiyamov B.E.
Assistant At The Department Of Human Anatomy Samarkand State Medical University, Uzbekistan

ABSTRACT

Article contains the review of literature data in vertebrobasilar insufficiency. Characteristic of vertebrobasilar system blood vessels topography. etiology and pathogenesis of brain stem blood circulation disturbances are described. The biochemical and pathophysiological mechanisms of brain tissue ischem^ changes. and main clinical manifestations and modern methods of vertebrobasilar insufficiency diagnostic are discussed.

KEYWORDS

Vertebral arteries. cerebral ischemia. vertebral basilar insufficiency.

INTRODUCTION

Circulatory disorders in the vertebral-basilar system (VBS) have long attracted the attention of specialists. They refer to severe and frequent variants of cerebrovascular pathology. According to the WHO Epidemiology and Demography Department. disorders of hemodynamics in the vessels of the vertebral-basilar basin account for more than 30% of all cerebral vascular diseases. Circulatory disorders in the VBS account for about 70% of transient cerebral circulatory disorders. Widespread occurrence, constant growth, high mortality rate, affection of people of working age, high percentage of disability among the diseased put the problem of cerebral vascular diseases of stem localization in the group of socially significant ones. Thus, in Russia the incidence rate of stroke patients reaches 3-4 per 1000 population per year. lethality is

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

13% in the structure of general mortality. The number of patients with the phenomena of chronic cerebral ischemia is steadily increasing. Cerebral stroke and progressive cerebral ischemia are currently the leading causes of disability. Among stroke survivors, only about 1/3 of patients return to work, and 1/3 are permanently disabled, needing more or less constant care. In addition. most patients with spondylogenic circulatory disorders in the vertebral-basilar system are at a young age - from 20 to 50 years emphasizing the importance of the problem. in 2004. World Health Organization. International Stroke Society and the World Stroke Federation launched the Global Stroke Initiative. in which stroke was declared a worldwide epidemic. In the Republic of Belarus the extremely high medical and social significance of the problem of chronic forms of vertebral-basilar circulation disorders is determined by the steady tendency of population aging and increase in the proportion of elderly people in the population. The intensive study of various aspects of vertebral artery pathology began relatively recently. In the 1950s, the syndrome of circulatory insufficiency in the VBS was singled out as an independent clinical concept. Vertebral-basilar insufficiency was defined as "reversible impairment of brain function caused by decreased blood supply to the area fed by the vertebral and main arteries" (thus, the ischemic nature and reversible nature of the disorders were emphasized). In 1953 O.V.Egorova described the vertebral nerve syndrome, and in 1956 N.K.Bogolepov described the clinic of posterior sympathetic syndrome in his textbook of nerve diseases. After a detailed analysis of the occurrence and development of these syndromes, it was suggested that there was a nosological unity of vertebral nerve syndrome, syndrome of insufficient blood circulation in the vertebral-basilar system (vertebral artery syndrome) and posterior cervical sympathetic syndrome. Today vertebral-basilar insufficiency is considered as a condition that develops due to insufficiency of blood supply to certain parts of the brain and causes the appearance of temporary and permanent symptoms. The peculiarities of the structure and functions of the vertebral-basilar arterial system and the peculiarity of clinical symptoms in case of dyscirculation in it have led to the allocation in the last version. International Classification of Diseases (ICD-H) "vertebrobasilar arterial system syndrome" within "transient transient transient cerebral ischemic attacks [attacks] and related syndromes" (ICD-H. O 45.0). Whereas previously chronic cerebral circulatory insufficiency in the IBS was considered in discirculatory encephalopathy. the term "chronic cerebral ischemia" (CCI) is introduced instead in the ICD-X. The various forms of cerebral vascular pathology. leading to CHEM. are classified in ICD under "Cerebrovascular Diseases" of ICD as follows: occlusion and stenosis of the pre-cerebral (I 65) and cerebral (I 66) arteries. not leading to brain infarction. other cerebrovascular diseases (I 67). Cerebral atherosclerosis (I 67.2). hypertensive encephalopathy (I 67.4). chronic generalized cerebral ischemia (I 67.8). consequences of cerebrovascular diseases (I 69).

