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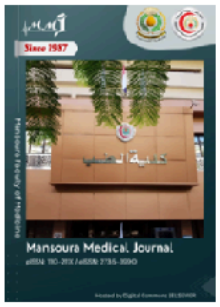

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Ultrasound Guided Nerve Block in Cervicogenic Headache

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ORIGINAL STUDY

Ultrasound-guided Nerve Block in Cervicogenic Headaches

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Abstract

Background: **Cervicogenic headache (CGH) is considered a common type of headache all over the world. There are a lot of therapeutic modalities for CGHs either noninvasive or invasive strategies. Based on the original cause of pain, there are several approaches to local or regional anesthesia injections comprising nerve root injections, trigger point blocks, peripheral nerve blocks, or facet blocks.**

Aim: **To assess the efficacy of the ultrasound (US)-guided greater occipital nerve block in CGH.**

Patients and methods: **The study was designed as a prospective interventional study that included 50 patients with CGHs after fulfillment of criteria according to ICHD-III classification and treated by US-guided nerve block. The anesthetic used consisted of 1 ml of 2 % lidocaine, 2.5 ml of 0.25 % marcaine, and 1 ml of betamethasone.**

Results: **There was a statistically significant decrease in the numeric pain scale from 6.64 pretreatment to 1.7 by 74.4 % after 30 min, and then a slight increase to 2.62 (60.5 %) after 2 weeks and a slight increase to 3.32 (60.5 %) after 4 weeks and then an increase to four (39.8 %) after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value regarding physical functioning, emotional functioning score, emotional well-being, pain domain, general health domain, and health change domain of quality of life.**

Conclusion: **US-guided greater occipital nerve block is demonstrated to be a novel, safe, and effective treatment method, which was associated with pain reduction (as revealed by the numeric pain scale) and better quality of life (as revealed by Rand-36).**

Keywords: Cervicogenic headache, Greater occipital nerve block, ICHD-III classification

1. Introduction

A cervicogenic headache (CGH) presents as unilateral pain, which starts in the neck. It is a frequent chronic and recurrent headache, which often starts after neck movement. It is often accompanied by a reduction in the range of motion of the neck. In addition, it may be confused with a migraine, tension headache, or other primary headache syndromes (Diaz *et al.*, 2019). Diagnosis of CGH depends on the fulfillment of criteria according to ICHD-III classification (Headache Society, 2018).

A CGH is believed to be referred pain emerging from irritation induced by cervical structures innervated by spinal nerves C1, C2 mainly, and to some extent C3; as a result, any structure innervated by the C1−C3 spinal nerves may be the source for a CGH (Al Khalili *et al.*, 2018). This might comprise the joints, disk, ligaments, and musculature. The lower cervical spine might have an indirect function with regard to pain production if dysfunctional; however there is no obvious proof of a direct referral pattern (Becker, 2010).

Through controlled nerve blocking of different structures in the cervical spine, it seems that the zygapophyseal joints, in particular those of C2/C3, are the commonest sources of CGH pain. Such an outcome is very common among cases with a previous history of whiplash (Maureen, 2017). MRI of the cervical spine may be useful in the identification of the cause of the CGH. Possible causes include

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cervical spine tumors in the bones or soft tissues, herniated or bulging disks, aneurysms, bone or joint abnormalities, birth defects, trauma, scoliosis, and infections (Maureen, 2017).

Medical treatment of CGH includes NSAIDs, muscle relaxants, and other pain relievers. Nonmedical treatment includes physical therapy, spinal manipulation, and surgery (Lance Whorton and Kegerreis, 2000). Although no optimum therapy is available so far, nerve block represents an emerging treatment option of CGH, and may temporarily relieve pain. Peripheral nerve blocks aim to suppress impulse transmission distally in a nerve terminal, as a result ending the pain signal perceived by the cerebral cortex. Ipsilateral greater occipital nerve (GON) significantly reduces the pain for 7 days following the blockade. Of note, the degree of improvement is relatively mild throughout the initial 48 h 'tilde pattern.' GON block could decrease the exaggeration in sensory input and antagonize a putative wind-up-like action, which could clarify the degree of improvement (Yaksh, 1993).

A nerve block is promising and effective, and it is used mainly by an anathesiologist; what is novel about our study is that it is used by neurologists as a bedside diagnostic and therapeutic method for headaches.

