Neurology Asia 2023; 28(2) : 329 – 336

Frequency of cognitive dysfunction in individuals with cervicogenic headache

[1]Savaş KARPUZ *MD*, [1]Ramazan YILMAZ *MD*, [1]Emine AKDERE *MD*, [2]Ahmet KÜÇÜK *MD*, [1]Havva COP, [1]Halim YILMAZ

[1]Physical Medicine and Rehabilitation Clinic, [2]Neurology Clinic, Konya Beyhekim Training and Research Hospital, Konya Turkey

Abstract

Background: Both primary and secondary headaches have been associated with cognitive dysfunction and depression. *Objective:* This study aims to investigate the effects of cervicogenic headache on cognitive function, quality of life and mood. *Methods:* This single-center, cross-sectional comparative study includes 30 patients diagnosed with cervicogenic headache by a neurologist and 30 healthy controls. The pain level of the participants was evaluated with the Visual Analog Scale (VAS), their mood with the Beck Depression Inventory (BDI) and the Pain Catastrophizing Scale, their quality of life with the Short Form-36 (SF-36), and their cognitive status by a psychologist with the Standard Mini Mental Test (SMMT) and Montreal Cognitive Assessment Scale (MoCA). *Results:* All MoCA domains except for orientation and the SMMT scores were significantly lower in the cervicogenic headache group compared to the control group (p< 0,05). In addition, scores for the pain catastrophizing scale and selected SF-36 sub-parameters (physical function, physical role difficulty, social functionality, pain and general health perception) were significantly lower in the cervicogenic headache group (p< 0,05). The Beck Depression Inventory score was significantly higher in patients with cervicogenic headache than in the control group (p=0.018).
Conclusion: Patients with cervicogenic headache exhibit worse cognitive performance during their headache. Additionally, compared to healthy controls, they had higher rates of depression and pain catastrophizing as well as a lower quality of life.

Keywords: Cervicogenic headache, cognitive dysfunction; quality of life, mood

INTRODUCTION

Cervicogenic headache is a unilateral, chronic and recurrent non-throbbing headache caused by a nociceptive source in the cervical spine. It is a referred pain that occurs due to irritation of the cervical structures innervated by the upper cervical nerves (C1, C2, and C3), begins or exacerbates after neck movement, and is usually accompanied by decreased range of motion (ROM) in the neck.[1] It can be confused with migraine, tension-type headache, or other primary headache syndromes.[2] Cervicogenic headache occurs in 0.4% to 20% of those who suffer from headache.[3] The prevalence of cervicogenic headache is 2.2% - 4.1%[4,5] in the general population.

Cognition includes all mental abilities and processes related to knowledge, including but not limited to attention, memory, reasoning, comprehension, and language production. Cognitive dysfunction is a decrease in function in one or more cognitive domains, including attention and concentration, executive function, information processing speed, language, visuospatial ability, psychomotor ability and/or learning and memory.

In individuals with chronic pain, lower scores have been observed in all cognitive functions compared to healthy individuals, especially with respect to memory, executive function, verbal working memory, non-verbal working memory, and attention.[6-8] Cognitive dysfunction is increased in tension-type headache, cluster headache, and migraine.[9-14] However, we could not find any studies investigating the extent of cognitive dysfunction in patients with cervicogenic headache in the literature. We therefore aimed to investigate the frequency of cognitive dysfunction in patients with cervicogenic headache, as well as their associated quality of life and prevalence of depression.

Address correspondence to: Savaş KARPUZ, Physical Medicine and Rehabilitation Clinic, Konya Beyhekim Training and Research Hospital, Devlethane Sokak No:2/A, 42060 Selçuklu/Konya. Tel: +905552057860, E-mail: svskrpz@hotmail.com

Date of Submission: 17 January 2023; Date of Acceptance: 3 February 2023

https://doi.org/10.54029/2023rzc

METHODS

This study was designed as a cross-sectional study. Local ethics committee approval was obtained for the study protocol. The study included 30 patients diagnosed with cervicogenic headache by the same neurologist between December 2021 and December 2022, as well as 30 healthy controls over the age of 18 who came to the hospital as a accompanying person. The diagnosis of cervicogenic headache was made according to the criteria of the International Classification of Headache Disorders 3rd edition.[15] The study protocol was explained to participants and informed consent was obtained.

