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Therapeutic strategy with indirect spinal manipulations in C2-C3 segments for long-term treatment of cervicogenic headache

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Abstract

Cervicogenic Headache (CGH) is a secondary type headache, associated with dysfunction in upper cervical segments (C1-C2-C3) and manifested by specific clinical features. It is unilateral, starting from one side of the posterior head and neck, migrating to the front, sometimes associated with ipsilateral arm discomfort and, in addition, neuralgia with ipsilateral conjunctival inection. Our research is based on 29 patients (with average age of 49.78 years ± 10.41 (34-73)) with headache: 19 females and 10 males. We examined the active range of motion (AROM). We used palpatory maneuvers, finding local symptoms (facet joints' tenderness) and symptoms in the segmental territory – cellulalgia in the supraorbital and submandibular region (found by the pinch-roll test – PR-SO and PR-SM). The clinical examination before and immediately after the indirect high velocity low amplitude (HVLA) manipulations, as well as at the end of treatment, showed a significant reduction in the Visual Analogue Scale (VAS) score, in local symptoms and especially in measurable indicators – skin fold (PR-SO and PR-SM) in mm. ($p<0.01$ and $p<0.001$). Our study shows that indirect HVLA-spine manipulations have an invariable place in the treatment of cervicogenic headache.

Key Words: cephalgia, cervicogenic headache, HVLA manipulations, manual medicine.

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Headache or cephalgia is the most widespread pain disorder, and approximately half of the population suffers from a headache.[1] Pathogenetically, headache is caused by disturbance of the pain-sensitive structures around the brain - the periosteum of the skull, muscles, nerves, arteries and veins, subcutaneous tissues, eyes, ears, sinuses and mucous membranes.[2] The term "cervicogenic headache" was introduced into clinical terminology by Dr. Ottar Sjaastad in 1983 by recognizing a subgroup of headache patients with concomitant head and neck pain.[3] This type of headache was difficult to diagnose and treat because its etiology and pathophysiology were not well understood.[4]

Cervicogenic Headaches (CGH) are unilateral, starting from one side of the posterior head and neck, migrating to the front, and are sometimes associated with ipsilateral arm discomfort.[5] Clinical symptoms are complemented by neuralgia – transient, tearing pain in the occipital region (in 75% unilateral, it can also be located in the temporal, frontal or orbital region) with ipsilateral conjunctival inection. The frequency of clinical episodes is 4-5 times daily to 2-7 times weekly.[6] The diagnostic criteria include persistent or tearing pain in the territory of C2 or C3, symptoms indicating compression, tension or irritation of the nerve and in addition – the disappearance of the headache following a diagnostic blockade.[6]

The biomechanics and functionality of the craniofacial and cervical regions play a significant role in the pathogenesis and persistence of CGH. Messina G. explored these interrelations in his studies on the styloid apophysis of the temporal bone, highlighting its role in the biomechanics of the tongue, mandible, and hyoid system.[7] Dysfunction or tension in this area can potentially influence neuromuscular dynamics, contributing to CGH symptoms. Similarly, in his work on orofacial muscle activity in children with swallowing dysfunction, Messina

et al.[8] demonstrated how impaired muscle coordination can have far-reaching implications on postural stability and cranio-cervical mechanics, which may further exacerbate CGH-like pain symptoms. These findings underline the importance of assessing craniofacial mechanics when diagnosing and treating CGH.

In rehabilitation contexts, innovative interventions are being explored. Alashram et al. conducted a systematic review on transcranial Direct Current Stimulation (tDCS) for upper extremity spasticity rehabilitation in stroke survivors. Their findings highlight the neuromodulation potential of tDCS, which may also have implications for chronic pain conditions like CGH, considering its impact on cortical excitability.[9]

Similarly, immersive technologies such as Virtual Reality (VR) have emerged as promising rehabilitation tools. Alashram, Padua, and Annino reviewed the use of VR for balance and mobility rehabilitation in patients with Traumatic Brain Injuries (TBI). This approach emphasizes the importance of neuroplasticity-driven therapies and offers potential for applications in CGH, particularly in targeting neck movements and proprioception deficits.[10]