For the normal course of metabolism of cerebral tissue it is necessary to have a stable cerebral blood flow that provides a sufficient supply of nutrients to the brain: proteins, lipids, carbohydrates (glucose) and oxygen. Stable maintenance of the cerebral blood flow at the level of 50-55 ml/100 g of brain tissue per 1 min at the hemispheric level and 33 ml/100 g of brain tissue per 1 min at the cerebellar level is maintained by autoregulation of the cerebral blood flow. which at the level of large vessels is performed reflexively due to adrenergic and cholinergic receptors of their walls with the help of the regulatory mechanism of carotid sinus and chemical regulation in vessels of microcirculatory channel (under excessive intake i.e. hypocapnia the

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

tone of pre-capillary arterioles increases; under insufficient intake in brain hypercapnia, tonus decreases; under conditions of increased amount of carbon dioxide, microvessels sensitivity to it increases). It has been established that the degree of the damaging effect of ischemia is determined primarily by the depth and duration of the cerebral blood flow decrease. The region of the brain with the most pronounced oligemia (< 10- 15 ml) becomes irreversibly damaged very quickly - within 6 - min from the moment of ischemia development (the core. or nuclear ischemic zone). Within a few hours the central point infarction is surrounded by ischemic. but living tissue - ischemic penumbra zone. or pe-numbra. in which the energy metabolism is generally preserved. only functional. but not structural changes are noted. The severity of ischemic changes is also influenced by the rheological properties of blood (viscosity, aggregation ability of the blood forming elements, etc) and perfusion pressure value. which is defined as the difference between the average BP and the average intracranial pressure. The critical level of cerebral perfusion pressure is 40 mm Hg. below this level cerebral blood flow decreases. and then stops. Processes. initiated in the first hours of acute ischemic brain damage. especially when the area of ischemia is extensive. induce and maintain other "remote" its consequences: genome reaction with the inclusion of genetically programmed molecular programs. dysfunction of astrocytic and microglial cell pools with the development of immune changes and local inflammation in the ischemic focus. microcirculation and blood-brain barrier disorders. Slowly progressing diffuse insufficiency of blood supply of cerebral tissue causes chronic cerebral ischemia and leads to progressive deterioration of brain functioning. In many works the ischemic process is considered as a universal mechanism involving hypoxic cascade of "calcium" cell deathOxidative stress, shifts in intracellular calcium ion balance, activation of proteases, and energy deficit represent the chain of metabolic changes occurring in the tissue during ischemia. The key role of the process of programmed neuronal death in the regulation of cell homeostasis of mature brain tissue is assumed. It is even more reasonable to speak about the presence of neuronal apoptosis in cases of nonstroke course of cerebral vascular disease. short-term ischemia during angiospasm or thromboembolism. manifested by reversible or remitting neurological deficit. Circulatory cerebral hypoxia. not being identical to the concept of "brain infarction", is a dynamic process and implies potential reversibility of functional and morphological changes in brain tissue. It is largely determined by angiogenesis, a natural biological response of the tissue to hypoxia and ischemia, modulated by the release of endogenous growth factors. Often this compensatory response to the hypoxic stimulus proves to be insufficient to return the perfusion levels to normal. Moreover. chronic hypoxia leads to a reduced ability of cells to produce growth factor in response to subsequent episodes of hypoxia and may be partially responsible for inadequate compensatory angiogenesis . At the beginning of the last decade, the phenomenon of "ischemic tolerance" of the brain was described. The essence of this phenomenon is that after a short-term ischemia episode (circulatory hypoxia) the resistance of neurons of vulnerable brain structures (hippocampus, new cortex, striatum, etc.) to a subsequent delayed damaging effect of severe ischemia leads to their death by necrosis or apoptosis increases significantly. In recent years the term "ischemic (hypoxic) preconditioning" has been more frequently used in the literature, which is considered as a form of adaptation of brain, heart and other organ cells to unfavorable factors (in particular. severe disturbances of oxygen supply. blood supply. excitotoxicity - glutamatergic system hyperstimulation, etc.). Hypoxic preconditioning

