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CEREBRAL VENOUS HEMODYNAMICS PECULIARITIES IN PATIENTS WITH CHRONIC CEREBRAL ISCHEMIA OF VERTEBROGENIC ORIGIN

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The purpose of the study was to evaluate both venous hemodynamics and cerebrovascular reactivity in patients with chronic cerebral ischemia of vertebrogenic nature. 66 patients with vertebrogenically caused chronic cerebral ischemia were randomized into clinical group I with compensated chronic cerebral ischemia and group 2 with subcompensated chronic cerebral ischemia. Relatively healthy people composed the control group. Cerebral veins were examined using ultrasound with vertebral vein maximum blood flow velocity, Rosenthal basal veins, and direct sinus blood flow determination. Cerebral venous reactivity together with pain intensity were also established. Cephalalgia, mainly combined with cervicalgia, was the most significant clinical symptom in patients with chronic cerebral ischemia. Patients with subcompensated chronic cerebral ischemia demonstrated a significant increase in venous blood flow through vertebral veins and direct sinus. The investigated patients also revealed significant hyporeactivity. Significant hyperreactivity to antiorthostatic load in the direct sinus was registered in both groups of patients. The data obtained revealed that cervicogenic headache is one of the most common clinical syndromes in patients with chronic cerebral ischemia of vertebrogenic origin. The clinical manifestation of chronic cerebral ischemia is accompanied by venous blood flow in the vertebral veins and a significant increase in direct sinus, with an emphasis in subcompensated patients. The authors conclude that hyperreactivity to antiorthostatic loading in the direct sinus is the main pattern of cerebral venous reactivity in patients with chronic cerebral ischemia, being clinically coupled with a slight presence of intracranial hypertension. They showed that cerebral venous reactivity indices are important for early and effective diagnosis of the studied pathology, as well as for assessing the success and efficacy of the provided complex treatment.

Key words: chronic cerebral ischemia, cervicogenic headache, venous hemodynamics, triplex scanning, pathophysiological mechanisms, cerebral venous reactivity.

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ОСОБЛИВОСТІ ЦЕРЕБРАЛЬНОЇ ВЕНОЗНОЇ ГЕМОДИНАМІКИ У ПАЦІЄНТІВ ІЗ ХРОНІЧНОЮ ІШЕМІЄЮ МОЗКУ ВЕРТЕБРОГЕННОГО ПОХОДЖЕННЯ

Метою дослідження була оцінка стану венозної гемодинаміки та венозної цереброваскулярної реактивності у пацієнтів з хронічною ішемією мозку вертебродгенного характеру. Обстежено 66 пацієнтів із вертебродгенно зумовленою хронічною ішемією мозку, яких розподілено на клінічну групу 1 – з компенсованою хронічною ішемією мозку, та клінічну групу 2 – з декомпенсованою хронічною ішемією мозку. Відносно здорові люди склали контрольну групу. Церебральні вени у пацієнтів досліджували за допомогою ультразвукового методу з визначенням максимальної швидкості кровотоку в хребетних венах, базальних венах Розенталя та прямому синусовому кровотоку. Визначали також реактивність церебральних вен разом з інтенсивністю болю. Цефалалгія, яку в більшості випадків реєстрували разом із цервікалгією, була найвиразнішим клінічним симптомом у пацієнтів із хронічною ішемією головного мозку. У пацієнтів із субкомпенсованою формою хронічної ішемії головного мозку реєстрували значне збільшення венозного кровотоку в хребетних венах і прямому синусі. У досліджуваних пацієнтів також виявлено виражену гіпореактивність. В обох групах пацієнтів зареєстровано значну гіперреактивність у прямому синусі на антиортостатичне навантаження. Отримані дані довели, що цервікогенний головний біль є одним із найпоширеніших клінічних синдромів у пацієнтів із хронічною ішемією головного мозку вертебродгенного походження. Клінічний прояв хронічної ішемії головного мозку супроводжується суттєвим збільшенням венозного кровотоку в хребетних венах та прямому синусі, що реєструється в найбільшому ступені у пацієнтів із субкомпенсованою формою захворювання. Автори зазначають, що гіперреактивність при антиортостатичному навантаженні у прямому синусі є основною закономірністю реактивності вен головного мозку у пацієнтів із хронічною ішемією головного мозку, клінічно поєднуючись із незначною внутрішньочерепною гіпертензією. На їхню думку, використання індексів реактивності вен головного мозку є важливим для забезпечення ранньої та ефективної діагностики досліджуваної патології, а також для оцінки успішності та ефективності проведеного комплексного лікування.