The fundamental method of establishing neck and head pain is the Active Range Of Motion (AROM), as many studies have shown significant decreases in AROM in patients with CGH.[11]

The diagnostic criteria according to the International Headache Society (IHS) are the presence of temporal relation to the onset of the cervical disorder and a significant improvement or resolution in parallel with the improvement or resolution of the cervical disorder or lesion.[12] According to the Vaga study, the pain should start posteriorly and end anteriorly and additionally suggests the following: i) unilateral head pain without side shift; ii) provocation via unphysiological neck positions; iii) external provocation in the neck/occipital area; iv) diffuse shoulder and arm pain.[13]

Maigne[14] described segmental symptoms of CGH that could be found by palpation over the facet joints (local segmental symptoms) and symptoms in the segmental territory. In the posterior primary rami of C2 and C3 the author described the "friction sign of the scalp" – pain and sensitivity on friction in the occipital region. In patients with CGH originating from C2 and C3, in the territory of the anterior primary rami, he found "cellulalgia" – a painful, thickened skinfold, formed by pinching and rolling (like a cigarette) in the supraorbital region ("eyebrow sign") and in the submandibular region, at the angle of the jaw ("the angle of the jaw sign").[14,15]

More recently, Antonaci and Inan proposed new diagnostic criteria for CGH.[16] They introduced evidence of causation: the CGH should be improved by at least 50% or resolved in parallel with improvement in or resolution of the cervical disorder or lesion. In the absence of a disorder or lesion, the same decrease of symptoms should be observed with specific treatment for cervicogenic headache and the headache should not respond to specific treatment for migraine (like triptans); reduced range of motions of cervical rotation of ≥ 10% should be observed on the symptomatic side. Headache is made significantly worse by provocative maneuvers or pressing (3–4 kg) with the finger against the upper trapezius and splenius area and against facet joints. The provoked headache should start posteriorly and spread to the anterior.

Material and Methods

Our study is based on 29 patients aged 34-73 (average 49.8 ± 10.4 years) with segmental symptoms of CGH: 19 females of 21 to 73 (65,5%, with average age of 51,1 years) and 10 males (34,48%, with average age of 47.4 years – 33 to 66). The headache was bilateral in 22 patients (75,9%), left-sided in 4 patients (13,8%) and right-sided in 3 patients (10,3%). Fourteen patients (48,28%) had, as an accompanying complaint, neck pain and 2 patients (6,90%) had contralateral migraine pain. Based on the complaints, all patients with bilateral headache (22 patients - 75.86%) could be diagnosed with "Tension-type headache" (TTH). Six of them (20.69%) fulfilled criteria for "Frequent episodic TTH" and the other 16 patients (55.17%) fulfilled the criteria for "Chronic TTH". Three of the patients (10.35%) with chronic TTH developed medication (non-steroidal anti-inflammatory drugs - NSAID) overuse headache. There were no patients with concomitant use of prophylactic medications. The previous treatment was with NSAID. There were no patients with a history of head/neck trauma.

Patients reported the duration of their headache lasting from 7 to 3650 days (average 1019 days). Three patients (10,3%) had acute headache with an onset within a week, 2 patients (6,90%) within 30 days, one (3.45%) within 60 days, one within 4 months and one within 6 months. The others (21 patients – 72.4%) reported a duration of more than one year, 7 of them (20.7%) of more than 4 years.

Clinical examination included neurological examination, evaluation of AROM, finding local symptoms of the spine, cellulalgia and "friction sign of the scalp". The procedures were conducted in accordance with the ethical principles set out in the Declaration of Helsinki and were performed with the ethical approval of the university. The study design was approved by the Department of Neurology at the Medical Faculty, Medical University - Sofia (approval date: 17.05.2022).