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

exposure triggers a cascade of signal transduction mechanisms involving intracellular regulatory systems. the genome. neuromodulatory peptides. stress-proteins. ensuring increase in the resistance of brain neurons to severe forms of hypoxia. The adaptive activation of glutamatergic. calcium. phosphoinositide regulatory systems. early genes. transcription factors are of great importance in this process. Thus. neural tissue hypoxia. caused by microcirculatory disorders. is only the trigger mechanism of "ischemic cascade" - pathophysiological changes of metabolic nature. which lead firstly to anabolic and then to catabolic processes. In order to systematize the complex hemodynamic and metabolic changes. occurring in the brain tissue at different stages of circulatory insufficiency. a simplified scheme of the "ischemic cascade" is proposed: reduction of cerebral blood flow; glutamate "excitotoxicity"; intracellular accumulation of calcium ions; activation of intracellular enzymes; increase of nitric oxide synthesis NO and development of oxidative stress; early response gene expression; "distant" consequences of ischemia (local inflammatory reaction. microcirculatory disorders. damages of the blood-brain barrier); apoptosis. The alternative choice between the genetic programs of apoptosis and anti-apoptotic protection and the realization of the mechanisms of necrotic and reparative processes is determined by the level of trophic provision of the brain tissue. During the first minutes of ischemia, the natural protective reaction of the brain is the synthesis of trophic factors and receptors to them. In case of rapid and active expression of genes encoding neurotrophins (growth factors), brain ischemia may not lead to infarct changes for a long time. In the case of ischemic damage formation, the high level of trophic factors ensures the regression of neurological deficit even if the morphological defect that caused it remains. The vertebral-basilar system (VBS) is formed by two vertebral arteries originating from the subclavian arteries at 1.4-3.6 cm on the right and 1.6-5 cm on the left from their origin and merging at the cerebral base into the basilar artery located in the basilar sulcus on the ventral surface of the bridge.

Topographically, there are four parts of the vertebral artery (see Figure): pars prevertebralis (prevertebral) (1) - between musculus scalenus anterior and musculus longissimus cervicis before entering the opening of the transverse process of the VI cervical vertebra; pars transversaria (2) - passes through openings in the transverse processes of the VI-II cervical vertebra (so-called The arteriae vertebralis); pars atlantis (3) (exiting from the transverse process of cervical vertebrae II. The artery turns laterally and enters the opening of the transverse process of the atlantum. it bends behind its superior articular fossa. it passes through the posterior atlanto-occipital membrane, dura mater and enters the canalis vertebralis); pars intracranialis (from the edge of the great occipital foramen to the level of fusion with the similar vertebral artery of the opposite side). The IBS provides blood supply to one third of the brain and covers the sections that differ significantly both structurally and functionally. These are the cervical spinal cord, brain stem and cerebellum, part of the optic tubercle and hypothalamic region, partly occipital, parietal, and mediobasal parts of the temporal lobes. The vertebral arteries are surrounded by periarterial sympathetic plexus regulating vascular tone. The regulatory action of the sympathetic nervous system is also expressed in the integrating influence on the microcirculation. The bundles. fibers and nerve endings are involved in formation of periarterial sympathetic plexus of vertebral artery. Of particular interest is the vertebral nerve. which is part of the deep cervical sympathetic system. The vertebral artery and the vertebral nerve constitute a close. inseparable functional unity.

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

Therefore, the pathology of these entities is also impossible to imagine in separation from each other.

At present, it has been established that the most frequent causes of vertebral-basilar circulation disorders are decreased blood flow in the vertebral artery system due to their occlusion or damage of the nerve apparatus. Moreover, in 65% of cases the vertebral-basilar circulation disorder is associated with the lesion of extracranial parts of the vertebral arteries. At the same time, both sudden and increasing arterial occlusion can develop, including under the influence of a number of extravalvular factors having mechanical and reflex effect on the size of their lumen.

