

ORIGINAL ARTICLE

THE CHARACTERIZATION OF HEADACHE IN PATIENTS WITH DYSIRCULATORY ENCEPHALOPATHY OF DIFFERENT GENESIS

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ABSTRACT

The aim of the study was to study the headache features in patients with dyscirculatory encephalopathy of different genesis.

Materials and methods: Clinical-neurological and clinical-instrumental examination of 90 persons aged 40 to 68 was performed. The first group consisted of 60 patients with dyscirculatory encephalopathy and arterial hypertension (DE and AH), the second group – 30 patients with dyscirculatory encephalopathy and cerebral atherosclerosis (DE and CA).

Results: In the study of headache in patients with DE + AH and DE + CA, the frequency of detection, the intensity on the VAS scale, and the nature of the headache, no significant difference was found between study groups.

Conclusions: According to the results of the study, it was proved that patients with DE + CA had headache in the root area, with the circumstances of headache being significantly outweighed «for no apparent reason» ($p = 0.007$) and with changing weather conditions ($p = 0.001$). Arterial hypertension was a major factor in headache ($p = 0.008$) and in patients with DE + AH.

KEY WORDS: dyscirculatory encephalopathy, hypertension, cerebral atherosclerosis, headache, visual-analogue scale VAS

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INTRODUCTION

Pain is an unpleasant sensory and emotional experience that is associated with existing or potential tissue damage, or is described by the patient in terms of such damage (International Association for the Study of Pain – IASP, 1979). Responding to the same painful irritation can vary greatly depending on the genetic predisposition, cultural traditions of the patient, age, gender, and some other factors. Pain occurs in various neurological, somatic and surgical diseases (vascular, inflammatory, tumors, traumas, reflex pain syndrome, polyneuropathy, etc.) and, accordingly, significantly impairs the quality of life of the patient. According to W.N. Cordell et al. [1], pain is the cause of referrals for patients in 52% of all emergency care cases, including primary care. Pain leads to a significant decrease in quality of life in a wide range of diseases. According to the WHO, the most common pain syndromes include headache, neck and back pain, and joint pain. Chronic spine pain ranks first in the prevalence of painful working-age syndromes (35-45 years) [2,3]. It is traditionally believed that 80% of patients with acute pain are fully recovered, only 17-20% of the pain persists. Many studies have shown that 40% of patients with an episode of acute pain continue to experience lower back pain for 6 months, in 62% of cases recurrence occurs within a year. In 5-7% of patients there is a persistent disability due to pain [4]. In clinical practice there are two types of pain – acute and chronic.

Acute (rapid) pain is signaled (danger signal) and caused by nociceptive effects: the sensation of pain occurs approximately 0.1 s from the onset of the pain stimulus (alternative names: piercing, prickly, electrical pain). All pain receptors (nociceptors) are free nerve endings. They are more common in the superficial layers of the skin, as well as in some internal tissues, such as the periosteum, arterial walls, articular surfaces, and skull bones.

Chronic pain differs from acute pain not only in duration but also in pathogenesis, clinical manifestations, treatment approaches and prognosis. Chronic pain changes the clinical picture, appears depressive disorders, decreased performance, disrupted sleep, and reduced quality of life. Increased life expectancy in advanced economies, the accumulation of an aging population, hypodynamia are part of the causes of the formation of a group of people with chronic pain syndrome, a component of which is neuropathic pain. The formation is caused by the lesion of the somato-sensory system due to involvement in the pathological process of peripheral nerve fibers (A and C – fibers) and central neurons. Neuropathic pain is present in 7-10% of the population in the general population [3,5]. Chronic neuropathic pain is more common in women (8% vs. 5.7% in men) and patients > 50 years of age (8.9% vs. 5.6% of people <49 years old) [6]. Among women, the prevalence of pain remains higher in all age groups than in men. Sex

Table I. Frequency of detection of individual comorbid conditions in the studied patient groups.

Concomitant pathology	AH+DE N=60	DE+CA N=30
Diabetes mellitus type 2, n (%)	10 (16)	0
Anemia, n (%)	1 (2)	2 (6)
Chronic coronary syndrome, n (%)	19 (31)	1 (3)
Cardiac arrhythmias, n (%)	1 (2)	0
Aortic valve insufficiency, n (%)	1 (2)	0
Hypothyroidism, n (%)	3 (5)	7(23)

Table II. Characterization of major syndromes of patients with DE + AH and DE + CA

Syndromes	AH + DE, n=60		DE+ CA, n=30		p
	a6c.	%	a6c.	%	
Cephalic	48	80	23	77	0,786
Vestibular	33	55	13	43	0,372
Atactic	5	8	0	0	0,165
Cerebrostenic	19	32	14	47	0,174
Anxiety-depressive	14	23	6	20	0,794
Cognitive	17	28	7	23	0,801
Pyramidal insufficiency	6	10	6	20	0,204

hormones alter the response to a painful stimulus, which may indirectly lead to different perceptions of pain for men and women [7]. Psychologically, women differ in the cognitive and emotional processes that accompany pain, and have differences from men in their pain behaviors.