The following exclusion criteria were applied: Those with neurological deficits; Those with rheumatic diseases such as fibromyalgia, polymyalgia rheumatica, ankylosing spondylitis, and rheumatoid arthritis; Those with a history of surgery in the cervical region in the last six months; Those with widespread pain; Those with significant pain in another anatomical region (eg, gonarthrosis); Those who used drugs or substances (alcohol, drugs, etc.) increasing the risk of cognitive impairment; Those with neurological diseases such as known cerebrovascular disease, multiple sclerosis, Parkinson's disease, and dementia; Those with a major psychiatric disease and communication problems; Those who had received psychiatric medical treatment in the last three months; Those with significant hearing or vision problems; Those with a history of uncontrolled systemic disease (cardiovascular, pulmonary, hepatic, renal, hematological, endocrine).

The participants were evaluated with a detailed anamnesis and physical examination, and their socio-demographic characteristics (age, gender, educational status, body mass index (BMI)) were recorded. All evaluations in the cervicogenic headache group were performed during the active headache period.

Participants' pain levels were measured with the Visual Analogue Scale (VAS), mood levels with the Beck Depression Inventory (BDI) and Pain Catastrophizing Scale (PCS), quality of life with Short Form-36 (SF-36), and cognitive status with Standard Mini Mental Test (SMMT) and the Montreal Cognitive Assessment scale (MoCA) by the same psychologist.

Evaluation parameters

Visual Analogue Scale (VAS): The patient was asked to mark the most appropriate value corresponding to his/her pain on a 10 cm long scale divided into 10 intervals with a width of 10 mm, explaining that 0 represented "no pain" and 10 represented "the most severe pain".

Beck Depression Inventory (BDI): The Beck Depression Scale was developed by Beck in 1961. The Turkish version was validated by Hisli in 1988. The inventory consists of 21 items related to depressive symptoms such as pessimism, sense of failure, dissatisfaction, feelings of guilt, restlessness, fatigue, decreased appetite, indecision, sleep disturbance, and social withdrawal. Each item contains four graded (0-3) self-evaluation statements that determine a behavior specific to depression. The total maximum score is 63, and a score over 17 indicates an increased risk of a depressive state.[16]

Pain Catastrophizing Scale (PCS): This developed to assess the impact of pain. There are a total of 13 questions in the scale, which has three separate subscales (rumination, magnification, and helplessness). Each question is scored from 0-4 points, and the total score can range from 0-52. Higher scores indicate worsening status.[17] The reliability and validity of the Turkish version has been established by Süren *et al*.[18]

Short Form-36 (SF-36): The SF-36 Quality of Life Scale is the most widely used general quality of life scale in clinical research. The scale consists of 36 items covering 8 different subscales: physical function, social function, inhibition in roles due to physical problems, physical pain, mental health, inhibition in roles due to emotional problems, life energy, and general health perception. Subscales are scored between 0 and 100, and high scores indicate good health. The reliability and validity study of the Turkish version has been performed by Koçyiğit *et al*.[19]

Standard Mini Mental Test (SMMT): This is a screening test developed by Folstein et al. in 1975 for the evaluation of cognitive functions. It has been found to be reliable and valid for the Turkish population.[20,21] It is an easily applicable test in daily medical practice and is very suitable for cognitive status screening in the elderly. Cognitive status is evaluated under 5 main headings: orientation (10 points), attention and calculation (5 points), recording memory (3 points), language (9 points), and recall (3 points). The total score is 30. Respondents who score 24 and below should be evaluated for dementia.

