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Infarctions in the Vertebrobasilar Basis: Clinical and Diagnostics

Fakhmiddin Khayriddinovich Muratov
Professor, DSc, PhD, MD of the Department of Nervous Disease and Medical Psychology, Tashkent Medical Academy, Tashkent, Uzbekistan

Sardor Ismatulloyevich Khatamov
3rd-year master of the Department of Nervous Disease and Medical Psychology, Tashkent Medical Academy, Tashkent, Uzbekistan

Nilufar Muminjonovna Karimova
3rd-year master of the Department of Nervous Disease and Medical Psychology, Tashkent Medical Academy, Tashkent, Uzbekistan

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ABSTRACT

Strokes in the vertebrobasilar region account for 20-25% of all ischemic strokes. Transient ischemic attacks in the vertebrobasilar territory may involve transient or minor brainstem symptoms and are more difficult to diagnose than ischemic events in the carotid territory. The risk of recurrent stroke in the vertebrobasilar region is as high as with a stroke in the carotid region, and vertebrobasilar stenosis increases the risk by 3 times. Patients with hydrocephalus or increased intracranial pressure may require emergency neurosurgical intervention. Basilar occlusion is associated with high mortality or severe disability, especially if blood flow in the vessel is not restored.

INTRODUCTION

The work carried out a clinical neuroimaging analysis in 79 patients with a clinical picture of ischemic infarction in the vertebrobasilar basin (VBB). The features of the neurological clinic of lacunar and non-lacunar posterior circular infarctions are described. To verify them, we used the magnetic resonance imaging (MRI) technique. The diffusion-weighted magnetic resonance imaging (DWMRI) technique turned out to be more informative for the diagnosis of acute lacunar and non-lacunar infarctions in the brain stem.

Occlusive lesions of arteries in the vertebral-basilar basin (VBB) lead to the development of posterior circulation infarcts with localization in various parts of the brain stem, thalamus, occipital lobes and cerebellum. The frequency of their development ranks second (20%) after infarction in the middle cerebral artery (MCA) (Kamchatov P.R., 2004) and is 10-14% in the structure of all ischemic strokes (Vinichuk S.M., 1999; Evtushenko S.K., 2004; Toi H. et al, 2003). According to other authors, in Europeans, pathology of intracranial arteries in the VBB is more common than in the carotid region (Worlow C.P. et al., 1998).

The posterior vertebrobasilar system is evolutionarily more ancient than the anterior carotid system. It develops completely separately from the carotid system and is formed by arteries that have different structural and functional characteristics: vertebral and main arteries and their branches.

In the vertebral-basilar system, three groups of arteries are distinguished (Worlow C.P. et al., 1998)

- small arteries, the so-called paramedian ones, arising directly from the trunks of the vertebral and basilar arteries, from the anterior spinal arteries, as well as deep perforating arteries originating from the posterior cerebral artery (PCA);
- short circumflex (or circular) arteries, respectively, supplying blood to the lateral territories of the brain stem, the tegmental area, as well as long circumflex arteries - posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), superior cerebellar artery (SCA), PCA with its branches and the anterior villous artery;
- Large or large arteries (vertebral and main) in the extra- and intracranial sections.

The presence in the posterior vertebrobasilar basin of arteries of different calibers with differences in their structure, anastomotic potential and with different areas of blood supply of small, deep perforating arteries, short and long circumflex arteries, as well as large arteries in most cases determine the localization of the lesion, its size and clinical course of posterior circular infarctions. At the same time, individual differences in the location of the arteries and the diversity of pathogenetic mechanisms very often determine the individual characteristics of the neurological clinic in acute ischemic strokes in the VBB.

Therefore, along with the presence of typical neurological syndromes, doctors often note not the clinical picture of vertebrobasilar stroke, which is described in clinical guidelines, but its atypical course, which makes it difficult to determine the nature of the stroke and choose adequate therapy. In such a clinical situation, only brain imaging methods can help.