2. Aim

The current study aimed to assess the efficacy of the US-guided GON block in CGH.

3. Patients and methods

The study was designed as a prospective interventional study that included 50 patients with CGH due to cervical disc degeneration fulfilling inclusion and exclusion criteria treated by ultrasound (US)-guided nerve block at the Neurology Outpatient Clinic at Mansoura University from 2022 to 2023 after approval of the Institutional Review Board (IRB). We included patients aged 18 years or older diagnosed with CGH according to the following diagnostic criteria (according to ICHD-III classification (Arnold, 2018)), which included unilateral or bilateral pain starting in the neck and radiating to the frontotemporal area, pain worsened by neck motion, MRI cervical spine showing degenerative disc disease, restricted cervical range of motion, and headache frequency of at least one weekly within more than 3 months. But in the current study, we excluded patients with a history of cervical spine surgery, direct or indirect trauma, or surgical approach comprising the head or neck during the last year; patients with evidence of other neurological, dermatological, or surgical disc prolapse, patients with history of bleeding diathesis, coagulopathy, or current use of anticoagulant drugs with a history of complications or allergy to local anesthetic agents or steroids and with uncontrolled hypertension, migraine, or other primary headaches.

3.1. Methods

Patients were subjected to thorough history taking including a comprehensive history of previous headaches and pharmacological treatment history. They also had a complete neurological examination and MRI cervical spine to detect the cause of the CGH. US-guided nerve block was done to all patients.

3.2. Preinjection preparation

Local examination of the area on injection was done to exclude evidence of cranial defects or other anatomic abnormalities near the target injection area such as scars, skin lesions, and test sensation in the GON dermatome. Initial assessment of patients was done with a 10-point numerical pain scale (from 0, no pain, to 10, the worst pain) and quality of life questionnaire using the Rand-36-Item Health survey. The cases were positioned in a sitting position with the neck bend forward and the head supported. The selection of the injection area was detected by identifying the more painful side. A US machine equipped with a multifrequency linear probe was used.

3.3. Technique

At first, the probe was positioned in the transverse orientation at midline to recognize the external occipital protuberance. The transducer was moved in a caudal direction over the location of C1 to find the C2 spinous process as recognized by its bifid appearance. When C2 was appropriately recognized, the transducer was moved in a lateral direction with the lateral edge of the transducer aimed at the transverse process of C1 to recognize the obliquus capitis inferior (OCI) muscle. The GON was identified as lying superficial to the OCI, traversing the muscle caudal to rostral and lateral to the medial. Before needle placement, the presence or absence of vascular structures as measured with Doppler US was reported. The needle was advanced in-plane with the transducer from medial to lateral

under direct US visualization till the tip was detected in the fascial plane between the OCI and semispinalis capitis. A 25-G, 2-inch spinal needle was used for all injections, a dose of 4 ml consisting of 1 ml of 2 % lidocaine, 2.5 ml of 0.25 % marcaine, and 1 ml of betamethasone was injected. The distribution of injectate was visualized as it included the GON between the two muscles. Determination of a successful GON block was done after 30 min after the injection when light-touch sensation was absent with regard to the GON dermatome.

3.4. Outcome evaluation

Pain measurement was assessed by patients' ratings of a 10-point numerical pain scale (from 0, no pain, to 10, the worst pain) and quality of life questionnaire using the Rand-36-Item Health survey v1.0 questionnaire.

3.4.1. Pain intensity assessment

Pain intensity was evaluated preinjection, half an hour postinjection, 2 weeks postinjection, and 4 weeks postinjection using a numeric rating scale marked from 0 to 10 with fixed intervals. The preinjection and 30-min postinjection pain ratings were acquired in the pain clinic. The 2-week and 4-week follow-up pain ratings were acquired using the telephone (Krebs *et al.*, 2007; Shim *et al.*, 2011).

3.4.2. Complications

At all follow-up time points following the GON block, we inquired about the development of complications over the telephone. Manifestations addressed comprised dizziness, blurring of vision, and local edema.

3.5. Ethical consideration

The current study was approved by IRB MS.21.07.1593, Faculty of Medicine, Mansoura University for approval. Informed verbal consent was acquired from all patients after confirmation of confidentiality and personal privacy. The data collected from cases were not used in different aims rather than the current study.