The mechanism of pain is due to the expression of Na channels on nerve fibers, reflects the process of compensatory neuroplasticity in response to nerve or root damage, leads to excess ectopic impulsion, directed to the posterior horn and causes its hyperactivity. Discharges of altered nociceptive fibers contribute to the release of excitatory amino acids and neuropeptides in the posterior horn, perform depolarization of postsynaptic membranes, which opens potential-dependent Ca-channels in the neurons of the horn, release of Ca into the cell, release the cells. Hyperactivity of nociceptive neurons of the posterior horn while reducing inhibitory GABA – influence exerts prerequisites for the violation of the regulatory influence on the segmental (limbic) structures in which the focus of excitation is formed, and this provides the conditions for maintaining the sensation of pain after removal of the focus [6,7]. Stressful events prevent the onset of pain – one of the most important factors in chronic pain management. The imbalance of the mediator systems resulting from stress reactions and concomitant depression affects the antinociceptive systems, modifying the sensation of pain. Studies have shown that the link between depression and pain is two-way: pain increases the risk of depression, and depression can be the root cause of pain. Also, pain

often leads to anxiety and tension, exacerbates emotional disturbances, which in themselves enhance the perception of pain – a vicious circle arises. Chronic pain can lead to impaired psychological well-being, sexual health, decreased quality of life and social maladaptation [8,9]. Continuous, non-smoking pain, in contrast to acute pain, contributes to the formation of patients with psycho-emotional disorders, which, in turn, support the process of chronic pain [10].

In this regard, the relevance of this problem is not only of medical but also socio-economic importance and needs further investigation.

THE AIM

The aim of the study is to study the features of headache in patients with dyscirculatory encephalopathy of different origins.

MATERIALS AND METHODS

Under our observation, there were 90 people aged 40 to 68 at the State Institution of Science «Research and Practical Centre of Preventive and Clinical Medicine» State Administration Department, Kyiv, Ukraine. Patients with dyscirculatory encephalopathy were divided into two groups statistically comparable by major disease, sex, and age. The first group consisted of patients with dyscirculatory encephalopathy (DE) and arterial hypertension (AH) – 60 people, the second one with DE without AH (on the background of cerebral atherosclerosis) – 30. Among the examined patients in the first group were 28 men and 32 women. In the second group – 12 men and 18 women. The mean age of men in the first group was 51.54 ± 0.76 , in the second 51.83 ± 2.24 , women in the first group 54.63 ± 0.42 , in the second 56.88 ± 0.72 . Clinical-neurological and clinical-instrumental examination was performed for all patients in order to establish the stage and form of vascular-brain pathology.

The study did not include patients with severe somatic pathology, uncompensated somatic diseases, pregnancy, patients with symptomatic hypertension, malignant lesions of the brain or other facet, hypertensive encephalopathy, or acute cerebrovascular disease, history of traumatic brain injury.

Clinical and laboratory study included general blood test, biochemical blood test, lipidogram. Clinical and instrumental examination methods included electrocardiography, blood pressure measurements, and heart rate.

Intensity of pain was assessed using the VAS-analog scale, where «0» means no pain and «10» is unbearable pain.

Statistical processing of the data obtained was carried out on a personal computer. Although the distribution of the obtained data differs from the normal one, nonparametric analysis methods were used. A statistically significant difference was considered at $p < 0.05$.

RESULTS

On the basis of the complaints, the anamnesis data, according to the results of clinical and instrumental examination,

Table III. Nature of headache among patients in the study groups (incidence rate of headache cases)

The nature of the pain	DE+AH N=48	DE+CA N=20	p
Diffuse, n(%)	5 (10)	4 (20)	0,432
Dull, n(%)	24 (50)	11 (55)	0,793
Clustered, n(%)	2 (4)	3 (15)	0,147
One sided, n(%)	7 (16)	5 (25)	0,316
Shingle, n(%)	3 (6)	1 (5)	1,000
Squeezing, n(%)	25 (52)	12 (60)	0,602

Note. The same patient could have noted several variants of the nature of the pain.

Table IV. Localization of headache among patients in the study groups (incidence rate of headache cases).