MATERIALS AND METHODS OF RESEARCH

A comprehensive clinical and neuroimaging examination was carried out on 79 patients (48 men and 31 women) aged from 37 to 89 years (on average 65.2±1.24 years). The study included all patients admitted with a clinical picture of acute ischemic stroke to the VBH. Patients were admitted within 6-72 hours from the onset of the first symptoms of the disease. The main cause of ischemic cerebrovascular accidents (CVA) was arterial hypertension in combination with vascular atherosclerosis (74.7%), in another 22.8% of those examined it was combined with diabetes mellitus; in 25.3% of patients the main etiological factor of the disease was atherosclerosis.

Patient information was recorded in standard protocols, which included demographics, risk factors, clinical symptoms, laboratory and neuroimaging results, outcome, etc.

The degree of impairment of neurological functions was assessed during patient hospitalization, during treatment and at the end of therapy using the NIHSS scale (National Institutes of Health Stroke Scale, USA).

At the same time, the scale of B. Hoffenberth et al (1990) was used, which suggests a more adequate assessment of clinical parameters in acute cerebrovascular accidents in the VBB. To assess the degree of recovery of neurological functions, the modified Rankin scale was used (G.Sulter et al., 1999). Subtypes of ischemic strokes were classified according to the National Institute of Neurological Disorders and Stroke Study Group (Special Report from the National

Institute of Neurological Disorders and Stroke (1990) Classification of cerebrovascular diseases III. Stroke 21: 637-676; TOAST criteria were also taken into account (Trial of ORG 10172 in Acute Stroke Treatment - a study of low molecular weight heparin ORG 10172 in the treatment of acute stroke) (A.J.Grau et al., 2001). The definition of lacunar syndromes was based on data from clinical studies by C.M. Fisher (1965; 1982) and neuroimaging methods.

Standard laboratory tests were carried out: examination of the levels of glucose, urea, creatinine, hematocrit, fibrinogen, acid-base balance, electrolytes, lipids, and indicators of blood coagulating properties.

All patients underwent ultrasound Dopplerography of the great vessels of the head in the extracranial region (USDG) and transcranial Dopplerography (TCD), in some cases - duplex scanning; a 12-electrode ECG was performed, blood pressure (BP) was monitored; volumetric MV was determined in the internal carotid artery (ICA) and vertebral artery (VA).

Spiral computed tomography (SCT) of the brain was performed in all cases immediately upon admission of patients to the hospital. It made it possible to determine the type of stroke: ischemia or hemorrhage. However, the use of SCT did not always make it possible to detect brainstem infarction during the acute period of the disease.

In such cases, routine magnetic resonance imaging (MRI) was used, since magnetic resonance images of the posterior cranial fossa are more informative than SCT. MRI of the brain was performed on a Magnetom Symphony device (Siemens) with a magnetic field strength of 1.5 Tesla and on a Flexart device (Toshiba) with a magnetic field strength of 0.5 Tesla. A standard scanning protocol was used, including obtaining TIRM (Turbo Inversion Recovery Magnifucle) and T2-weighted images (T2-BI) in the axial plane, T1-weighted images (T1-BI) in the sagittal and coronal planes.

However, in the presence of several pathological foci, using MRI techniques it was difficult to determine the extent of their age and to verify the source of the infarction in the medulla oblongata, especially in the acute period. In such cases, a more sensitive neuroimaging technique was used—diffusion-weighted magnetic resonance imaging (DW MRI).

Diffusion-weighted imaging (DWI) can identify the site of acute cerebral ischemia within a few hours of stroke, as evidenced by a decrease in the measured diffusion coefficient (MDI) of water and an increase in the MR signal on DWI. Limitation of water diffusion occurs due to insufficiency of energy (loss of tissue ATP, weakening of the function of the sodium-potassium pump) and the ensuing cytotoxic edema of ischemic brain tissue (Neumann-Haefelin T at al., 1999).