3.6. Statistical analysis

Data analysis was conducted by SPSS software (PASW statistics for Windows, version 18; SPSS Inc., Chicago, Illinois, USA). Qualitative data were described using numbers and percentages. Quantitative data were described using median in the context of non-normally distributed data and mean ± SD for normal distribution of data following assessing normality using the Kolmogrov–Smirnov test. Significance of the obtained results was judged at the 0.05 level. χ^2, Fisher's exact test, and Monte Carlo tests were used to compare qualitative data between groups as appropriate. Mann–Whitney *U* test was used for comparison between two studied groups and more than two studied groups, respectively, for non-normally distributed data.

4. Results

The present study was a prospective interventional study carried out on 50 patients with CGH treated by US-guided nerve block. Table 1 demonstrates that the mean age of the studied cases was 47.62 and the SD was 11.13 ranging from 23 to 68 years, 80 % were female, 18 % diabetic, 12 % diabetic and hypertensive, and 70 % had no associated comorbidities.

The percent of improvement in the numeric pain scale was highest after 30 min (74.4 %) followed by after 2 weeks (60.5 %), after 4 weeks (50 %), and after 8 weeks (39.8 %). Table 2 demonstrates that there was a statistically significant decrease in the numeric pain scale from 6.64 pretreatment to 1.7 after 30 min, then a slight increase to 2.62 after 2 weeks, and a slight increase to 3.32 after 4 weeks, and then increased to 4 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$). There was a statistically significant increase in physical functioning score from 77.04 pretreatment to 96.6 after 30 min, then a slight decrease to 96 after 2 weeks, and a slight decrease to 95.5 after 4 weeks and then a decrease to 94.5 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$). There was a statistically significant decrease in role limitation due to the physical functioning score that changed from

Table 1. Sociodemographic characteristics and medical history of the studied cases.

	$N = 50$ [n (%)]
Age (years)	
Mean ± SD	47.62 ± 11.13
Minimum–maximum	23–68
Sex	
Male	10 (20.0)
Female	40 (80.0)
Medical history	
Free	35 (70.0)
Diabetes mellitus	9 (18.0)
Diabetes mellitus and hypertension	6 (12.0)

Table 2. Numeric pain scale changes, physical functioning, and role limitation changes in physical functioning and emotional functioning between different follow-up points of time.

	$N = 50$	Paired t test
Numerical pain scale		
Pre	6.64 ± 0.89	
After 30 min	1.70 ± 1.07	$t = 32.43$, $P < 0.001$*
After 2 weeks	2.62 ± 1.54	$t = 19.61$, $P < 0.001$*
After 4 weeks	3.32 ± 1.73	$t = 14.59$, $P < 0.001$*
After 8 weeks	4.0 ± 1.84	$t = 10.83$, $P < 0.001$*
Physical functioning		
Pre	77.04 ± 6.51	
After 1 week	96.60 ± 3.97	$t = 18.06$, $P < 0.001$*
After 2 weeks	96.0 ± 3.64	$t = 18.48$, $P < 0.001$*
After 4 weeks	95.50 ± 4.07	$t = 17.06$, $P < 0.001$*
After 8 weeks	94.5 ± 3.68	$t = 16.23$, $P < 0.001$*
Role limitation: physical functioning		
Pre	51.90 ± 6.62	
After 1 week	15.60 ± 7.33	$t = 33.71$, $P < 0.001$*
After 2 weeks	8.80 ± 7.11	$t = 37.75$, $P < 0.001$*
After 4 weeks	31.20 ± 141.38	$t = 1.03$, $P = 0.307$
After 8 weeks	20.90 ± 5.68	$t = 27.34$, $P < 0.001$*
Role limitation: emotional functioning		
Pre	10.40 ± 4.49	
After 1 week	7.21 ± 3.05	$t = 6.26$, $P < 0.001$*
After 2 weeks	9.10 ± 3.14	$t = 2.36$, $P = 0.02$*
After 4 weeks	8.30 ± 2.96	$t = 3.78$, $P < 0.001$*
After 8 weeks	8.80 ± 3.72	$t = 2.61$, $P = 0.012$*

Parameters described as mean ± SD.
*P value comparing each reading with baseline, P value significant if less than or equal to 0.05.