Ключові слова: хронічна ішемія мозку, цервікогенний головний біль, венозна гемодинаміка, триплексне сканування, патофізіологічні механізми, церебральна венозна реактивність.

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The cervical spine (CS) is the most active, loaded, and mobile region; it carries the main arteries and nerves and contains the spinal cord and brainstem [10, 12]. It is the least protected from external influences (trauma, certain dystrophic lesions of the bone and cartilage apparatus, their combinations, etc.).

The pain syndrome in the form of cephalalgia, cervicocranial pain, and dorsalgia dominates the last decades; the prevalence of vertebrogenic pain in industrialized countries equals 60–80 % [2] and is constantly growing and ranks fourth in terms of disability [7, 9].

Pathomorphological degenerative-dystrophic (DD) changes in the spine in most cases can explain cervical pain syndrome with muscle spasm, amplification, including due to vascular compression with subsequent circulatory disorders, both regional and cerebral [6]. Reflex vegetative and vascular syndromes involve spasm/dystonia of the vessels of the head and neck, changes in the reactivity of the autonomic system, hypodynamia, insufficient physical activity, as well as constitutional features against the background of dysfunction of the central, peripheral, vegetative parts of the nervous system [1].

Cervicogenic headache (CH) is known to occur as a result of irritation of structures innervated by the spinal nerves C_I, C_{II} and C_{III} [15]. Cervicogenic headache, as a rule, has a unilateral cervicoccipital localization and is clearly associated with movements in the cervical spine. This pain often radiates from the back to the front of the head, may be in the face, is aggravated by certain head movements, positions, or at night, when the head is in an uncomfortable position for a long time, and is accompanied by a limitation of the range of motion in the neck.

According to the vertebral artery (VA) extracranial segment location peculiarities inside the CS mobile bone canal, a VA syndrome occurs. Both sympathetic nodes and perivascular autonomic plexuses are involved in this syndrome manifestation [15]. The VA trunk, together with autonomic plexus compression and reflex spasm, provides reduced blood flow to the back parts of the brain with subsequent cerebral ischemia [8, 11]. The abovementioned disorders result in chronic cerebral ischemia (CCI) gradual development.

The cervical lordosis straightening and intervertebral disc protrusion, as well as spinal motor segments at the cervical level, are the most common neuroimaging patterns in CCI patients [6, 15]. According to our data, a correlation was noted between the severity of vertebral dysfunction and cerebral dysgemia in CH. We suppose these events are due to decreased vasomotor reactivity inside the vertebrobasilar basin (VBB) arteries together with impaired vasomotor reactivity and cerebral blood flow autoregulation [8, 11]. However, the pathogenetic importance of vascular factors, hemodynamic patterns, and comparisons of hemodynamic characteristics across headache types remain undetermined. These circumstances require clarification of the pathogenesis using Doppler imaging.

Thus, the CCI development, especially in VBB, is facilitated by many factors associated not only with general hemodynamic harm against the background of arterial hypertension, atherosclerosis, diabetes mellitus, heart disease, and vasculitis but also by vertebrogenic factors caused by congenital and acquired changes inside the cervical vertebrae craniovertebral transition zone [13].

The purpose of the study was to evaluate both venous hemodynamics and cerebrovascular reactivity

in patients with chronic cerebral ischemia of vertebrogenic nature.

Materials and methods. 66 patients with vertebrogenically caused CCI (40 men and 26 women) aged between 35 and 60 years (average age equal to 43.3 ± 1.9 years) were examined. These patients were randomized into Group 1 (n=30) with CCI in the compensated stage and Group 2 (n=36) with CCI in the subcompensated stage. The control group (CG) included 20 relatively healthy people of the corresponding age.

All personal information gathered during this study was processed in compliance with the European Parliament and Council's Regulation EU 2016/679 of 27 April 2016 on the protection of natural persons concerning the processing of personal data and on the free movement of such data (General Data Protection Regulation). The research was conducted as part of the GAJU 066/2022/S project, approved by the Ethics Committee No. 6/2022. The study followed the basic bioethical norms of the World Medical Association Declaration of Helsinki on Ethical Principles (amended in 2008), the General Declaration on Bioethics of the Convention on the Rights of Man, and Biomedicine (1997). Patients were informed of the objectives of the study, the aims of their intensive ultrasound imaging and other types of clinical examination, together with the potential use of the data collected in these clinical examinations for scientific research. We kept personal data confidential, obtained informed consent, and participation in the study was voluntary.