Therefore, it is believed that DWI is especially sensitive in identifying a focus of ischemia with a reduced ATP content and a high risk of irreversible neuronal damage (von Kummer R., 2002). Brain tissue after acute focal ischemia with a high MR signal on DWI and a low ICD corresponds to an infarct focus.

Another modern sensitive technique for brain neuroimaging is perfusion-weighted (PV) MRI, which is used in clinical practice, provides information about the hemodynamic state of brain tissue and can detect perfusion disorders both in the ischemic core zone and in the surrounding collateral areas.

Therefore, during the first hours after the onset of stroke, areas of perfusion disorders on a perfusion-weighted image (PWI) are usually more extensive than on DWI. It is believed that this zone of diffusion-perfusion mismatch (DWI/PVI) reflects the ischemic penumbra, i.e. “tissue at risk” for functional impairment (Neumann-Haefelin T at al., 1999).

We performed DW MRI in the axial plane when examining 26 patients (32.9%): 12 patients

were examined within 24 hours after the onset of infarction, including 1 within 7 hours, 2 before 12 hours from the onset of the disease. The remaining patients underwent DWI on days 2-3 and over the course of the disease: 4 patients were examined 3 times, 8 patients were examined 2 times - 14.1 times.

Magnetic resonance angiography, which allows visualization of large extra- and intracranial arteries, was performed in 17 patients (30.4%) with nonlacunar ischemic infarction.

The purpose of our study is to evaluate the value of clinical and neuroimaging methods in the diagnosis of lacunar and non-lacunar posterocircular infarctions.

RESULTS AND DISCUSSION

Clinical neuroimaging examination of 79 patients (48 men and 31 women, aged 60 to 70 years) with a clinical picture of ischemic stroke in the VBB allowed us to identify the following clinical forms of acute ischemic disorders of cerebral circulation: transient ischemic attacks (TIA) (n=17), lacunar TIA (n=6), lacunar infarction (n=19), non-lacunar infarction in the VBB (n=37). In patients with TIA and lacunar TIA, the neurological deficit regressed within the first 24 hours from the onset of the disease, although in patients with lacunar TIA small foci of lacunar infarction were detected on MRI. We analyzed them separately. Therefore, the main study group consisted of 56 patients.

Taking into account the causes and mechanisms of development of acute ischemic infarction, the following subtypes of ischemic infarction were identified: lacunar infarction (n=19), atherothrombotic (n=21), cardioembolic (n=12) infarction and infarction of unknown cause (n=4)

The frequency of localization of detected ischemic infarction in the VBB, verified by neuroimaging methods, was different. As can be seen from the data presented, most often foci of infarction were detected in the area of the pons (32.1%), thalamus (23.2%), less often - in the area of the cerebral peduncles (5.4%). In many of the subjects (39.4%), posterior circular infarctions were caused by multifocal lesions: the medulla oblongata and cerebellar hemispheres (19.6%); various parts of the brain stem and cerebellar hemisphere, occipital lobe of the brain; cerebellar hemispheres and thalamus; occipital lobes of the brain.

Although on the basis of clinical data it was impossible to accurately determine the arterial localization of the lesion, neuroimaging methods made it possible to conduct a clinical description of the infarction in the VBB, taking into account the vascular territory of the blood supply and, using TOAST criteria, to classify all posterior circular ischemic infarctions into lacunar and non-lacunar.

Classification of ischemic infarctions in the VBB according to etiological and pathogenetic characteristics:

- lacunar infarctions due to damage to small perforating arteries, caused by microangiopathies against the background of arterial hypertension and diabetes mellitus, provided there are no sources of cardioembolism and stenosis of large vertebrobasilar arteries (n=19);
- non-lacunar infarctions due to damage to the short and/or long circumflex branches of the vertebral and basilar arteries in the presence of sources of cardioembolism and the absence of stenosis of the large vertebrobasilar arteries (n=30);
- Non-lacunar infarctions due to occlusive lesions of large arteries (vertebral and main), in the extra- or intracranial sections, i.e. caused by macroangiopathies (n=7).