51.9 pretreatment to 15.6 after 1 week, then decreased to 8.8 after 2 weeks and increased to 31.2 after 4 weeks and then decreased to 20.9 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$) except for the physical functioning score measured in the 4th week. There was a statistically significant decrease of role limitation due to the emotional functioning score that changed from 10.4 pretreatment to 7.21 after 1 week, and then increased to 9.1 after 2 weeks, and decreased to 8.3 after 4 weeks and then increased to 8.8 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$).

Table 3 demonstrates that there was a statistically significant decrease in energy score from 39.8 pretreatment to 10.8 after 1 week, then increased to 15.6 after 2 weeks and increased to 16.8 after 4 weeks, and then increased to 21.5 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$). There was a statistically significant increase in emotional well-being from 87.2 pretreatment to 92.24 after 2 weeks, then increased to 92.8 after 4 weeks, and then decreased to 92.24 after

Table 3. Changes in energy, emotional well-being, and social functioning domain of quality of life between different follow-up points of time.

	$N = 50$	Paired t test
Energy		
Pre	39.80 ± 3.77	
After 1 week	10.80 ± 2.70	$t = 40.6$, $P < 0.001$*
After 2 weeks	15.60 ± 5.01	$t = 26.67$, $P < 0.001$*
After 4 weeks	16.80 ± 5.32	$t = 23.48$, $P < 0.001$*
After 8 weeks	21.50 ± 3.53	$t = 21.47$, $P < 0.001$*
Emot-ional well#being		
Pre	87.20 ± 10.16	
After 1 week	90.68 ± 12.05	$t = 1.56$, $P = 0.126$
After 2 weeks	92.24 ± 2.49	$t = 3.55$, $P = 0.001$*
After 4 weeks	92.80 ± 2.51	$t = 3.81$, $P = 0.001$*
After 8 weeks	92.24 ± 2.49	$t = 3.43$, $P = 0.001$*
Social functioning		
Pre	82.50 ± 8.70	
After 1 week	93.0 ± 2.47	$t = 8.01$, $P < 0.001$*
After 2 weeks	93.02 ± 2.98	$t = 8.66$, $P < 0.001$*
After 4 weeks	91.80 ± 2.42	$t = 7.32$, $P < 0.001$*
After 8 weeks	87.98 ± 4.32	$t = 3.76$, $P < 0.001$*

Parameters described as mean ± SD.
*P value comparing each reading with baseline, P value significant if less than or equal to 0.05.

8 weeks. A statistically significant difference was detected for each follow-up in comparison with pretreatment value ($P < 0.001$, each) except for the 1-week value, which demonstrates a nonstatistically significant difference from pretreatment value. There was a statistically significant increase in social functioning from 82.5 pretreatment to 93.0 after 1 week, then increased to 93.02 after 2 weeks and decreased to 91.8 after 4 weeks, and then decreased to 87.98 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$).

Table 4 shows that there was a statistically significant decrease in pain domain of quality of life from 62.4 pretreatment to 13.6 after 1 week, then increased to 21.0 after 2 weeks, decreased to 14.3 after 4 weeks, and then increased to 24.7 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$, each). Table 5

Table 4. Changes in pain domain of quality of life between different follow-up points of time.

Pain	$N = 50$	Paired t test
Pre	62.40 ± 8.99	
After 1 week	13.6 ± 7.15	$t = 30.13$, $P < 0.001$*
After 2 weeks	21.0 ± 6.14	$t = 24.76$, $P < 0.001$*
After 4 weeks	14.30 ± 6.99	$t = 28.67$, $P < 0.001$*
After 8 weeks	24.70 ± 4.78	$t = 28.76$, $P < 0.001$*

Parameters described as mean ± SD.
*P value comparing each reading with baseline, P value significant if less than or equal to 0.05.

Table 5. Changes in general health domain of quality of life between different follow-up points of time.

General health	$N = 50$	Paired t test
Pre	78.54 ± 10.63	
After 1 week	91.34 ± 4.06	$t = 8.31$, $P < 0.001$*
After 2 weeks	92.0 ± 3.19	$t = 8.33$, $P < 0.001$*
After 4 weeks	91.60 ± 2.93	$t = 8.78$, $P < 0.001$*
After 8 weeks	92.30 ± 3.07	$t = 9.19$, $P < 0.001$*

Parameters described as mean ± SD.
*P value comparing each reading with baseline, P value significant if less than or equal to 0.05.

illustrates that there was a statistically significant increase in general health domain of quality of life from 78.54 pretreatment to 91.34 after 1 week, then increased to 92.0 after 2 weeks and decreased to 91.6 after 4 weeks, and then increased to 92.3 after 8 weeks. A statistically significant difference was detected for each follow-up in comparison with the pretreatment value ($P < 0.001$, each). Table 6 demonstrates that there was a statistically significant increase in health change domain of quality of life from 91.74 pretreatment to 95.9 after 2 weeks ($P = 0.009$). Similarly, there is a statistically significant increase in the health change domain of quality of life from 91.74 pretreatment to 97.2 after 4 weeks ($P = 0.001$).