For the purpose of the study, we chose to use cellulalgia, as the skinfold could be measured before and after treatment. To find cellulalgia, the eyebrow (pinch-roll in the supraorbital region, PR-SO) was pinched between the thumb and index fingers, kneaded and rolled like a cigarette ("eyebrow sign").[14] It was investigated from one end to the other, going over the skin of the forehead (Figure 1a). When the sign was positive, the fold was painful and often thickened throughout all or part of the length of the brow. The same maneuver was performed for "the angle of the jaw sign" - pinch-roll test in the submandibular region, PR-SM) (Figure 1b). Friction applied to the scalp replaced the pinch-roll test for occipital region. It consisted of pressing firmly with the pad of the fingers against the scalp and mobilizing it with small to-and-fro

Figure 1. *Examination of cellulalgia a, in the supraorbital region with the "Pinch-Roll" method in the left supraorbital fold; b, in the submandibular region, in the left mandible angle.*

motion.[14,15] These maneuvers were controlled and compared with the opposite side.

The therapy of CGH consisted of indirect high velocity low amplitude spinal manipulations (HVLA-SM) in C2-C3 (average 2.44 HVLA-SM per patient). The patient was in supine position, the manipulation initiated with a slight flection 12-15°, additional slight rotation (8-10°) to the opposite of manipulated facet side and 8-10° ipsilateral side bending to relax the vertebral artery. The thrust was minimal, performed by the ipsilateral doctor`s hand (thumb) from the maxilla towards contralateral frontal tuber (eminence). (Figure 2). Patients did not receive medications, except for three of them (10.3 %) who took 500 mg Paracetamol, because of a reaction to the manipulation (transient ligament laxity) between 12 and 24 hours after the SM.

To evaluate the effect of HVLA-SM and for the purpose of the study, we additionally measured the PR-SO and PR-SM skin fold in mm with a caliper before and after each HVLA-SM.

Patients also assessed the pain symptoms before and after the manipulations, after 6-9 hours, 24 hours, 48 hours and on every next 24 hours up to the second visit using the Visual Analogue Scale (VAS). The VAS was also assessed before and after each HVLA-SM and at the end.

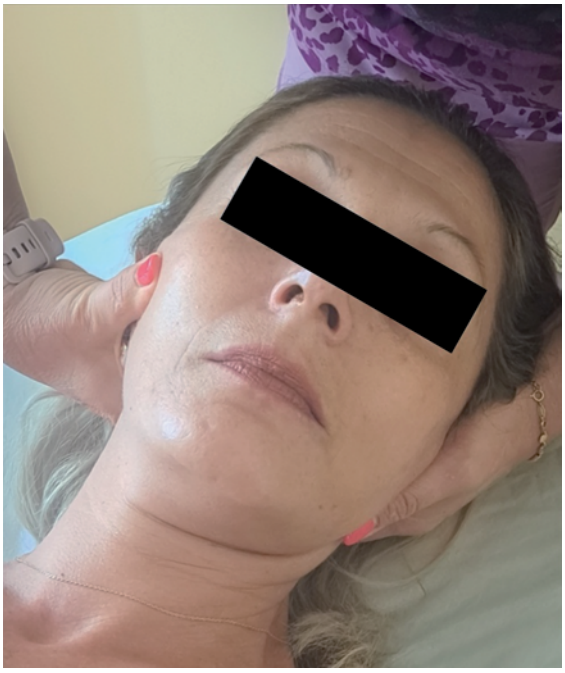

Figure 2. *Indirect HVLA-SM in C2-C3 segment.*

Results

Clinical examination established local segmental symptoms in the C2-C3 joint in all patients, in 26 of them bilaterally (89.66%), in two only on the left side (6.90%) and in one only on the right side (3.45%). In 5 of the patients, we observed "friction sign" bilaterally (17.24%), in 7 only on the left side (24.14%) and in three of them only on the right side (10.34%). During the clinical examination, the AROM was restricted in 24 of our patients (82.76%) (Table 1).