As can be seen from the data presented, damage to small branches was the cause of lacunar

infarctions in 33.9% of cases; damage to the short or long circumflex branches of the vertebral or basilar arteries was the most common (53.6%) cause of nonlacunar infarction; occlusion of large arteries also led to the occurrence of nonlacunar infarction and was detected in 12.5% of subjects. The localization of the lesion on MRI and DW MRI of the brain relatively often correlated with the neurological clinic.

I. LACUNARY INFARCTIONS IN THE VBB

The clinical characteristics and outcome of 19 patients with lacunar infarctions (LI) in the VBI,. by neuroimaging methods, were studied. If the LI diameter was more than 1 cm during the first study, it often increased with repeated MRI.

Lacunar infarctions occurred as a result of damage to a separate paramedian branch of the VA, OA, or one perforating thalamogenicular artery, a branch of the PCA, against the background of arterial hypertension, which was often combined with hyperlipidemia, and in 6 patients with diabetes mellitus. The onset of the disease was acute, sometimes accompanied by dizziness, nausea, and vomiting. The background neurological deficit on the NIHSS scale corresponded to 4.14±0.12 points, on the B. Hoffenberth scale - 5.37±0.12 points, i.e. corresponded to mild neurological dysfunction.

More often (n=9) a purely motor infarction (PMI) was detected, caused by damage to the motor pathways in the area of the base of the bridge, which are supplied with blood by small paramedian arteries arising from the main artery. It was accompanied by paresis of the facial muscles and arms, or the entire arm and leg suffered on one side. Complete motor syndrome was detected in 3 patients, partial - in 6 (face, arm or leg), they were not accompanied by objective symptoms of sensory disorders, obvious dysfunctions of the brain stem: loss of visual fields, hearing loss or deafness, tinnitus, diplopia, cerebellar ataxia and severe nystagmus.

For illustration, we present an MRI of the patient, performed 27 hours from the onset of the disease, T2 TIRM - weighted tomogram in the axial projection, which revealed a lacunar infarction in the right parts of the pons. The diagnosis of LI is confirmed by DW MRI findings and a diffusion map. ChDI was clinically determined.

Lacunar infarctions in the thalamus in 5 patients caused the development of pure sensory syndrome (PSS), the cause of which was damage to the lateral parts of the thalamus due to occlusion of the thalamogenic artery. Hemisensory syndrome was complete in 2 patients and incomplete in 3. Complete hemisensory syndrome was manifested by a decrease in superficial and/or deep sensitivity or numbness of the skin according to the hemitype in the absence of homonymous hemianopia, aphasia, agnosia and apraxia. In incomplete hemisensory syndrome, sensory disorders were recorded not on the entire half of the body, but on the face, arm or leg. In 2 patients, cheiro-oral syndrome was detected, when sensory disturbances occurred in the area of the corner of the mouth and palm homolaterally; One patient had cheiro-oral-pedal syndrome; it was manifested by hypalgesia of pain sensitivity in the area of the corner of the mouth, palms and feet on one side without motor disorders.

In 2 patients, lacunar infarction of the thalamus was accompanied by the spread of ischemia towards the internal capsule, which led to the development of sensorimotor stroke (SMS). Neurological symptoms were caused by the presence of a lacuna in the lateral nucleus of the thalamus, but there was an effect on the adjacent tissue of the internal capsule. The neurological status revealed disturbances in sensitivity and movements, but disturbances in sensitivity preceded disturbances in motor skills.

2 patients were diagnosed with “atactic hemiparesis”. Gaps were revealed at the base of the bridge. The neurological clinical picture was manifested by hemiataxia, moderate weakness of the leg, and mild paresis of the arm. Dysarthria and clumsy hand syndrome (dysarthria-clumsy -

hand syndrome) was detected in one patient; it was caused by the localization of the lacuna in the basal parts of the pons and was accompanied by dysarthria and severe dysmetry of the arms and legs.