5. Discussion

This study included 50 patients to assess the efficacy of the US-guided GON block in CGH at the Neurology Outpatient Clinic at Mansoura University.

There are many lines of treatment for CGH either noninvasive approaches (pharmacotherapy, physiotherapy, and so on) or invasive therapeutic strategies (such as radiofrequency, acupuncture, and anesthetic block) (Zipfel *et al.*, 2016). Till now, no available strict guidelines for the choice of the regimen for the treatment or for the injectate choice.

Previous reports for the injectate in the literature included: lidocaine 1 and 2 % (10–20 mg/ml), mepivacaine 2 % (20 mg/ml), and bupivacaine 0.25 and 0.5 % (2.5–6 mg/ml) as short-term, medium-term, and long-term treatments correspondingly. Also injecting corticosteroids (such as triamcinolone, methylprednisolone, betamethasone, and dexamethasone) in combination with anesthetics can prolong the duration of analgesia by almost 6 h. We used in our study a 4 ml combination injectate (consisting of 1 ml of 2 % lidocaine, 2.5 ml of 0.25 % marcaine, and 1 ml of betamethasone) was used.

Table 6. Changes in the health change domain of quality of life between different follow-up points of time.

Health change	$N = 50$	Paired t test
Pre	91.74 ± 10.83	
After 1 week	94.36 ± 5.29	$t = 1.52$, $P = 0.136$
After 2 weeks	95.90 ± 4.37	$t = 2.71$, $P = 0.009$*
After 4 weeks	97.2 ± 3.66	$t = 3.43$, $P = 0.001$*
After 8 weeks	92.20 ± 3.66	$t = 0.282$, $P = 0.779$

Parameters described as mean ± SD.
*P value comparing each reading with baseline, P value significant if less than or equal to 0.05.

In our study, pain improved significantly after injection using a numeric pain scale and quality of life questionnaire (Rand-36 questionnaire). Pain improvement declined gradually with time but was still significantly better 8 weeks after injection. Numeric pain scale scores decreased by 74.4, 60.5, 50, and 39.8 % 30 min, at 2 weeks, 4 weeks, and 8 weeks after injection, respectively ($P < 0.001$). Also, pain domain of the quality of life questionnaire showed a statistically significant decrease in each follow-up in comparison with the pretreatment value ($P < 0.001$).

Previous reports in the literature revealed variable percentages of pain improvement after the GON block.

In Vincent *et al.* (1998), using 1–2 ml 0.5 % bupivacaine injection at the GON, visual analog scale (VAS) improved by 50 % at 1 week after injection.

Haspeslagh *et al.* (2006) found that radiofrequency was not superior to local injection as VAS, quality of life scores were assessed at 8, 16, 24, and 48 weeks and found that the VAS and the Rand-36 scale improvement were not significantly different between both groups.

The Naja *et al.* (2006) study used the VAS to assess pain improvement after nerve stimulator-guided occipital nerve block. In their study, VAS was reduced by approximately 50 % from baseline after 2 weeks.

The Gabrhelik *et al.* (2011) study assessed VAS and Medication Quantification Scale at 3 months post-therapy and found a significant reduction in VAS by 58 % ($P < 0.001$) and by 21 % at 9 months. When compared with baseline scores, the consumption of analgesic medication was decreased significantly at 3 months by 47 % ($P < 0.001$) and at 9 months by 26 % ($P < 0.01$).

Similarly in the study by Lauretti *et al.* (2015), assessment of the quality of life scale and VAS after using different techniques (classical and subcompartemental) using 5 ml consisted of a mixture of 10 mg dexamethasone, 40 mg lidocaine, and saline. The classical GON technique resulted in a significant decrease in VAS and Rand-36 by 90 % after 2 weeks ($P < 0.01$) and by 60 % after 24 weeks.