Montreal Cognitive Assessment Scale (MoCA): This is a test used to evaluate cognitive features such as attention and concentration, executive functions, memory, language, visuospatial functions, abstract thinking, calculation, and orientation. It was developed by Nasreddine et al. and is recommended for use in mild stages of cognitive dysfunction.[22] The design of this test allows it to detect particularly mild cognitive dysfunction with higher sensitivity compared to the Standard Mini Mental Test (SMMT),. The Turkish MOCA version has been validated by Selekler *et al*.[23] The highest score that can be obtained from the test is 30, and a score of 26 and above is considered normal.[23]

Statistical analysis

SPSS 25.0 software (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY, USA: IBM Corp.) was employed for statistical analysis of the collected data. The Shapiro-Wilk test was used to determine normality of the data under consideration. Data with a normal distribution were described with mean ± standard deviation (± sd), and data that did not fit a normal distribution were described with median and range. When two groups were compared, the independent sample t-test was used for data with a normal distribution. The Mann-Whitney U test was used for non-normally distributed data and one-way ordinal data.

Correlation analysis was performed by Pearson correlation analysis for normally distributed data, and the Spearman correlation analysis using a Bonferroni correction was used for non-normally distributed data. A $p<0.05$ value was accepted for statistical significance.

RESULTS

A total of 60 people, 17 men and 43 women, with a mean age of 34.85±11.19, participated in the study. There were no significant differences between the groups in terms of age, gender, educational status and BMI ($p>0.05$) (Table 1).

With respect to cognitive performance, scores for all MoCA domains except orientation and SMMT scores were significantly lower in the group with cervicogenic headache ($p< 0.05$). 50% of the patients in the cervicogenic headache group and all members of the control group had a normal score (26 and above) on MoCA testing (Table 2).

The total score and scores for all sub-categories of PCS (Helplessness, Magnification, Rumination) were higher in patients with cervicogenic headache ($p< 0.05$).

The BDI score was significantly higher in patients with cervicogenic headache compared to the control group ($p=0.018$).

The SF-36 sub-parameter scores of patients with cervicogenic headache were significantly lower than in normal controls in terms of physical function, physical role difficulty, social functionality, pain and general health perception ($p< 0.05$) (Table 2).

No significant correlation was found between VAS pain scores and SMMT and MoCA scores in patients with cervicogenic headache, but a significant positive correlation was found between the MoCA and SMMT scores ($r=0.447$ $p=0.013$). Additionally, a negative correlation was detected between MoCA and BDI scores ($r=-0.418$ $p=0.022$) (Table 3). In the cervicogenic headache group, the MoCA scores were significantly lower in those with depressed mood ($p<0.001$) (Table 4).

DISCUSSION

Our study shows that patients with cervicogenic headache have worse cognitive performance during headache than normal individuals of similar age and educational level. In addition, they have a higher prevalence of depression, greater extent of pain catastrophizing and a lower quality of life.

Pain catastrophizing is defined as the tendency to magnify pain and feel helpless in the face

Table 1: Demographic characteristics of the participants

	Cervicogenic Headache (n=30)	Control (n=30)	t	p
Age (mean ± SD)	40.86±10.79	38.72±9.26	4.19	0.326
Education time (year±SD)	9.43±4.63	9.23±5.32	3.81	0.877
Gender (female %)	76.6	66.6	2.93	0.083

independent sample t-test

Table 2: Comparison between groups

	Cervicogenic headache (n=30)	Control (n=30)	t	p
VAS	6.0667±2.94	3.83±2.13	3.36	0.001 #
BDI	15±8.87	10.36±5.39	2.44	0.018#
PCS				
Helplessness	12.6±6.39	6.1±3.88	4.75	< 0.000#
Magnification	5.8±3.79	3.13±1.79	3.48	0.001#
Rumination	7.73±5.27	3.16±3.01	4.11	< 0.000#
Total	25.43±14.78	12.4±7.35	4.32	< 0.000#
SF-36				
Physical functioning	64.35±26.87	91.33±11.13	-5.08	< 0.000#
Role limitations physical	35±37.48	70±40.15	-3.49	0.001#
Role limitations emotional	44.44±42.28	57.77±37.07	-1.29	0.199#
Energy/Vitality	47.83±17.40	54.87±20.32	-1.43	0.157#
Mental health	55.46±18.3	63.06±17.56	-1.64	0.106#
Social functioning	57.08±19.05	70.41±20.09	-2.63	0.011#
Pain	42.83±19.56	72.83±1.29	-6.13	< 0.000#
General health perceptions	42.83±12.01	58.53±10.37	-5.41	< 0.000#
SMMT	26(19-30)	29(9-30)		< 0.000##
MoCA				
Visuospatial and executive functions	3.56±1.07	4.36±0.76	-3.32	0.002#
Naming	2.5±0.62	2.93±0.25	-3.49	0.001#
Attention	3.93±1.57	5.56±0.67	-5.21	< 0.000#
Language	1.53±1.22	2.2±0.92	-2.38	0.021#
Abstraction	0.9±0.88	1.4±0.72	-2.39	0.02#
Memory	1.66±1.47	3.4±1.37	-4.7	< 0.000#
Orientation	5.8±0.61	5.96±0.18	-1.43	0.161#
Total	19.9±4.25	25.83±2.49	-6.59	< 0.000#