Lacunar infarctions in the VBB were characterized by a good prognosis, restoration of neurological functions occurred on average on the 10.2±0.4 day of treatment: in 12 patients there was complete recovery, in 7 minor neurological microsymptoms (dysesthesia, pain) remained, which did not affect the performance of their tasks. previous responsibilities and daily life activities (1 point on the Rankin scale).

II. NONLACUNARY INFARCTIONS IN THE VBB

The clinical characteristics of patients with nonlacunar infarction with VBB of various etiologies were divided into 2 groups depending on the damage to the vertebrobasilar vessels:

Short or long circumflex branches of the vertebral (VA) or basilar (BA) arteries and large arteries of the vertebrobasilar bassin.

The most common neurological symptoms in patients with acute ischemic infarction due to damage to the short or long circumflex branches of the vertebral (VA) or basilar (BA) arteries were: systemic dizziness, headache, hearing impairment with noise in the same ear , motor and cerebellar disorders, sensory disorders in Zelder areas and/or mono- or hemitype.

The clinical and neurological profile of posterior circular infarctions due to damage to large arteries (vertebral and main) in all patients was manifested by a defect in the visual field, movement disorders, disturbances in statics and coordination of movements, pontine gaze paresis, and less often - dizziness, hearing impairment.

Analysis of the background neurological deficit in patients with non-lacunar infarctions due to damage to the short or long circumflex arteries of the VA or OA indicates that the impairment of neurological functions according to the NIHSS scale corresponded to moderate severity (11.2 ± 0.27 points), and according to the B. Hoffenberth scale - severe disorders (23.6±0.11 points). Thus, scale B.

Hoffenberth et al (1990) compared with the NIHSS scale when assessing acute vertebrobasilar stroke more adequately reflected the impairment of neurological functions and the severity of the patient’s condition. At the same time, in case of infarctions in the VSB due to damage to large arteries and the development of a gross neurological defect, the scales used unidirectionally reflected the volume of neurological deficit, probably because extensive ischemic infarctions predominated in patients. The initial blood pressure level in patients with occlusion of the large arteries of the spinal vein was significantly lower than in patients with lesions of the short or long circumflex branches of the vertebral or basilar artery. In some patients with occlusion of large arteries, which caused the development of large-focal brainstem infarction, arterial hypotension was recorded upon admission.

On the other hand, arterial hypertension in the first day after a stroke in patients with damage to the short or long circumflex branches of the VA and OA could be a manifestation of a compensatory cerebrovascular reaction (Cushing's phenomenon), which occurred in response to ischemia of the brainstem formations. Noteworthy was the lability of blood pressure during the day, with its increase in the morning hours after sleep.

The clinical picture of nonlacunar infarctions caused by damage to the short and/or long circumflex branches of the vertebral and basilar arteries in the presence of sources of cardioembolism and the absence of stenosis of the large vertebrobasilar arteries was heterogeneous with a different clinical course. All other things being equal, the development of focal changes in the posterior parts of the brain depended on the level of the lesion, the arterial

bed and the size of the infarct.

Blockage of the posterior inferior cerebellar artery was manifested by alternating Wallenberg-Zakharchenko syndrome. In the classic version, it was manifested by systemic dizziness, nausea, vomiting, dysphagia, dysarthria, dysphonia, impaired sensitivity on the face of a segmental dissociated type in the Zelder zones, Berner-Horner syndrome, cerebellar ataxia on the side of the lesion and movement disorders, hypoesthesia of pain and temperature sensitivity on the torso and limbs on the opposite side. The same neurological disorders were characterized by blockage of the intracranial part of the VA at the level of the origin of the posterior inferior cerebellar artery and paramedian arteries.