Most researchers share the opinion of N.V. Vereshchagin on the definition of 4 main types of vertebral artery lesions with impaired patency according to etiological factors:

1. occlusive lesions (atherosclerotic stenoses and thromboses. emboli. arteritis of various etiology. fibromuscular dysplasia, etc.);
2. extravasal compression (compression of arteries by osteophytes, articular processes, muscles, vessels, tumors, scars, etc);
3. Deformities (pathological tortuosity. thromboses); 4;
4. anomalies (hypoplasia. anomalies of arteries origin, location and insertion, etc.).

Venous disorders play a certain role in the pathogenesis of vertebral artery syndrome. Even a small stagnation in the vertebral veins. located in the close bone canal. can turn a relative compression of the vertebral artery into an absolute one. A separate group consists of aneurysms (unthrombosed and unruptured) and anomalies in the form of increased size and lumen dilation of arteries. their unusual connection or location. in which blood flow disorders are caused by altered hemodynamic conditions with preserved patency of arteries. as well as traumatic injuries (ruptures. hemorrhages in the arterial wall, etc.). Due to its phylogenetic youth, the vertebral artery, especially its posterior cerebral branch, is particularly vulnerable, and its damage or stenosis most often occurs in the extracranial segment. However, atherosclerosis, often combined with arterial hypertension, remains one of the main etiological factors of cerebral circulatory disorders in general and in the vertebral-basilar basin in particular. Most often, atherosclerosis of the vertebral arteries with narrowing of the lumen and. consequently, with a decrease of blood flow in their basin is the cause of vertebral-basilar insufficiency in elderly people. Besides, thrombi may form in the location of plaques, which further narrow the lumen of the affected arteries and may be a source of thromboembolism during thrombus migration with blood flow into the smaller caliber vessels of the vertebral-basilar basin. It has been shown that occlusion of the vertebral or basilar arteries due to their atherosclerotic lesions is the most frequent cause of cerebellar infarcts with hemodynamic mechanism or caused by small emboli. Atherosclerotic plaques. limiting blood flow in the arteries. lead. in addition. to an increase in vasoconstrictor reactions. which. according to D.D. Molokov and E.M. Burtsev. are often found in the vessels of the vertebro-basilar basin. These reactions are the cause of transient symptoms. indicating short-term ischemia in the region of the brain stem and occipital lobes of the large brain.-cochleovestibular and visual disturbances. The cause of vertebral-basilar insufficiency in young and middle-aged people is often external compression of the vertebral arteries as a result of compression by osteophytes (bone growths), herniated disc. spasms of the neck muscles. A variety of mechanisms may underlie vertebrogenic circulatory disorders in the vertebral artery system, often