Independent Sample T-Test, ## Mann-Whitney *U Test,* VAS: Visual Analogue Scale, BDI: Beck Depression Inventory, PCS: Pain Catastrophizing Scale, SF-36: Short Form-36, SMMT: Standard Mini Mental Test, MoCA: Montreal Cognitive Assessment Scale.

of pain due to the relative inability to inhibit thoughts about pain before, during, or after a painful situation. Catastrophizing one's pain contributes to more intense pain and increased emotional distress.24 Those who are prone to pain catastrophizing experience greater disability in painful situations.[25] It has been shown that the level of pain catastrophizing is higher in cluster and migraine headaches than in healthy controls and that it is related to prognosis.[26,27] In our study, it was found that the tendency to catastrophize pain was higher in patients with cervicogenic headache compared to healthy controls, which is consistent with the literature on other headache types. Pain catastrophizing should be considered in patients with cervicogenic headache.

The effect of headaches on the psychological state has long been the subject of research. Individuals with headache, especially migraine, show more depressive symptoms than their peers.[28-30] Nakamura *et al*. has found that individuals with cervicogenic headache have a higher incidence of various symptoms such as fatigue and irritability. Our study shows that BDI scores in the cervicogenic headache group were higher than in the control group. Depression may be a causative factor for headache rather than an associated condition observed with headache.[31] The prevalence of depression has been found to be as high as 36% in tension headache, 15.57% in migraine, and 34.6% in cluster headache.[32-34] Our study finds a comparable prevalence of depression (33.3%) in patients with cervicogenic headache. Cognitive function in primary and secondary headaches has also been the focus of research. The data related to cognitive dysfunction in the

Table 3 : Correlation of pain level, mood and cognitive test results

		VAS	MoCA	SMMT	BDI
			#	##	#
VAS	Rho		-0.079	-0.255	0.359
	p	1	0.677	0.173	0.052
	n	30	30	30	30
		#		##	#
MoCA	Rho	-0.079		0.447	-0.418
	p	0.677	1	0.013	0.022
	n	30	30	30	30
		##	##		##
SMMT	Rho	-0.255	0.447		-0.189
	p	0.173	0.013	1	0.318
	n	30	30	30	30
		#	#	##	
BDI	Rho	0.359	-0.418	-0.189	
	p	0.052	0.022	0.318	1
	n	30	30	30	30

#Pearson correlation. Correlation is significant at the 0.01 level, ##Spearman's correlation. Correlation is significant at the 0,05 level. VAS: Visual analogue scale, SMMT: Standard Mini Mental Test, MoCA: Montreal Cognitive Assessment scale, BDI: Beck depression inventory

interictal period in migraine are contradictory, but cognitive function is lower in the ictal period compared to healthy controls, and 44.7% of them have cognitive dysfunction.[35-38] In tension-type and cluster-type headaches there is also a decrease in ictal cognitive function.[9,39] Cognitive dysfunction has been shown in headaches attributed to genetic vasculopathies, arteritides, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstrictive syndrome, sickle cell disease, concussion, and idiopathic intracranial hypertension.[40-45] Cognitive dysfunction is observed during the attack in 28-77% of patients with secondary headaches.[14] In our study, cognitive dysfunction was detected during the attack in 50% of patients with cervicogenic headache, a fugure comparable to that found in literature for secondary hadaches. These data suggest that it may be benefical to evaluate cognitive dysfunction in the follow-up of patients with cervicogenic headache and to inform patients that their cognitive functions may decrease during pain attack.