Variants of Wallenberg-Zakharchenko syndrome were often observed, which occurred with occlusive lesions of the paramedian arteries of the VA, medial or lateral branches of the PICA and were clinically manifested by systemic dizziness, nystagmus, and cerebellar ataxia.

MRI of the brain revealed infarcts in the medial or lateral parts of the medulla oblongata and lower parts of the cerebellar hemispheres.

In the case of cardioembolic occlusion of the paramedian or short circumflex branches of the basilar artery, nonlacunar infarctions occurred in the pontine region.

Their neurological clinical picture was polymorphic and depended on the level of damage to the arterial bed and the location of the infarction.

Blockage of the paramedian arteries of the bridge was manifested by alternating Foville syndrome - peripheral paresis of the facial muscles and external rectus muscle of the eye on the side of the lesion with contralateral hemiparesis or Millard-Hubler syndrome: peripheral paresis of the facial muscles on the side of the lesion and hemiparesis on the opposite side.

When the branches of the main artery supplying the midbrain were blocked, paresis of the muscles innervated by the oculomotor nerve occurred on the side of the lesion and hemiplegia on the opposite side (Weber syndrome) or hemiataxia and athetoid hyperkinesis in the contralateral limbs (Benedict syndrome) or intentional hemitremor, hemiataxia with muscle hypotonia (Claude's syndrome). With a heart attack in the quadrigeminal artery basin, upward gaze paralysis and convergence insufficiency (Parinaud's syndrome) occurred, which was combined with nystagmus.

Bilateral infarctions in the region of the paramedian and short circumflex arteries of the OA were characterized by the development of tetraparesis, pseudobulbar syndrome and cerebellar disorders.

Cerebellar infarction occurred acutely as a result of cardiac or arterio-arterial embolism of the anterior inferior cerebellar artery or superior cerebellar artery and was accompanied by general cerebral symptoms and impaired consciousness.

Blockage of the PICA led to the development of an infarct focus in the area of the inferior surface of the cerebellar hemispheres and the pons. The main symptoms were dizziness, tinnitus, nausea, vomiting and on the side of the lesion, paresis of the facial muscles of the peripheral type, cerebellar ataxia, Berner-Horner syndrome. With occlusion of the SMA, the focus of the infarction formed in the middle part of the cerebellar hemispheres and was accompanied by dizziness, nausea, and cerebellar ataxia on the side of the focus. Cerebellar ischemic strokes also occurred when the vertebral or basilar arteries were blocked.

Blockage of the internal auditory (labyrinthine) artery, which in most cases originates from the anterior inferior cerebellar artery (can also arise from the main artery) and is the terminal one, occurred in isolation and was manifested by systemic dizziness, unilateral deafness without signs

of damage to the brain stem or cerebellum.

Blockage of the PCA or its branches (calcar and parieto-occipital artery) was usually accompanied by contralateral homonymous hemianopia, visual agnosia with preservation of macular vision.

In the case of left-sided localization of the infarction, amnestic or semantic aphasia and alexia occurred. Damage to the branches of the PCA, which supply blood to the cortex of the parietal lobe at the border with the occipital lobe, was manifested by cortical syndromes: disorientation in place and time, visual-spatial disturbances. Large focal infarctions of the occipital lobe of the brain were accompanied by hemorrhagic transformation of the infarction.

Thalamic infarctions occurred as a result of damage to the thalamo-subthalamic (thalamoperforating, paramedian branches) and thalamogenicular arteries, which are branches of the posterior cerebral artery.

Their occlusion was accompanied by depression of consciousness, paresis of upward gaze, neuropsychological disorders, memory impairment (anterograde or retrograde amnesia), and contralateral hemihypesthesia. More severe disorders (depression of consciousness, paresis of upward gaze, amnesia, thalamic dementia, akinetic mutism syndrome) occurred with bilateral thalamic infarction, which developed as a result of atheromatous or embolic occlusion of the common pedicle of the thalamo-subthalamic artery, the paramedian branches of which supply blood to the posteromedial parts of the thalami.