The clinical examination before and immediately after the HVLA manipulations showed a significant reduction in local symptoms (Figure 3) and especially in the measurable indicators PR-SO and PR-SM in the territory of the examination. The measurement by a caliper of PR-SO (Figure 4a/b) and PR-SM (Figure 5a/b) showed statistically significant reduction of the skin fold in mm after the first indirect HVLA-manipulation and at the end of the therapeutic course ($p<0.01$ and $p<0.001$) (Figure 6). The patients did not report any serious or non-serious adverse events after HVLA-SM during the whole period of treatment, except three of them, who complained about transient stiffness and neck pain between 12 and 24 hours only after the first manipulation.

Measurement of pain by VAS showed a significant reduction from mean 42.5 mm (SD ± 36.28) before manipulations to mean 16.38 mm (SD ±11.31) after the first manipulation. Measured mean VAS at the end of treatment (after the second or third manipulation) was 1.50 mm (SD ± 3.51) (Figure 7).

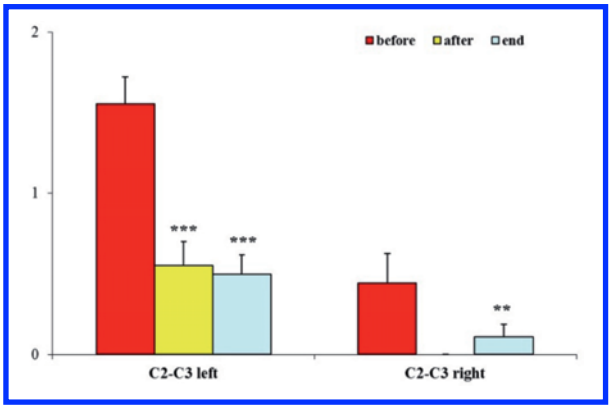

Figure 3. *Statistical significance (**p <0.01 and ***p <0.001) after the first indirect HVLA-manipulation and in the end of the therapeutic course, clinically established by the change of local segmental symptoms in the territory of C2-C3 during the clinical examinations.*

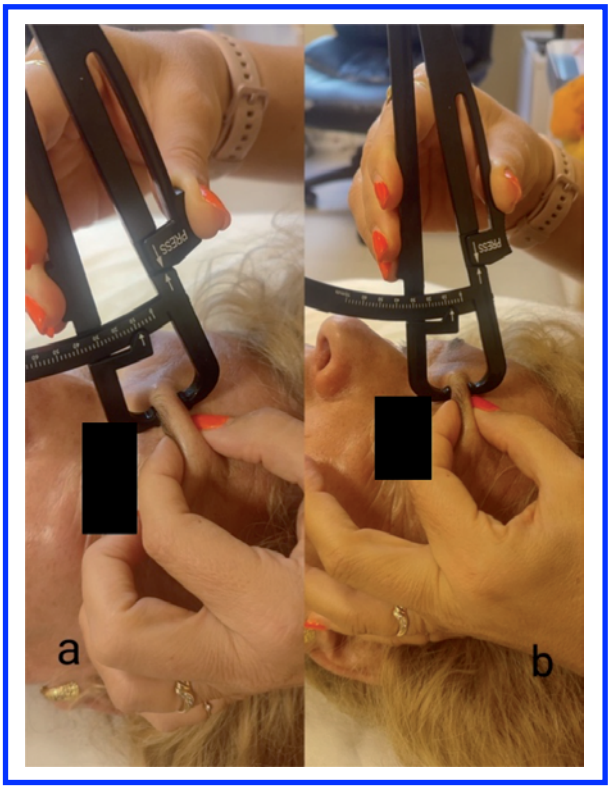

Figure 4. *Examination of cellulalgia in the supraorbital region with a caliper in the left supraorbital fold a, before the HVLA manipulation – 10 mm; b, after the HVLA manipulation – 8 mm.*

Table 1. *Number of patients with established segmental symptoms during clinical examination before the HVLA-manipulations.*

Segmental symptoms	Bilaterally	Left side	Right side
C2-C3 joint	26	2	1
PR supraorbital	11	10	8
PR submandibular	6	9	7
"Friction sign"	5	7	3