Pain localization	DE+AH N=48	DE+CA N=20	p
Frontal area, n(%)	23 (48)	14 (70)	0,115
Temporal area (areas), n(%)	22 (46)	17 (85)	0,003
Occipital area, n(%)	17 (35)	11 (55)	0,179
Timian area (areas), n(%)	12 (25)	8 (40)	0,251

Note. The same patient could have noted several variants of the nature of the pain.

Table V. Headache circumstances and associated conditions among patients in the study groups (incidence of headache cases)

Circumstances and associated conditions	DE+AH N=48	DE+CA N=20	p
Mental load, n(%)	10 (21)	8 (40)	0,134
Exercise, n(%)	8 (17)	5 (25)	0,503
Normal BP, n(%)	0	2 (10)	0,083
Increased BP, n(%)	24 (50)	3 (15)	0,008
For no apparent reason, n(%)	9 (19)	11 (55)	0,007
Changes to weather conditions, n(%)	2 (4)	15 (75)	<0,001

Note. The same patient could provide several options for answering questions.

90 patients were diagnosed with symptom complex, which corresponds to the criteria for the diagnosis of cerebral insufficiency, among patients of the first group in 19 (31.7%) were diagnosed with I stage of dyscirculatory encephalopathy (DE) and 41 (68.3%) of DE stage II. In the second group of patients, 12 (40%) were diagnosed with stage I DE and 18 (60%) – stage II DE.

AH was observed in all patients in the first group. According to the above classification, 8 (13.3%) patients had arterial hypertension stage I and 52 (86.7%) – stage II of arterial hypertension.

Among the surveyed patients were reported such concomitant conditions as chronic coronary syndrome, diabetes mellitus type 2, cardiac arrhythmias, aortic valve

insufficiency, hypothyroidism, anemia. The frequency of detection of individual comorbid conditions in the study groups is shown in Table I.

According to the evaluation of subjective and objective neurological symptoms, patients of the first group had 80% cephalic, 55% vestibular, 32% cerebrostenic, 28% mystic syndromes and 23% had anxiety-depressive disorders. Among the patients in the second group, 77% had cephalic, 43% vestibular, 47% cerebrostenic, 23% mystic syndromes, 20% had anxiety-depressive disorders and 20% had pyramidal insufficiency. No significant difference was found between the study groups (Table II).

The incidence rate of headache patients in the comparison groups was 48 (80%) in the DE + AH group versus 20 (67%) in the DE + CA group ($p = 0.197$). Intensity of headache, on a VAS scale (among persons with headache) (median, interquartile interval): 5 (3-6) points in group DE + AH versus 5 (3-6) points in group DE + CA ($p = 0.724$).

The nature of the headache in patients in both groups was predominant: blunt (50% in patients in the first group and 55% in patients in the second group), squeezing 52% in patients in the first group and 60% in patients in the second group) headache, but a significant difference between no study groups were identified (Table III).

Headache in patients of (DE + AH) and (DE + CA) patients was localized mainly in the frontal and temporal areas, and less frequently in the occipital area. Significantly significant difference ($p = 0.003$) was observed for localization in the root region in patients with DE + CA (Table IV).

In the circumstances of the occurrence of headache, patients with DE + CA significantly outweighed «for no apparent reason» ($p = 0.007$) and with changes in meteorological conditions ($p < 0.001$), in patients with DE + AH with elevated blood pressure ($p = 0.008$). The circumstances of headache among patients in the study groups are presented in Table V.

Thus, when analyzing the frequency of occurrence of individual syndromes, we came to the conclusion that patients of both groups were dominated by cephalic, vestibular, and cerebrostenic syndromes, but no significant difference was found between the study groups.

DISCUSSION

The data obtained by us coincide with the results of the researches of O. M. Treshchinska, E. I. Gusev, O. M. Konovalova, V. I. Skvortsova, I. Yu. Head. In their publications, the authors note that patients with DE mostly report complaints and focus on such subjective manifestations as headache, dizziness, tinnitus, rapid fatigue, emotional lability, etc., but pain syndromes should be recognized as prevalent in clinical settings. the picture of DE, which overwhelmingly determines the severity of patients [11,12,13].

The cause of the headache can be both spasm and enlargement of the arteries, as well as insufficient venous outflow, slowing blood flow [6]. Particular attention should be paid to the opinion of some authors that patients with headache with AH seek help in the second phase – the

phase of paralytic expansion of extracerebral vessels, when there is excessive stretching of extracerebral arterial walls with increased pressure, which increases the amplitude of the amplitude of the amplitude. pain nerve receptors in the artery wall [7].