In Sahin *et al.* (2016), VAS decreased by 66.6 % after GONB combined with bupivacaine and

dexamethasone after the first block and the attack period was decreased at 88 % rate, with no decrease at the attack frequency, following the third blockade. It was noticed that pain frequency has reduced at a rate of 43.75 %, period at 72.91 %, and intensity at 50 %.

Also in the study by Ertem and Yilmaz (2019), 3–4 ml of 2 % lidocaine and 1 ml of methylprednisolone acetate were used. A significant reduction in numeric pain scale ($P < 0.001$) was recognized. The number of headaches decreased in a significant manner at 3 months ($P < 0.001$).

Also, Mohamed *et al.* (2021) found that VAS improved by 48 % ($P = 0.001$) and headache frequency reduced by 34 % after 2 weeks; VAS improved by 42 % ($P = 0.020$), and headache frequency reduced by 31 % after 4 weeks after GONB.

There was significant improvement in the numeric pain scale after 1 month by 60 %, and by 77 % after 3 months, and by 70 % after 6 months. The quality of life domains improved after pain reduction was reported but with Oswestry disability index by Ismail and Abdul Wahab (Ismail and Abdul Wahab, 2022).

This rapid pain improvement after injection in our study and in the previous literature work can be explained as the pain is due to a vicious circle of painful stimuli and nonpainful stimuli as the neck movement due to irritation of the dorsal horn and sudden interruption of this circle due to the nerve block caused the rapid dramatic improvement till the primary lesion would reestablish the abnormal reduction in pain threshold. In addition, it may be the etiology of why various structures may be blocked with comparable actions, as the significance of the anesthesia is mainly reliant on the drop of the exaggerated total sensory input, instead of the block site.

Another mechanism of pain propagation happens when Na + molecules attach to receptors on the nerve cells. When enough of these receptors are stimulated, a pain signal travels from one nerve cell to another, all the way to the brain. Lidocaine (short-acting, rapid onset) and Marcaine (long-acting, delayed onset) work by preventing Na+ from attaching to the nerve's receptor and betamethasone is anti-inflammatory that prevents local side effects and prolongs the action of local anesthesia.

However, Vincent *et al.* (1998) disagreed with this finding as they found that pain fades directly after injection, returns to levels sometimes greater than the original one during the following 1–2 days, and after that tends to fade another time for a different period of time. They called this a 'tilde pattern' owing to its resemblance to the tilde (~) mark. They explained this phenomenon by the possibility that the pain is associated with the trauma induced by the injection owing to the distention the injected liquid approaches in the area.

This propensity to pain recurrence was detected in the initial 48 h following the block, was in the subgroup of cases with an initial pain of more than 50 (VAS), and may be due to using only 1–2 ml 0.5 % bupivacaine injection, which has a slower onset of action and a longer duration. It causes mild vasoconstriction and therefore delays absorption and action, and it was blind not US-guided nerve block.

The dramatic pain improvement in our study was reflected in the patients' quality of life (using the Rand-36 questionnaire) as all domains of the Rand-36 questionnaire (energy, emotional well-being, social functioning, pain domain of quality of life, general health, and health change domain of quality of life) showed statistically significant improvement after 1, 2, 4, and 8 weeks as the pain domain has two items affecting the Rand-36 scale (how much pain during the past 4 weeks, how pain interfered with normal life), how pain increased physical limits and decreased emotional well-being.

Generally, the use of the US as a guide in injection procedures added more accuracy and safety to those procedures and, of course, efficacy. Some complications of US-guided injection procedures were reported in the literature including local sensitivity and pain, local edema, hypotension, syncope, local hematoma, local sensitivity, and edema Sahin *et al.* (2016). In our study, we faced only local edema in old age patients, and it was mild and improved after 24 h with anti-inflammatory drugs.

5.1. Limitations of the study

(1) The study did not assess headache frequency, period, or use of repetitive nerve block, or compare different types of injectates or compare nerve block with other methods. It needs to compare outcomes after unilateral and bilateral blocks and needs a longer duration for assessment, a large number of samples, and a control group.

5.2. Conclusion

In the context of CGH, US-guided GONB has been demonstrated to be a novel, safe, and an effective treatment method, which was associated with pain reduction (as revealed by the numeric pain scale) and better quality of life (as revealed by Rand-36).

Conflicts of interest

There are no conflicts of interest.