Cognitive dysfunction is a part of the symptomatology of both clinical and subclinical depression.[46,47] Cognitive dysfunction may persist even when remission is obtained in depression.[48] Cognitive dysfunction associated with depression affects not only middle-aged and elderly people, but also young people aged 12 to 25 years.[49] Increasing age is associated with increased vulnerability to cognitive decline associated with depression.[50] Depression scores are higher in patients with migraine and those with cognitive dysfunction.[51] Treatment of migraine and concomitant depression improves cognitive function.[52] We could not find any studies examining the relationship between mood and cognitive functions in patients with cervicogenic headache in the literature. In our study, a negative correlation was found between MoCA score and BDI score in patients with cervicogenic headache,

Table 4: Comparison of pain and cognitive scores of patients with and without depression in cervicogenic headache

	Cervicogenic headache with depression (n=10)	Cervicogenic headache without depression (n=20)	t	p
MoCA	14.2±5.12	25±5.73	-2.69	<0.001
VAS	6.7±2.54	5.75±3.14	0.828	0.415

Independent sample t test. MoCA: Montreal Cognitive Assessment scale, VAS: Visual analogue scale

similar to that found in migraine patients. In addition, it was found that while the level of pain was similar to those without depression in individuals with cervicogenic headache with depression, cognitive performance was lower. Further studies examining the relationship between cognitive performance in this patient group are required.

Headaches cause significant personal distress, deterioration in the quality of life, and a significant economic burden on patients. Recurrent headache attacks and fear of the next attack can affect family, social and work life.[53] Studies investigating the quality of life in patients with cervicogenic headache are limited. In a study comparing the quality of life of individuals with cervicogenic headache, migraine, tension-type headache and healthy controls with SF-36 scores, all sub-SF-36 scores were found to be lower in patients with cervicogenic headache compared to healthy controls, and physical function score was lower than in patients with migraine and tension-type headache.[54] In our study, the scores of patients with cervicogenic headache were lower than the control group in terms of physical function, physical role difficulties, social functionality, pain and general health perception, in line with the literature.

The limitations of this study are first, the number of participants in this study is relatively small, from a single center. Second, computer-based measurement methods where there is no instruction bias or discrepancy due to human factors were not used in the evaluation of cognitive function. However, we think that this study is valuable because it is the first study to examine cognitive functions in patients with cervicogenic headache.

In conclusion, in this study, it was determined that the patients with cervicogenic headache exhibited worse cognitive performance, their depression rates and pain catastrophizing scores were higher, and thus, their quality of life was lower than people of similar age and education level. It is beneficial to evaluate patients with cervicogenic headache in this respect and to inform them about these issues.

DISCLOSURE

ClinicalTrials.gov ID: NCT05572489

Ethics: Ethics committee approval was obtained for the research (05.11.2021/ 33/28).