In the study of headache in patients with DE + AH and DE + CA, the frequency of detection, the intensity on the VAS scale, and the nature of the headache, no significant difference was found between study groups. Significantly significant difference ($p = 0.003$) was observed in localization in the rooted area in patients with DE + CA and in the circumstances of headache among patients with DE + CA significantly outweighed «without obvious reasons» ($p = 0.007$) and when changing meteorological conditions ($p = 0.007$), and in patients with DE + AH with increasing blood pressure ($p = 0.008$). The results obtained may be due to the fact that an important concept of pain is that pain is the result of a dynamic interaction of biological, psychological and socio-cultural factors.

At different stages of the disease, the proportion of different factors may vary. If biological (anatomical, genetic, physiological) factors predominate in the acute phase of illness, then psychological (affective, cognitive, behavioral) and social (gender, national traditions) factors may come to the fore. Biological factors can initiate, support and modulate physical disorders, whereas psychological changes affect the assessment and perception of internal physiological signals. In turn, psychological factors affect biological, altering the production of hormones, neurotransmitters, the state of the autonomic nervous system and biochemical processes in the brain [1,14,15].

CONCLUSIONS

According to the results of the study, it was proved that patients with DE + CA had headache in the root area, with the circumstances of headache being significantly outweighed «for no apparent reason» ($p = 0.007$) and with changing weather conditions ($p = 0.001$). Arterial hypertension was a major factor in headache ($p = 0.008$) and in patients with DE + AH.

REFERENCES

1. Cordell W. H., Keene K. K., Giles B. K. et al. The high prevalence of pain in emergency medical care. *Am. J. Emerg. Med.* 2002; 20(3): 165–169.
2. Kukushkin M. L., Hitrov N. K. Obshchaya patologiya boli [General pathology of pain]. *Medicine*. 2004; 141 p. (Ru)
3. Habirov F. A. Klinicheskaya nevrologiya pozvonochnika [Clinical neurology of the spine]. Kazan, 2001; 472 p. (Ru)
4. Colloca L., Ludman T., Bouhassira D. et al. Neuropathic pain. *Nat. Rev. Dis. Primtr.*, 2017; 3: 170–172.
5. Meana M. The meeting of pain and depression comorbidity in women. *Can. J. Psychiatry*. 1998; 43 (9): 893–899.
6. Bouhassira D., Attal N. Diagnosis and assessment of neuropathic pain: the saga of clinical tools. *Pain*, 2001; 152 (3 Suppl.): 74–83.
7. Bouhassira D., Lanteri-Minet M., Attal N. et al. Prevalence of chronic pain with neuropathic characteristics in the general population. *Pain*, 2008; 136 (3): 380–387.

8. Hains B.C., Saab C.Y., Klein J.P. et al. Altered sodium channel expression in second-order spinal sensory neurons contributes to pain after peripheral nerve injury. *J. Neurosci.* 2004; –Vol. 24: 4832–4839.
9. Meana M. The meeting of pain and depression: comorbidity in women. *Can. J. Psychiatry*. 1998; 43 (9): 893–899.
10. ter Kuile M.M., Weijnenborg P.T., Spinhoven P. Sexual functioning in women with chronic pelvic pain: the role of anxiety and depression. *J. Sex. Med.* 2010; 7(5): 1901–1910.
11. Nnoaham K.E., Hummelshoj L., Webster P. et al. World Endometriosis Research Foundation Global Study of Women's Health consortium. Impact of endometriosis on quality of life and work productivity: a multicenter study across ten countries. *Fertil. Steril.* 2011; 96(2): 366–373.
12. Gusev E.I., Konovalov A.N., Skvortsova E. I. Hronicheskaya nedostatochnost mozgovogo krovoobrashcheniya [Chronic insufficiency of cerebral circulation]. *Neurology: national leadership. M.*, 2011. (Ru)
13. Treshchinskaya M. A. Arterialnaya gipertenziya i cerebrovaskulnaya patologiya [Arterial hypertension and cerebrovascular pathology]. *Medicine and pharmacy news*. 2013; 30–35. (Ru)
14. Golovach I. Yu. Discirkulatornaya encefalopatiya: nekotoriye patogeneticheskiye, klinicheskiye i terapevticheskiye aspekti [Discirculatory encephalopathy: some pathogenetic, clinical and therapeutic aspects]. *Medicines of Ukraine*. 2011; 4: 60–67. (Ru)
15. Van Roenn G. X., Peys G. A., Preoder M. I. Diagnostika i lecheniya boli [Pain diagnosis and treatment]. M.: Binom, 2012; 494 p. (Ru)

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Conflict of interest:

The Authors declare no conflict of interest.

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