Degeneration of bone and cartilage structures in CS was confirmed by neuroimaging methods, by clinical examinations and anamnesis, as well as using a questionnaire of the functional state of a patient with neck pain syndrome and the degree of his disability (restrictive activity) – the Neck Disability Index (NDI) [9].

The neck pain syndrome duration was indicated anamnestically in the range from 6 months to 5 years (average duration 2.5 ± 0.2 years). Pain syndrome intensity was evaluated using a 10-point visual analog scale (VAS) [4].

Ultrasound investigation of the vessels was performed using an "Ultima-PA" ultrasound scanner ("RADMIR", Ukraine). Triplex mode examination was used to investigate the maximum blood flow velocity (V_{max}) in the vertebral veins (VV), basal veins of Rosenthal (BV), and direct sinus (DS).

A study of veins was performed with the study of maximum blood flow velocity (V_{max}) in the vertebral veins (VV), basal veins of Rosenthal (BV), and straight sinus (RS). To assess cerebral venous reactivity, we used the reactivity coefficients determination for ortho- (CrvvOL) and antiorthostatic (CrvvAOL) loads in VV, ortho- (CrbvOL) and antiorthostatic (CrbvAOL) loads in the BV, ortho- (CrdsOL) and antiorthostatic (CrdsAOL) loads in the DS.

The data obtained were statistically analyzed using the parametric ANOVA criterion and the nonparametric Kruskal-Wallis criterion. $p < 0.05$ was accepted as the index of significant differences.

Results of the study. All examined patients had pain syndromes. First of all, this refers to pain in the cervical localization as a DD manifestation of CS disorders. According to NDI, all patients with CCI

had indexes on the border of mild and moderate disability equal to 13.6 ± 3.8 points.

Cervical pain prevailed in 61 (92.4 %) patients, pain in the upper back was registered in 31 (46.9 %) patients, and complaints of its irradiation to the upper extremities were detected in 29 (43.9 %) patients. Pain intensity ranged from 1 to 5 points; the average score was equal to 4.4 ± 0.5 points (Table 1).

Table 1

The indexes of pain syndromes and hemodynamic parameters (Vmax) in cerebral veins in patients with CCI

	All examined patients		
	Group 1, n=30	Group 2, n=36	Control group, n=20
Pain syndromes indexes			
Cervicogenic headache	$\frac{30^{***}}{100.0}$	$\frac{35^{***}}{97.2}$	$\frac{2}{10}$
Cervicalgia	$\frac{28^{***}}{93.3}$	$\frac{35^{***}}{97.2}$	$\frac{1}{5.0}$
Pain in the shoulder, back, limb	$\frac{15^{**}}{50.0}$	$\frac{16^{**}}{44.4}$	$\frac{1}{5.0}$
Irradiation	$\frac{14^{*}}{46.7}$	$\frac{15^{*}}{41.7}$	$\frac{1}{5.0}$
VAS, points	$5.1 \pm 0.8^{**}$	$3.8 \pm 0.2^{*}$	0.8 ± 0.1
Cerebral veins hemodynamic parameters			
VV	24.8 ± 4.2	$37.3 \pm 4.5^{*}$	20.8 ± 3.2
BV	21.7 ± 3.6	25.1 ± 3.8	18.4 ± 4.6
DS	32.3 ± 3.6	$39.8 \pm 4^{*}$	25.5 ± 4.6

Notes: the numerator indicates the absolute numbers, the denominator indicates relative numbers; * – $p < 0.05$, ** – $p < 0.01$, *** – $p < 0.001$ – statistical differences of the investigated index compared with the same in practically healthy patients.

Cephalalgia was observed in 65 (98.5 %) patients and was mainly combined with neck pain. Unilateral cervicocapital headache with irradiation from the back to the front of the head, which intensified with certain head movements or positions, including at night, was recorded in 50 (75.8 %) patients. Restrictions on the volume of movements in the neck were recorded.

Headache was practically absent in the control group – only 2 patients (10.0 %, $p < 0.05$) had complaints.

In patients with CCI, transcranial duplex scanning was used to study the velocity parameters of blood flow in the cerebral veins, cerebral arteries, and cerebral plexuses, as well as the reactivity coefficients in the straight sinus during antiorthostatic functional loading. We investigated blood flow parameters thoroughly in the main cerebral venous collectors in patients with CCI (Table 1).