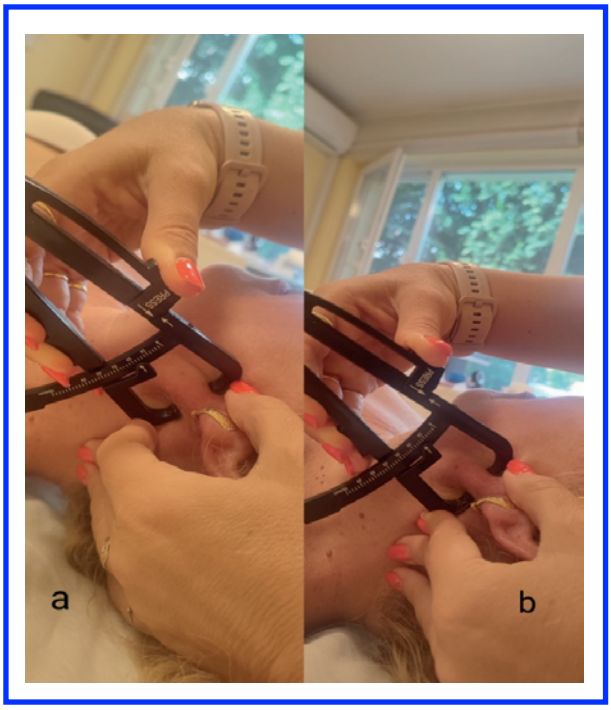

Figure 5. *Examination of cellulalgia in the submandibular region with a caliper in the left submandibular fold a, before the HVLA manipulation – 17 mm; b, after the HVLA manipulation – 14 mm.*

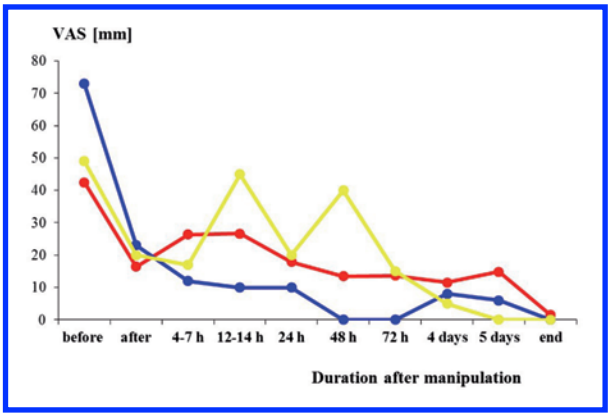

Figure 7. *Significant reduction of subjective pain intensity of 3 patients after the first manipulation, reported before the first manipulation, after the first manipulation, after 4-7 hours, 12-14 hours, 72 hours, 4 and 5 days after, after the second manipulation and at the end of treatment (after the last manipulation), measured by VAS. The blue line represents the changes of VAS of DM, 40-year-old female, with 7-day duration of headache. The yellow one represents HE, 34-year-old female, with combination of CGH and migraine, duration of 180 days, and red line – MS, 53-year-old female, with headache duration of 5 months.*

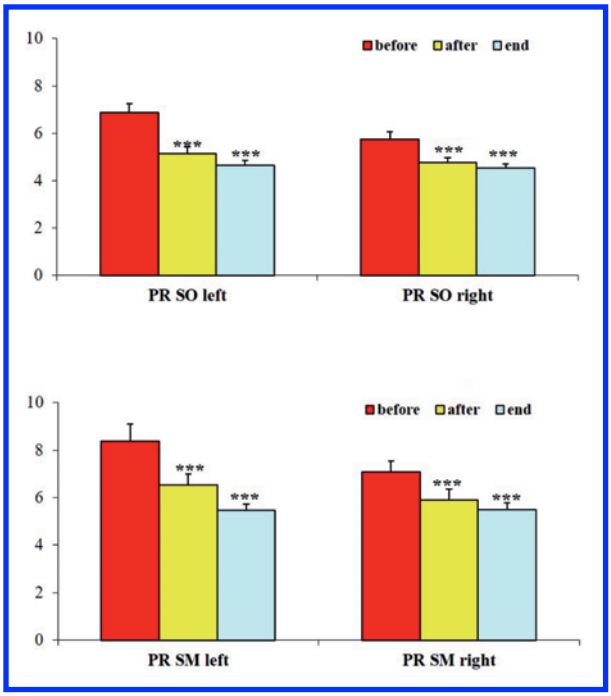

Figure 6. *Statistical significance (***p < 0.001) after the first indirect HVLA-manipulation and in the end of the therapeutic course, clinically established by the change of cellulalgia – PR SO (up) and PR – SM (down), measured with a caliper in mm.*