Occlusion of the thalamo-genicular artery caused the development of an infarction of the ventrolateral region of the thalamus and was accompanied by Dejerine-Roussy syndrome: on the side opposite the lesion, transient hemiparesis, hemianesthesia, choreoathetosis, ataxia, hemialgia and paresthesia were detected.

Blockage of the posterior villous arteries, which are branches of the PCA, led to the development of infarction in the region of the posterior parts of the thalamus (cushion), geniculate bodies and was manifested by contralateral hemianopsia, sometimes mental disturbances. Occlusion of the vertebral artery (VA) occurred at both extracranial and intracranial levels. With occlusion of the extracranial part of the VA, short-term loss of consciousness, systemic dizziness, visual impairment, oculomotor and vestibular disorders, disturbances in statics and coordination of movements were noted, paresis of the limbs, and sensory disturbances were also detected.

Often there were attacks of sudden falling - drop attacks with impaired muscle tone, autonomic disorders, breathing disorders, and cardiac activity. MRI of the brain revealed infarct foci of the lateral parts of the medulla oblongata and the lower parts of the cerebellar hemispheres.

Occlusion of the intracranial part of the VA was manifested by alternating Wallenberg-Zakharchenko syndrome, which in the classical version was also detected when the PICA was blocked.

Blockage of the basilar artery was accompanied by damage to the pons, midbrain, cerebellum, and was characterized by loss of consciousness, oculomotor disorders caused by pathology of the III, IV, VI pairs of cranial nerves, the development of trismus, tetraplegia, impaired muscle tone: short-term decerebrate rigidity, hormetonic convulsions, which were followed by muscle hypo - and atony. Acute embolic occlusion of the OA in the bifurcation area led to ischemia of the ristral parts of the brainstem and bilateral ischemic infarction in the blood supply of the posterior cerebral arteries.

Such a heart attack was manifested by cortical blindness, oculomotor disorders, hyperthermia, hallucinations, amnesia, sleep disorders, and in most cases resulted in death.

Thus, posterior circular ischemic infarctions are different etiologically, heterogeneous in clinical course and with different outcomes.

The results of our study indicate that the MRI technique is sensitive in detecting acute ischemic posterocircular strokes.

However, it did not always allow visualization of acute lacunar infarction or ischemic foci in the brain stem, especially in the medulla oblongata. To identify them, the diffusion-weighted MRI technique was more informative.

The sensitivity of DWI in detecting acute brainstem infarction up to 24 hours after the onset of stroke was 67%; the infarct focus was not detected during this time in 33% of patients, i.e. One third of those examined with clinical symptoms of brainstem infarction had false negative results. Repeated examinations of patients after 24 hours using DW MRI of the brain revealed the infarcted area.

The insufficient information content of the DWI technique in determining acute infarction when localized in the brain stem can be explained by two factors. Firstly, the presence of small ischemic foci, since the perforating arteries vascularize very small areas of the brain stem. Secondly, neurons of the brain stem are more resistant to ischemia than neurons of the evolutionarily younger cerebral hemispheres. This could be one of the reasons for their higher tolerance to ischemia and the later development of cytotoxic edema of brain stem tissue (Toi H. et al., 2003).

Conclusion

1. Lacunar infarcts in the pons and thalamus much observed. In patients with lacunar infarcts in which the internal capsule was damaged along with the thalamus, complete recovery took slightly longer than in other affected areas.
2. Movement disorders in lacunar infarctions occur as a result of damage to the motor pathways in the pons area.
3. In all patients, the clinical and neurological condition of posterior circular infarcts caused by damage to large arteries (vertebral and main) was accompanied by visual field defects, movement, sensation, bulbar syndrome, and coordination disorders. It has been established that blood pressure in patients with infarctions due to occlusion of large arteries is lower than in infarcts of small branches of the vertebrobasilar region.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open the publication.

Availability of data and material – Available.

Competing interests – No.

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