In Group 1, BV blood flow parameters did not differ substantially from the analogous data in the control group (21.7 ± 3.6 cm/s vs 18.4 ± 4.6 cm/s). The VV (24.8 ± 4.2 cm/s vs 20.8 ± 3.2 cm/s) and DS (32.3 ± 3.6 cm/s vs 25.5 ± 4.6 cm/s) velocity parameters were enhanced. In Group 2 patients, a moderate increase in Vmax in the BV was noted (25.1 ± 3.8 cm/s vs 18.4 ± 4.6 cm/s). A significant increase in velocity parameters was recorded in DS (39.8 ± 3.4 cm/s vs 25.5 ± 4.6 cm/s; $p < 0.05$). Group 2 patients also revealed a significant blood flow increase in the

cerebral vessels (37.3 ± 4.5 cm/s vs 20.8 ± 3.2 cm/s; $p < 0.05$).

The most demonstrative differences between the groups (Group 2 vs Control group) were noted in DS and VV (Fig. 1).

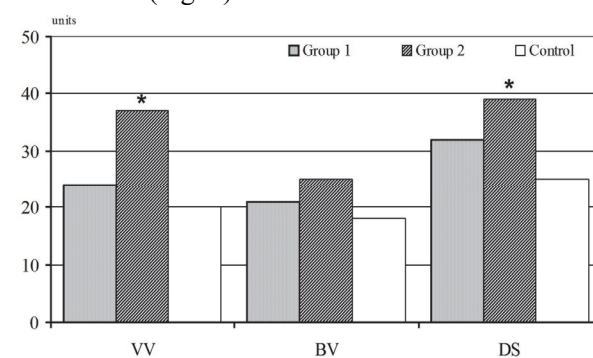


Fig. 1. Vmax ratio in cerebral veins in patients with cerebral chronic ischemia. Notes: * – $p < 0.05$ – statistical differences of the investigated index compared with the same in practically healthy patients (Kruskal-Wallis test).

We registered hyporeactivity in the cerebral vessels in examined patients with CCI in orthostatic test (Group 1 – 1.55 ± 0.06 vs control -1.88 ± 0.06 ; Group 2 – 1.46 ± 0.05 vs control -1.88 ± 0.06 , in both cases $p < 0.05$). Hyporeactivity was more expressed in Group 2 patients.

Cerebral venous reactivity in response to the AOL test showed cerebral vessel hyporeactivity. Cerebral vessels' response to the AON test slightly exceeded control indexes in both clinical groups.

The evident hyperreactivity in response to AOL was registered in DS (Group 1 – 1.48 ± 0.04 vs

1.31 ± 0.04 ; Group 2 – 1.51 ± 0.03 vs 1.31 ± 0.04 , in both cases $p < 0.05$; Table 2).

Table 2

Cerebral venous reactivity indexes in patients with CCI

Clinical groups	The investigated ultrasound indexes, $M \pm m$, cm/s					
	CrvvOL	CrbvOL	CrdsOL	CrvvAOL	CrbvAOL	CrdsAOL
Group 1, n=30	$1.55 \pm 0.06^*$	0.29 ± 0.04	0.31 ± 0.04	0.18 ± 0.05	1.37 ± 0.04	$1.48 \pm 0.04^*$
Group 2, n=36	$1.46 \pm 0.05^*$	0.31 ± 0.04	0.28 ± 0.03	0.20 ± 0.05	1.36 ± 0.06	$1.51 \pm 0.03^*$
Control group, n=20	1.88 ± 0.06	0.28 ± 0.05	0.29 ± 0.05	0.27 ± 0.04	1.29 ± 0.05	1.31 ± 0.04

Notes: * – $p < 0.05$ – statistical differences of the investigated index compared with the same in practically healthy patients.

The most significant factor of decompensation in patients with CH was the increase in the maximum blood flow velocity (Vmax) in the VV (78.3 %), the maximum blood flow velocity (Vmax) in the DS (60.9 %) and BV (52.2 %), as well as hyperreactivity to antiorthostatic load (AOL) in the DS (60.9 %). The latter we supposed to be as an autoregulatory pattern in CH, which suggests the presence of subclinical intracranial hypertension of a venous nature in these contingent patients.

Discussion. Therefore, the data obtained revealed impaired venous blood flow and cerebral vein reactivity in the prevalent majority of CCI patients. Our data align with studies by several authors who have shown a relationship between cephalalgias, including those of vertebrogenic origin, and chronic venous dyscirculatory syndrome [5]. This is due to the characteristics of the venous wall, the presence of muscle fibers, valves, the dependence of venous pressure in the sinuses on other vascular basins, impaired autonomic regulation, etc. [3].