Financial support: None

Conflict of Interest: None

REFERENCES

1. Silverberg ND, Martin P, Panenka WJ. Headache trigger sensitivity and avoidance after mild traumatic brain injury. *J Neurotrauma* 2019;36(10):1544-50. DOI: 10.1089/neu.2018.6025
2. Al Khalili Y, Ly N, Murphy PB. Cervicogenic headache. Treasure Island (FL): StatPearls Publishing LLC. 2022.
3. Anthony M. Cervicogenic headache: prevalence and response to local steroid therapy. *Clin Exp Rheumatol* 2000;18(2 Suppl 19):S59-64.
4. Sjaastad O, Bakketeig LS. Prevalence of cervicogenic headache: Vågå study of headache epidemiology. *Acta Neurol Scand* 2008;117(3):173-80. DOI: 10.1111/j.1600-0404.2007.00962.x
5. Knackstedt H, Bansevicius D, Aaseth K, Grande RB, Lundqvist C, Russell MB. Cervicogenic headache in the general population: the Akershus study of chronic headache. *Cephalalgia* 2010;30(12):1468-76. DOI: 10.1177/0333102410368442
6. Moriarty O, McGuire BE, Finn DP. The effect of pain on cognitive function: A review of clinical and preclinical research. *Prog Neurobiol* 2011;93(3):385-404. DOI: 10.1016/j.pneurobio.2011.01.002
7. Povedano M, Gascón J, Gálvez R, Ruiz M, Rejas J. Cognitive function impairment in patients with neuropathic pain under standard conditions of care. *J Pain Symptom Manage* 2007;33(1):78-89. DOI: 10.1016/j.jpainsymman.2006.07.012
8. Berryman C, Stanton TR, Jane Bowering K, Tabor A, McFarlane A, Lorimer Moseley G. Evidence for working memory deficits in chronic pain: A systematic review and meta-analysis. *Pain* 2013;154(8):1181-96. DOI: 10.1016/j.pain.2013.03.002
9. Vuralli D, Ayata C, Bolay H. Cognitive dysfunction and migraine. *J Headache Pain* 2018;19(1):109. DOI: 10.1186/s10194-018-0933-4
10. Donaldson LB, Yan F, Liu YF, Nguyen SA, Rizk HG. Does cognitive dysfunction correlate with dizziness severity in patients with vestibular migraine? *Am J Otolaryngol* 2021;42(6):103124. DOI: 10.1016/j.amjoto.2021.103124
11. Qu P, Yu JX, Xia L, Chen GH. Cognitive performance and the alteration of neuroendocrine hormones in chronic tension-type headache. *Pain Practice* 2018;18(1):8-17. DOI: 10.1111/papr.12574
12. Chutko LS, Surushkina S, Rozhkova AV, Iakovenko EA, Bykova Iu L, Nikishina IS. Asthenic disorders and cognitive dysfunction in patients with tension headache. *Zhurnal Nevrologii i Psikhiatrii Imeni SS Korsakova* 2013;113(5):31-5.
13. Iskra DA, Lobzin VY, Kalygin SA. Primary headache - risk factor or comorbid pathology in cognitive impairment. *Zhurnal Nevrologii i Psikhiatrii Imeni SS Korsakova* 2017;117(12):141-6. DOI: 10.17116/jnevro2017117121141-146
14. Begasse de Dhaem O, Robbins MS. Cognitive impairment in primary and secondary headache disorders. *Curr Pain Headache Rep* 2022;26(5):391-404. DOI: 10.1007/s11916-022-01039-5
15. Headache Classification Committee of the International Headache Society (IHS)