Discussion

CGH is described as a headache caused by a disorder of the cervical spine and its bony, discal and/or soft tissue elements, usually but not invariably accompanied by neck pain.[17] It begins mainly with musculoskeletal dysfunction in the upper three cervical segments. Entrapment of C2 or C3 branches plays a key role in its pathogenesis – C3 branch entrapment in foramen intervertebrale, due to disc prolapse, spondylosis or arthrosis; and C2 branch (passing posteriorly to the C1-C2 joint) entrapment as a result of fibrosis, meningioma, neuroma or AVM. This entrapment over time leads to the development and progression of cervicogenic headache, pathogenetically explainable with the convergence of sensory input at the trigeminocervical (spinal trigeminal) nucleus.[18,19]

The nociceptive input originating from the neck is directed to the trigeminocervical nucleus, which descends in the spinal cord down to the level of C3/4.[20] This gray nucleus is anatomically and functionally related to the perception of pain sensations in the head area, which explains the headache resulting from musculoskeletal dysfunction in the upper three cervical segments, a concept known as convergence. The trigeminal pathway theory, including central sensitization of pain, likely from the spinal trigeminal nucleus, is indicated as a major factor in the pathophysiology of CGH.[20]

This theory has been confirmed clinically by the injection of corticosteroids in the greater and lesser occipital nerves and the subsequent reduction in headache explained by

the blockade of the trigeminal nociceptive input relay.[21] The study of Chua *et al.* has shown impairment in sensory testing of the head in CGH patients compared to patients with neck dysfunction without headache.[22] The immediate reduction of the size of the skin fold in mm in our patients, measured by a caliper in the territory of the trigeminal nerve (in the supraorbital region) after spinal manipulation in C2-C3, is another proof of the connection between the upper cervical segments and the sensory area of the trigeminal nerve. This diagnostic procedure, described by R. Maigne in 1968, is simple and easy to test for a specialist trained in segmental examination and is an objective sign measurable with a caliper. The reduction in PR-SO and PR-SM after HVLA-SM indicates that the origin of this headache is at least partly located in the upper cervical segments and is reversible by removing this pathogenic reaction. According to the most recent criteria, the effect we had after HVLA-SM proves that it really is a CGH.[16]

CGH should be distinguished differentially from the two most common types of headaches in clinical practice – migraine and tension-type headache.[23] Of all cases of headache, tension-type headache accounts for almost 40%, while migraine accounts for about 10-15% according to epidemiological studies.[1] The diagnosis in these two categories of headache is more or less based mainly on the patient`s history. In contrast, through objective segmental examination by palpation, CGH could be found and quantitatively measured as shown in our patient collective. Moreover, 22 patients (75.86%) in our group fulfilled the criteria for TTH. Some authors state that the clinical signs in favor of CGH are the side-locked type of pain, provocation of typical headache by digital pressure on neck muscles and by head movement, and posterior-to-anterior radiation of pain.[23] This is similar to our palpation technique over facet joints demonstrating the local segmental symptoms by segmental examination. Examining the local segmental symptoms, we also found that pressure maintained on the facet joint point reproduced the usual referral pattern – another proof of the cervicogenic origin of headache in our patients. An additional sign of no less importance is the aggravation of pain by sustained neck positions.[24] Migraine or tension-type headaches can also present with neck pain, which makes it sometimes difficult to distinguish in new patients without long-term follow-up.[25] In patients with migraine or fibromyalgia an increase in basal blood lactate has been demonstrated as a recognized migraine trigger. Lactate is involved in peripheral nociception, and it appears (together with adenosine triphosphate - ATP) to facilitate the response of ASIC (acid-sensing ion channels - neuronal voltage-insensitive sodium channels activated by extracellular protons permeable to Na^+) following exposure to a low pH. A greater variance in lactate levels has been demonstrated in fibromyalgic subjects, as well as higher lactate levels at rest compared with subjects with migraine and non-pathological subjects. Thus, lactate levels could be a marker for the distinguishment between CGH and migraine.[26] Bruxism is another cause for headache with a similar pattern and should be taken into consideration in the differential diagnosis of cervical headache. Ultrasound and thermographic assessment can help us to differentiate this cause of headache.[27-29]