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

combined and pathogenetically related. Of all the compression factors, compression of the vertebral artery and traumatization of its sympathetic periarterial plexus by the laterally enlarged and deformed hook-shaped processes of the vertebral bodies are the most important, and the degenerative changes of the spine are accelerated with the addition of atherosclerosis of the vertebral arteries. Vertebrobasilar insufficiency can also be caused by deformation of the vertebral artery canal due to subluxation of the cervical vertebrae. The possibility of realization of one of the mechanisms of cerebrovascular pathology through disruption of the neurogenic regulatory circuit with the formation of a lower level of cerebrovascular reactivity (as a result of past infectious diseases, closed brain injury, latent intoxication and other pathogenic influences) was pointed out by Y.S. Ivanov. G.F. Semin. Painful spasm of the vertebral artery due to irritation of the sympathetic nerve plexus plays a major role in the development of vertebral-basilar insufficiency. Pain in the neck. for example. in osteochondrosis. can contribute to the formation of a pathological autonomic reflex. At this, hyperactivation of the sympathetic nerves innervating the vertebral artery occurs as a result of painful irritation of the spine structures. this is accompanied by its prolonged and stable spasm. According to Kipervas I.P., irritation of the sympathetic plexus of the vertebral artery and vertebral nerve by vertebral and circumvertebral structures causes spasm of vessels of the vertebral-basilar basin to a greater extent than compression of the vertebral arteries. Stenosis of the subclavian artery supplying the upper extremity may also cause symptoms of vertebral-basilar insufficiency. This condition, called subclavian steal syndrome, is caused by redistribution of blood from the vertebrobasilar system to the arteries supplying the upper extremity during exercise. Transient disorders of the vertebrobasilar circulation (transient ischemic attacks) are more common than strokes (about a ratio of 4:1) and in about 95% of cases are caused by atherosclerosis of the cerebral and precerebral arteries. lesions of the small cerebral arteries due to arterial hypertension. diabetes mellitus or cardiogenic emboli. In rarer cases they are caused by vasculitis. hematologic diseases (erythraemia. sickle cell anemia. thrombocytopenia. leukemia). immunologic disorders (anti-phospholipid syndrome). venous thrombosis. migraine. dissection of the precerebral (carotid. spinal) or cerebral arteries. In women, the use of oral contraceptives. In recent years, antiphospholipid syndrome (APS) has been named as one of the suspected causes of juvenile cerebral circulatory disorders. It is based on the production of antibodies to phospholipid components of the coagulation cascade. endothelial membranes. platelets and nerve cells with the development of immunologically mediated coagulopathy. a number of neurological and somatic disorders. Antibodies to phospholipids. binding to platelets. vascular endothelium. clotting and anticoagulation factors. contribute to arterial and venous thrombosis. Some antibodies are anti-neuronal and. interacting with antigens of brain tissue. can cause neurological dysfunction. The leading neurological manifestations of primary AFS are ischemic cerebrovascular disorders (TIAs and ischemic strokes), which are most often associated with small cerebral artery thrombosis, and less often with cardiogenic or arterio-arterial embolism. Occasionally, dissection (dissection of the vertebral artery wall) as a result of neck trauma or even crude medical manipulation during chiropractic treatment can be the cause of IBS. Occasionally, a rather rare systemic disease, fibromuscular dysplasia, forms multiple stenoses of the middle caliber arteries with the process spreading to the vertebral arteries, which may also lead to impaired blood flow in the vertebral-basilar basin.

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

In the initial period, IBS can be compensated by redistribution of blood flow along the vertebral artery of the healthy side, as well as by redistribution of blood flow along the Wilisian circle due to the increase in the volume blood flow along the internal carotid arteries. This mechanism is possible in people with good elastic properties of the brachiocephalic arteries. With long-term existing pathology. as well as with frequent repeated stresses. the compensatory mechanism depletes. which leads to a decrease in the volumetric blood flow through the cerebral vessels. This causes chronic hypoxia of the cerebral substance and can subsequently lead to ischemic stroke. The literature data indicate polysymptomatic clinical manifestations of circulatory disorders in the vessels of the vertebral-basilar basin. The degree of their severity is determined by the peculiarities of the etiology of these disorders and the associated mechanisms of their development and compensation conditions. In spite of the polymorphism of clinical manifestations of vertebral-basilar circulatory insufficiency its dominant symptom is the symptom of vestibular dysfunction. Vestibular dysfunctions in case of circulatory insufficiency in the system of vertebral arteries consist of disturbances of functions of all parts of the vestibular system starting from the receptor. represented by static formations of the labyrinth. conductive pathways. nuclear formations. localized in the brain stem. cerebellar structures and interested parts of cerebral hemispheres. The high frequency of vestibular dysfunction in IBS is explained by the location of vestibular nuclei and supranuclear structures in the zone of vascularization of the vertebral-basilar pool. high reactivity of the vestibular system and increased sensitivity of formations of this system to circulatory disorders. as a result, vestibular formations react to hypoxia first among all structures of the brainstem. Undoubtedly. the paramount importance in the development of vestibular dysfunction at vertebro-basilar circulatory insufficiency will have changes in the vascular-tissue structures of the medulla oblongata and cerebellum that are directly supplied with blood from vessels of this system. Due to insufficient blood supply of the vestibular system, most patients complain of dizziness, in severe cases accompanied by nausea and vomiting. An important feature of vertigo in insufficiency of cerebral circulation in the vestibular system is the dependence of the appearance or intensification of this symptom on the position and change of the head position, i.e. the clinical manifestations of de Klein's symptom. I.M. Zhulev and co-authors indicate that head movement and functioning of the vertebral artery and the greater occipital nerve are determined by the special position of the inferior oblique muscle of the head, the analogue of the rotating muscles of the spinal motion segment. The majority of patients have impaired statics and coordination of movements (atactic symptom complex). tinnitus. which has different timbre - from high (squeak. whistle) to low tones (surf noise. buzzing). Most authors point to vertebrogenic. i.e. mainly peripheral origin of this symptom (due to irritation and compression of the sympathetic plexus of the vertebral artery in cervical osteochondrosis). Serious symptoms indicating a pronounced disorder of blood circulation in the vertebrobasilar system are disorders of speech (dysarthria. transient anarthria) and swallowing. The symptom of transient marked ischemia of stem structures are drop attacks - sudden fall of the patient due to transient decrease or loss of postural tone. not accompanied by loss of consciousness and the appearance of precursor symptoms. Some patients. suffering from vertebro-basilar insufficiency. note various visual disturbances: flickering of flickers before the eyes. the appearance of iridescent circles. flickering of sparkling dots. Photoptysias intensify when the torso leans forward, when the head is tilted, when the torso suddenly