The International classification of headache disorders, 3rd ed. *Cephalalgia* 2018;38(1):1-211. DOI: 10.1177/0333102417738202
16. Hisli N. Beck Depresyon envanterinin gecerliligi uzerine bit calisma (A study on the validity of Beck Depression Inventory) *Psikoloji Dergisi* 1988;6:118-22.
17. Sullivan MJ, Bishop SR, Pivik J. The pain catastrophizing scale: development and validation. *Psychological Assessment* 1995;7(4):524. https://doi.org/10.1037/1040-3590.7.4.524
18. Süren M, Okan I, Gökbakan AM, *et al*. Factors associated with the pain catastrophizing scale and validation in a sample of the Turkish population. *Turkish J Med Sci* 2014;44(1):104-8. DOI: 10.3906/sag-1206-67
19. Kocyigit H. Kisa Form-36 (KF-36)'nm Turkce versiyonunun guvenilirligi ve gecerliligi. *Ilac ve Tedavi Dergisi* 1999;12:102-6.
20. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. *J Psychiatric Res* 1975;12(3):189-98. DOI: 10.1016/0022-3956(75)90026-6
21. Güngen C, Ertan T, Eker E, Yaşar R, Engin F. Reliability and validity of the standardized Mini Mental State Examination in the diagnosis of mild dementia in Turkish population. *Turk Psikiyatri Dergisi* 2002;13(4):273-81.
22. Nasreddine ZS, Phillips NA, Bédirian V, *et al*. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *J Am Geriatr Soc* 2005;53(4):695-9. DOI: 10.1111/j.1532-5415.2005.53221.x
23. Selekler K, Cangoz B, Uluc S. Montreal bilişsel değerlendirme ölçeği (MOBİD)'nin hafif bilişsel bozukluk ve alzheımer hastalarını ayırt edebilme gücünün incelenmesi. *Türk Geriatri Dergisi* 2010;13(3):166-71.
24. Sullivan MJ, Thorn B, Haythornthwaite JA, *et al*. Theoretical perspectives on the relation between catastrophizing and pain. *Clin J Pain* 2001;17(1):52-64. DOI: 10.1097/00002508-200103000-00008
25. Sullivan MJ, Adams H, Rhodenizer T, Stanish WD. A psychosocial risk factor–targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Phys Ther* 2006;86(1):8-18. DOI: 10.1093/ptj/86.1.8
26. Gil-Martínez A, Navarro-Fernández G, Mangas-Guijarro MÁ, *et al*. Comparison between chronic migraine and temporomandibular disorders in pain-related disability and fear-avoidance behaviors. *Pain Med* 2017;18(11):2214-23. DOI: 10.1093/pm/pnx127
27. Díaz-de-Terán J, Sastre-Real M, Lobato-Pérez L, Navarro-Fernández G, Elizagaray-García I, Gil-Martínez A. Cluster headache, beyond the pain: a comparative cross-sectional study. *Neurol Sci* 2021;42(9):3673-80. DOI: 10.1007/s10072-020-04996-0
28. Arena JG, Blanchard EB, Andrasik F. The role of affect in the etiology of chronic headache. *J Psychosom Res* 1984;28(1):79-86. DOI: 10.1016/0022-3999(84)90043-6
29. Bertolotti G, Vidotto G, Sanavio E, Frediani F. Psychological and emotional aspects and pain. *Neurol Sci* 2003;24 (Suppl 2):S71-5. DOI: 10.1007/s100720300046
30. Just U, Oelkers R, Bender S, *et al*. Emotional and behavioural problems in children and adolescents with primary headache. *Cephalalgia* 2003;23(3):206-13. DOI: 10.1046/j.1468-2982.2003.00486.x
31. Scharff L, Turk DC, Marcus DA. Psychosocial and behavioral characteristics in chronic headache patients: support for a continuum and dual-diagnostic approach. *Cephalalgia* 1995;15(3):216-23. DOI: 10.1046/j.1468-2982.1995.015003216.x
32. Chen YC, Tang CH, Ng K, Wang SJ. Comorbidity profiles of chronic migraine sufferers in a national database in Taiwan. *J Headache Pain* 2012;13(4):311-9. DOI: 10.1007/s10194-012-0447-4
33. Kim BS, Chung PW, Kim BK, *et al*. The impact of remission and coexisting migraine on anxiety and depression in cluster headache. *J Headache Pain* 2020;21(1):58. DOI: 10.1186/s10194-020-01120-7
34. Yücel B, Kora K, Ozyalçín S, Alçalar N, Ozdemir O, Yücel A. Depression, automatic thoughts, alexithymia, and assertiveness in patients with tension-type headache. *Headache* 2002;42(3):194-9. DOI: 10.1046/j.1526-4610.2002.02051.x
35. Gil-Gouveia R, Martins IP. Cognition and cognitive impairment in migraine. *Curr Pain Headache Rep* 2019;23(11):84. DOI: 10.1007/s11916-019-0824-7
36. Qin T, Chen C. Cognitive dysfunction in migraineurs. *Medicina (Kaunas)* 2022;58(7). DOI: 10.3390/medicina58070870
37. Gu L, Wang Y, Shu H. Association between migraine and cognitive impairment. *J Headache Pain* 2022;23(1):88. DOI: 10.1186/s10194-022-01462-4
38. Lee SH, Kang Y, Cho SJ. Subjective cognitive decline in patients with migraine and its relationship with depression, anxiety, and sleep quality. *J Headache Pain* 2017;18(1):77. DOI: 10.1186/s10194-017-0779-1
39. Torkamani M, Ernst L, Cheung LS, Lambru G, Matharu M, Jahanshahi M. The neuropsychology of cluster headache: cognition, mood, disability, and quality of life of patients with chronic and episodic cluster headache. *Headache* 2015;55(2):287-300. DOI: 10.1111/head.12486
40. Wang MM. CADASIL. *Handb Clin Neurol* 2018;148:733-43. DOI: 10.1016/B978-0-444-64076-5.00047-8
41. Lahaye C, Sanchez M, Rouet A, Gross A, *et al*. A curable pseudo-dementia related to an atypical presentation of giant cell arteritis. *Age Ageing* 2020;49(3):487-9. DOI: 10.1093/ageing/afaa010
42. Adank MC, Hussainali RF, Oosterveer LC, *et al*. Hypertensive disorders of pregnancy and cognitive impairment: A Prospective cohort study. *Neurology* 2021;96(5):e709-e18. DOI: 10.1212/WNL.0000000000011363
43. Longoria JN, Pugh NL, Gordeuk V, *et al*. Patient-reported neurocognitive symptoms influence instrumental activities of daily living in sickle cell disease. *Am J Hematol* 2021;96(11):1396-406. DOI: 10.1002/ajh.26315
44. Ashina H, Al-Khazali HM, Iljazi A, *et al*. Psychiatric