Manual therapists have for some time treated the cervical spine in efforts to relieve headache, but only recent discoveries about the convergence of pain have made it possible to scientifically explain its therapeutic effect through manipulations in the cervical region.[30] Jull et al., in a randomized controlled trial of high methodological quality, showed that manual therapy was an effective form of management for CGH.[31] Bronfort et al. did not support the use of manual therapy for the longterm management of other headache forms, especially migraine or tension-type headache.[32] It was proposed that, due to the various etiologies, manual therapy approaches should be effective only in certain cases.[33]

Using self-myofascial release in patients with migraine a recent study also showed an improvement in stabilometry with the decrease of Ellipse surface OE and CE ($p<0.05$) and the length of the sway path (CE) ($p<0.05$).[34] These results are promising, because manual therapy, due to the release of the neck muscles, can contribute in maintaining balance. Also, osteopathic manipulative treatment showed to improve postural control with open eyes and improved dysfunctional patterns of the lumbosacral and upper cervical spine in young professional football players.[35]

Our results show, that both subjective (VAS) and objective (segmental symptoms) changed after the manual treatment. This confirms the cervicogenic origin of the headache and points out the indirect spinal manipulations as effective treatment of CGH. The following of VAS in some patients is presented on Figure 7. In the case of the patient with 7-day duration of headache the reduction of VAS decreases gradually day after day up until the end of treatment (blue line, Figure 7). The patient with a longer duration experienced slight increasing of pain on VAS (as a reaction to the manipulation) to the second visit, but declined at the end (red line, Figure 7). More relapses of pain were experienced by the patient (with a combination of CGH and migraine headache). That might be because of the persisting central sensitization. More patients followed for a longer period are required in order to discover true interdependencies.

Limitations of our study are the relatively small sample size, limited follow-up (several weeks to the alleviation of pain) and a lack of control group. For future studies we plan to compare and follow for 12 months two bigger groups – with HVLA-SM and a control group. It is necessary every patient to keep a diary of headaches and more questionnaires

Conclusions

Our study shows that the presence of CGH can be proven by a palpatory method of segmental examination and that the indirect HVLA-SM have an invariable place in its treatment. This approach can avoid an unnecessary long-term drug treatment. The data provided will serve to enrich the scientific literature and expand the list of therapeutic options for medical professionals.

List of abbreviations

CGH, Cervicogenic Headache
HVLA-SM, High Velocity Low Amplitude Spinal Manipulation
tDCS, Transcranial Direct Current Stimulation
VR, Virtual Reality
TBI, Traumatic Brain Injuries
AROM, active range of motion
PR-SO, Pinch-Roll Supraorbital
PR-SM, Pinch-Roll Submandibular
VAS, Visual Analogue Scale
TTH, Tension-Type Headache
NSAID, non-steroidal anti-inflammatory drugs
ATP, Adenosine Triphosphate
ASIC, Acid-Sensing Ion Channels

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Contributions

All authors contributed equally.

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Nothing to disclose.

Conflict of interest

The authors declare that they have no competing interests.

Ethics approval

The study design was approved by the Department of Neurology at the Medical Faculty, Medical University - Sofia (approval date: 17.05.2022).

Data availability statement

The data presented in this study is available on request from the corresponding author.

Informed consent statement

Informed consent was obtained from all subjects involved in the study, or responsible caregivers, whichever is appropriate.

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