To discuss this data item, one should focus on cephalgia itself and related pain types, including cervicogenic headache. Cervicogenic headache is pain that is felt in the head, but is caused by pathology of the cervical spine. According to experimental data, the sources of such pain can be the upper cervical synovial joints (atlanto-occipital, lateral atlantoaxial, and arcuate processes C_{II}-C_{III}). Intra-articular injection of local anesthetics into the lateral atlanto-axial joint, blockade of the third occipital nerve, or facet joint C_{II}-C_{III} leads to relief of the pain syndrome [11, 15].

The mechanisms of cerebral spine degenerative-dystrophic changes and pain development are the second item to discuss when analyzing our data. It is that the above-named emerging hypoxic factor closes a vicious circle with an impact on DD changes development in CS and therefore can be considered one of the leading in their pathogenesis. This circumstance requires further study, especially the effects of DD changes in the CS on the development of a number of pathological manifestations of insufficiency in VBB, and the possible development of ischemic type strokes in this basin, which are the most severe, since they affect the brainstem, brain, and thalamic region.

Third, we suppose it is important to understand the necessity and rationale for determining vegetative changes in examined patients. While DD changes in the

spine, especially in the cervical region, are common, vertebral artery syndrome is frequent, given the location of the vertebral artery's extracranial segment within the mobile bone canal of the cervical spine. Sympathetic nodes and perivascular vegetative plexuses are affected due to minimal blood perfusion in such conditions [15]. In conditions of vertebral artery trunk compression along with vegetative plexus reflex spasm, blood flow to the back parts of the brain decreases even more, which we consider to be a secondary pathogenetic chain of CNS ischemia development [8, 11, 14].

Finally, we should explain the interaction between venous cerebral hemodynamics and cervicogenic cephalalgia pathogenesis. Thus, in patients with CCI, Vmax in cerebral circulation and a significant increase in CrdsAOL were observed, reflecting signs of subclinical intracranial hypertension [5]. The venous link in cerebral circulation, combined with disturbances in cerebral venous regulation mechanisms, is thought to be a leading pathogenetic factor in headache, with increases in both frequency and intensity [6]. Both cerebral circulation and DS blood flow changes are, to a greater extent, markers of intracranial hypertension of venous nature [8, 15].

Venous dyscirculation regression after treatment demonstrates the reliability of Vmax of the cerebral circulation and Vmax of DS indexes for evaluating the degree of venous dysfunction and monitoring the efficacy of venous outflow disorder treatment. The potential of cerebral venous reactivity indexes for diagnostic use in venous dyscirculation was also proved by our previous clinical studies.

Analysis of reactivity indexes to static loads in CCI demonstrated hyporeactivity to OL and AOL in HV as the leading autoregulatory pattern in this type of headache. These indicators indicate a violation of the mechanisms of regulation of venous blood flow. The pronounced hyperreactivity to AOL in both groups indicated the presence of subclinical intracranial hypertension of venous origin, which may be one of the pathogenetic factors in the development of headache in cervical spine pathology.

The results showed the need for cerebral venous outflow regulation mechanisms, particularly using cerebral venous reactivity indexes to enable early and effective diagnosis of the pathology studied, as well as

to assess the success and efficacy of the complex treatment provided.

Limitations. We did not include in our venous blood flow and pain expression clinical examinations patients with cerebral chronic ischemia who had expressed physical, cognitive, and medical

limitations which in fact negatively influenced their daily survival and long-term recovery. We also excluded those patients who demonstrated both vestibular and vegetative disturbances in response to ortho- and antiorthostatic loads while checking their reactivity.

Conclusion

Cervicogenic headache is one of the most common clinical syndromes in patients with chronic cerebral ischemia of vertebrogenic origin, most often in combination with cervicgia.

Patients with cervicogenic headache showed a significant increase in venous blood flow in the vertebral veins and the direct sinus, with the greatest increase in patients with the subcompensated stage of chronic cerebral ischemia.

Hyperreactivity to antiorthostatic load in the straight sinus is the main pattern of cerebral venous reactivity in patients with chronic cerebral ischemia, which is associated with the presence of subclinical intracranial hypertension.

Cerebral venous reactivity indexes are important to ensure early and effective diagnosis of the pathology studied, as well as to assess the success and efficacy of provided complex treatment.

Prospects for further research include venous cerebral hemodynamics indexes and additional studies in patients with cervicogenic headache, which prove the importance of intracranial venous hypertension in headache pathogenesis. Further studies using venous cerebral hemodynamics and venous cerebrovascular reactivity indexes should demonstrate the efficacy of drug and non-drug therapy in cervicogenic headache treatment.

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