The American Journal of Medical Sciences and Pharmaceutical Research
(ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

changes position from horizontal to vertical. Some patients have increased fatigability, general weakness, somnolence during the day and disorders of night sleep (frequent awakenings followed by a long period of sleeplessness), impaired attention and performance. It should be noted that many symptoms of vertebral-basilar insufficiency are also found in other diseases. Therefore, IBS diagnosis should be based on the analysis of subjective manifestations of the disease (patient's complaints), evaluation of anamnestic data, data of the general somatic, neurological and laboratory-instrumental examination, and include several stages:

- diagnosis of vertebral-basilar TIA is based on clinical criteria;

- etiological diagnosis - diagnosis of the underlying disease, the manifestation of which is TIA, is based on general somatic and clinical-neurological and, if necessary, clinical-physiological, psychological, and laboratory-instrumental examination of the patient;)

- vasotopic diagnosis - diagnosis of the predominant level of vertebral-basilar system vascular lesions - is based on a thorough clinical examination with involvement of additional methods (ultrasound Doppler imaging. computed tomography. radionuclide methods of neuroimaging. magnetic resonance methods of neuroimaging. angiography).

On the whole, the pathology of vertebral arteries appears as heterogeneous lesions of vessels feeding the posterior parts of the brain.

by a common feature - alteration of arterial permeability. The data on the character, localization and frequency of vertebral arteries lesions. their comparison in different groups of patients with all the diversity of vertebral arteries lesion forms allows to identify two main factors determining the blood flow disturbance in vertebral-basilar system: vascular lesions proper and lesion of arterial neural apparatus. The action of these mechanisms can either be joint or one of them can clearly prevail over the other. In conditions limiting the possibilities of collateral circulation, these types of arterial lesions can become a decisive factor in the development of cerebral hemodynamic disturbances.

The fact that there is a connection between impaired blood flow in the vessels of the vertebral-basilar basin, morphological changes in the brain structures that receive blood supply from them, clinical symptoms, the course and outcome of the disease is beyond doubt. Decreased or discontinued blood flow in the vertebral arteries and their branches can cause diffuse or focal brain lesions with infarcts of different size and localization. The severity of pathohistological changes is determined by both peculiarities of angioarchitectonics and sensitivity of neuronal elements to the damaging factor (hypoxia). An important strategy of secondary neuroprotection is the development and implementation of drugs with pronounced neurotrophic and growth factor properties. Growth factors. representing endogenous polypeptides. are ideal candidates for the treatment of stroke. as they possess neuroprotective. reparative and proliferative properties.