References

Al Khalili, Y., Jain, S., Murphy, P.B., 2018. Headache, cervicogenic. StatPearls 3, 1–11.

Arnold, M., 2018. Headache classification committee of the international headache society (IHS) the international classification of headache disorders. Cephalalgia 1, 1–211.

Becker, W.J., 2010. Cervicogenic headache: evidence that the neck is a pain generator. Headache 4, 699–705.

Diaz, D., Moore, C., Kane, A., 2019. Physical therapy management of adults with mild traumatic brain injury. Semin. Speech Lang. 1, 36–47.

Ertem, D.H., Yilmaz, I., 2019. The effects of repetitive greater occipital nerve blocks on cervicogenic headache. Turk J Neurol 25, 82–86.

Gabrhelík, T., Michálek, P., Pieran, M., 2011. F723 pulsed radiofrequency therapy versus greater occipital nerve block in the management of refractory cervicogenic headache – a pilot study. Eur J Pain Suppl 5 (1), 194.

Haspeslagh, S.R., Van Suijlekom, H.A., Lame, I.E., Kessels, Alfons G.H., van Kleef, Maarten, Weber, Wim E.J., 2006. Randomised controlled trial of cervical radiofrequency lesions as a treatment for cervicogenic headache [ISRCTN07444684]. BMC Anesthesiol 6, 1–11.

Headache Society, 2018. The international classification of head ache disorder. Cephalalgia 1, 1–211.

Ismail, D.M., Abdul Wahab, S.M., 2022. Greater occipital nerve block for chronic neck pain provides extended pain relief and improves quality of life: comparative study versus digital manual therapy. SVU-Int J Med Sci 2, 586–598.

Krebs, E.E., Carey, T.S., Weinberger, M., 2007. Accuracy of the pain numeric rating scale as a screening test in primary care. J Gen Intern Med 22, 1453–1458.

Lance Whorton, R., Kegerreis, S., 2000. The use of manual therapy and exercise in the treatment of chronic cervicogenic headaches: a series of case studies. J Manual Manipulat Ther 4, 193–203.

Lauretti, G.R., Correa, S.W., Mattos, A.L., 2015. Efficacy of the greater occipital nerve block for cervicogenic headache: comparing classical and subcompartmental techniques. Pain Pract, 7, 654–661.

Maureen, D., 2017. Cervical MRI Scan, what is a cervical MRI scan? Healthline 12, 1–12.

Mohamed, Z.E., Zarad, C.A., Flifel, M.E., Mohamed, Zenat Eldadamony, Zarad, Carmen Ali, Abou Elmaaty, Ali A., 2021. The efficacy of ultrasound-guided multifidus cervicis plane block versus greater occipital nerve block for cervicogenic headache. Egypt J Neurol Psychiatry Neurosurg 57, 1–6.

Naja, Z.M., El-Rajab, M., Al-Tannir, M.A., Ziade, Fouad M., Tawfik, Omar M., et al., 2006. Occipital nerve blockade for cervicogenic headache: a double-blind randomized controlled clinical trial. Pain Pract 2, 89–95.

Sahin, B.E., Coskun, O., Üçler, S., Inan, Nurten, İnan, Levent Ertuğrul, Özkan, Şeyma Nur, 2016. The responses of the greater occipital nerve blockade by local anesthetics in headache patients. J Neurol Sci 1, 33.

Shim, J.H., Ko, S.Y., Bang, M.R., Shim, Jae Hang, Ko, So Young, Bang, Mi Rang, Jeon, Woo Jae, et al., 2011. Ultrasound-guided greater occipital nerve block for patients with occipital headache and short term follow up. Korean J Anesthesiol 1, 50–54.

Vincent, M.B., Luna, R.A., Scandiuzzi, D., Vincent, M.B., Peres, M., Barros, M.C., Vincent, J.M., 1998. Greater occipital nerve blockade in cervicogenic headache. Arq Neuropsiquiatr 56, 720–725.

Yaksh, T.L., 1993. The spinal pharmacology of facilitation of afferent processing evoked by high-threshold afferent input of the postinjury pain state. Curr Opin Neurol Neurosurg 2, 250–256.

Zipfel, J., Kastler, A., Tatu, L., Behr, J., Kechidi, R., Kastler, B., 2016. Ultrasound-guided intermediate site greater occipital nerve infiltration: a technical feasibility study. Pain Physician 7, E1027.