and cognitive comorbidities of persistent post-traumatic headache attributed to mild traumatic brain injury. *J Headache Pain* 2021;22(1):83. DOI: 10.1186/s10194-021-01287-7
45. Grech O, Clouter A, Mitchell JL, *et al*. Cognitive performance in idiopathic intracranial hypertension and relevance of intracranial pressure. *Brain Commun* 2021;3(3):fcab202. DOI: 10.1093/braincomms/fcab202
46. Dehn LB, Beblo T. Depressed, biased, forgetful: The interaction of emotional and cognitive dysfunctions in depression. *Neuropsychiatr* 2019;33(3):123-30. DOI: 10.1007/s40211-019-0307-4
47. Wagner S, Doering B, Helmreich I, Lieb K, Tadić A. A meta-analysis of executive dysfunctions in unipolar major depressive disorder without psychotic symptoms and their changes during antidepressant treatment. *Acta Psychiatr Scand* 2012;125(4):281-92. DOI: 10.1111/j.1600-0447.2011.01762.x
48. Czerwińska A, Pawłowski T. Cognitive dysfunctions in depression - significance, description and treatment prospects. *Psychiatr Pol* 2020;54(3):453-66. DOI: 10.12740/PP/OnlineFirst/105415
49. Goodall J, Fisher C, Hetrick S, Phillips L, Parrish EM, Allott K. Neurocognitive functioning in depressed young people: A Systematic review and meta-analysis. *Neuropsychol Rev* 2018;28(2):216-31. DOI: 10.1007/s11065-018-9373-9
50. Dotson VM, Zonderman AB, Davatzikos C, Kraut MA, Resnick SM. Frontal atrophy and attention deficits in older adults with a history of elevated depressive symptoms. *Brain Imaging Behav* 2009;3(4):358. DOI: 10.1007/s11682-009-9078-z
51. Bao J, Ma M, Dong S, *et al*. Early age of migraine onset is independently related to cognitive decline and symptoms of depression affect quality of life. *Curr Neurovasc Res* 2020;17(2):177-87. DOI: 10.2174/1567202617666200207130659
52. Pozhidaev KA, Parfenov VA. Cognitive and emotional disorders in patients with migraine and signs of leukoencephalopathy. *Zhurnal nevrologii i psikhiatrii imeni SS Korsakova* 2021;121(3):7-12. DOI: 10.17116/jnevro20211210317
53. Arslantas D, Tozun M, Unsal A, Ozbek Z. Headache and its effects on health-related quality of life among adults. Turk Neurosurg 2013;23(4):498-504. DOI: 10.5137/1019-5149.JTN.7304-12.0
54. van Suijlekom HA, Lamé I, Stomp-van den Berg SG, Kessels AG, Weber WE. Quality of life of patients with cervicogenic headache: a comparison with control subjects and patients with migraine or tension-type headache. *Headache* 2003;43(10):1034-41. DOI: 10.1046/j.1526-4610.2003.03204.x