Conclusions: The treatment of ischemia not amenable to conventional methods of revascularization is of great importance. Exogenous administration of molecules to stimulate angiogenesis is also of great importance. At the same time, a certain role in solving the problem of cerebrovascular diseases is assigned to "therapeutic angiogenesis" - strengthening of natural processes of the body for the development of collateral vessels in ischemic tissues. The efficacy of

The American Journal of Medical Sciences and Pharmaceutical Research (ISSN – 2689-1026)
VOLUME 05 ISSUE 05 Pages: 49-57
SJIF IMPACT FACTOR (2020: 5. 286) (2021: 5. 64) (2022: 6. 319) (2023: 7. 396)
OCLC – 1121105510
Publisher: The USA Journals

such technique has been confirmed in various animals on models of limb ischemia or myocardium and two approaches to the problem of artificial stimulation of collateral vessels growth have been defined. The first one is related to the introduction of recombinant angiogenic growth factors. The second approach is based on the transfer of the gene. encoding SSFR. using a viral vector. native or plasmid DNA and has a theoretical advantage. since the growth factor synthesized in this way has a higher concentration and correspondingly more significant biological effect with a small number of infected cells. This strategy can provide effective treatment of patients. suffering from CHD or peripheral arterial occlusive lesions.

REFERENCES

1. Butko. D. Butko D. Y. State of cerebral hemodynamics and statokinetic functions in patients with vertebral-basilar vascular insufficiency // Jour. neurolog. and psychiatr. n. S. Korsakov. - 2004- T. 104. № 12. - C. 38- 42.
2. Khodjieva D. T., Khaydarova D. K. Clinical and neuroph clinical and neurophysiological ch ogical characteristics of teristics of post-insular cognitive disorders and issues of therapy optimization. Central Asian Journal of Pediatrics. Dec.2019. P 82-86
3. . Vereshchagin. N.V. Vertebro-basilar system pathology and disorders of cerebral circulation / N.V. Vereshchagin. - M.: Medina. 1980. -312 c.
4. Gannushkina. Physiology and pathophysiology of cerebral circulation / I.V. Gannushkina // Vascular diseases of the nervous system / ed. by E.V. Schmidt. - M.: Medicine. 1975. - C. 66-105.
5. Gusev. E. I. Semax in the prevention of progression and development of exacerbations in patients with dyscirculatory encephalopathy / E. I. Gusev. V. I. Skvortsova. E. I. Chukanova // Journal of Neurology and Psychiatry. C. C. Korsakov. - 2005. - T. 105. № 2. - C. 35-40.1.
6. Khaidarov Nodir Kadyrovich, Shomurodov Kahramon Erkinovich, & Kamalova Malika Ilhomovna. (2021). Microscopic Examination Of Postcapillary Cerebral Venues In Hemorrhagic Stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 3(08), 69–73.
7. Kirkovsky V.V.. Detoxification therapy in peritonitis: Method. handbook for physicians and students. Minsk: Polifakt-Alfa. 1997.
8. Khodjieva D.T., Pulatov S.S., Khaidarova D.K. All about hemorrhagic stroke in elderly and senile persons (own observations) // Science of Young People (Eruditio Juvenium). 2015. №3. C. 87-96.
9. Khamdamov B.Z. Indicators of immunocitocine status in purulent-necrotic lesions of the lover extremities in patients with diabetes mellitus.//American Journal of Medicine and Medical Sciences, 2020 10(7) 473-478 DOI: 10.5923/j.ajmm.2020.- 1007.08
10. Ilkhomovna, K. M., Eriyigitovich, I. S., & Kadyrovich, K. N. (2020). Morphological Features Of Microvascular Tissue Of The Brain At Hemorrhagic Stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 2(10), 53-59.
11. Khodjieva D. T., Khaydarova D. K., Khaydarov N. K. Complex evaluation of clinical and instrumental data for justification of optive treatment activites in patients with resistant forms of epilepsy. American Journal of Research. USA. № 11-12, 2018. C.186-193.