

ORIGINAL ARTICLE

Comparative Efficacy of Greater Occipital Nerve Blockade and Sphenopalatine Ganglion Blockade in Patients with Episodic Migraine: A Randomized Trial**Ahmed O. M. Abdallah¹, Adel Alqarni², Ahmed A. Ahmed³, Mahmoud A. M. Eisa⁴, Mostafa H. H. Bakr⁴***Anesthesia and Intensive Care and Pain Management Department, Faculty of Medicine, ¹New Valley University, New Valley, ⁴Assiut University, Assiut, Egypt, ²College of Medicine, King Saud University, ³King Saud University Medical City, Riyadh, Saudi Arabia.***Correspondence to:** *Ahmed O. M. Abdallah; MD, Lecturer of Anesthesia and Intensive Care and Pain Management Department, Faculty of Medicine, New Valley University, New Valley, Egypt.
E-mail: twisy200235@med.nvu.edu.eg***Background**

Episodic migraine significantly impairs quality of life, and some cases fail to respond to conventional prophylactic treatments. Alternative therapy approaches are provided by interventional nerve blocks, such as sphenopalatine ganglion (SPG) and greater occipital nerve (GON) blockades. This work compared the effectiveness of GON blockade and SPG blockade in reducing headache intensity, duration, frequency, and disability in cases with episodic migraine.

Methods

This study was prospective, randomized, controlled, and single-blind and was conducted on 60 cases, both sexes, aged 18-65 years, diagnosed with episodic migraine per the international classification of headache disorders, 3rd edition criteria, and failed to achieve adequate relief with at least one prophylactic migraine treatment. Cases were randomly assigned into two groups to receive either the GON blockade in the GON group or the SPG blockade in the SPG group. Blocks were performed using 2mL of 2% lidocaine.

Results

Intensity, migraine disability assessment scores, and number of NSAIDs per day were significantly lower at 1, 2, and 3m in the GON Group as opposed to the SPG Group ($P<0.05$). Migraine duration and the reduction in the number of NSAIDs per day were notably increased in the GON Group in contrast to the SGP Block Group ($P<0.05$). Adverse events (nasal irritation, temporary difficulty in swallowing, vasovagal reaction) and patient satisfaction were comparable.

Conclusions

GON blockade is a superior short-term intervention for cases unresponsive to standard prophylactic therapy, offering greater reductions in headache intensity, frequency, and disability as opposed to SPG blockade.

Keywords

Episodic Migraine, Headache Intensity, Occipital Nerve, Pain Management, Sphenopalatine Ganglion.

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INTRODUCTION

Migraine is a long-term neurological condition marked by repeated headache episodes, impacting millions worldwide [1].

Similarly, the sphenopalatine ganglion (SPG), located in the pterygopalatine fossa and composed of sensory, sympathetic, and parasympathetic fibers, is involved in the autonomic components of migraines [2].

Although both GON and SPG blocks have demonstrated varying degrees of effectiveness, direct head-to-head comparisons are limited [3,4].

In this study, the efficacy of SPG and GON blocking was compared in lowering the incidence, severity, duration, and impairment of headaches in people with episodic migraine.

MATERIAL AND METHODS

This study was prospective, randomized, controlled, and single-blind and was conducted on 60 cases, both sexes, aged 18-65 years, diagnosed with episodic migraine per the International Classification of Headache Disorders, 3rd edition (ICHD-3) and failed to obtain adequate relief with at least one prophylactic migraine treatment. The research was conducted between May 2025 to May 2026, following the approval from the ethical committee (approval code: 2025430007). This study was done according to Declaration of Helsinki. The cases provided informed written consent.

Persistent migraine (at least 15 headache days per month), history of nerve blocks within the last 6 months, allergy to anesthetic agents, pregnancy, lactation, and active psychiatric conditions affecting compliance were excluded.

Prior to the intervention, all participants underwent a comprehensive review of their medical history, clinical examination, and laboratory testing. Furthermore, they were familiarized with the numerical rating scale (NRS) for pain assessment to ensure that they could accurately report their pain level.

Randomization and blinding:

To maintain the integrity of the study, a random allocation process was utilized, employing computer-generated numbers (<https://www.randomizer.org>). Each participant's code was placed in an opaque, sealed envelope to preserve blinding. The cases were randomly assigned into two groups (1:1 ratio) to receive either the GON blockade in the GON Group or the SPG blockade

in the SPG group. To maintain the blinding, the outcome assessor was blind to the performed block.

Two mL of 2% lidocaine were injected into the GON Group at the medial third of the line that separates the mastoid process from the occipital protuberance. This procedure targeted the greater occipital nerve, which originates from the C2 and C3 spinal roots and connects with the trigeminocervical complex, a key structure in migraine pathophysiology. By blocking this nerve, the intervention aimed to reduce pain transmission and neurogenic inflammation. The administration schedule for this group consisted of weekly injections for four weeks, followed by monthly injections for two months.

The SPG Group underwent a transnasal administration of 2mL of 2% lidocaine utilizing a cotton swab placed at the middle superior turbinate to reach the posterior nasopharyngeal wall. The pterygopalatine fossa contains the sphenopalatine ganglion, which is essential for the autonomic control of the cranial vasculature. Inhibiting vasodilation and the transmission of migraine-related pain signals was the goal of blocking this ganglion. The treatment schedule mirrored that of the GON group: once weekly for four weeks, then monthly treatments for two months.

Baseline demographic and clinical characteristics were recorded for all participants, including migraine history, frequency, and severity. The primary outcomes assessed included reductions in headache intensity (measured using the Numeric Rating Scale), headache duration and frequency, and improvements in Migraine Disability Assessment scores (MIDAS). Secondary outcomes involved the reduction of acute medication use and the documentation of any adverse events (AEs).

The study timeline comprised an initial baseline assessment, followed by weekly interventions for four weeks and monthly sessions during the subsequent two months. Follow-up evaluations were performed at one, two, and three months to assess clinical progress and treatment outcomes.

The study's primary outcome was to assess the NRS. In contrast, secondary outcomes encompassed assessing the impact of both interventions on the use of acute migraine medications, evaluating patient-reported outcomes related to disability and quality of life, monitoring the safety and tolerability of both interventions, determine if either intervention leads to prolonged remission of episodic migraine and compare the rate of AEs between the two treatment groups.

Sample size:

Using G*Power 3.1.9.2 (Universitat Kiel, Germany), we were able to estimate the sample size. Based on our pilot investigation, which included five cases in each group, we discovered that the average (\pm SD) of NRS was 4.2 ± 1.3 in GON and 5.4 ± 1.4 in SPG. Each group had 30 cases based on an effect size of 0.89, a 1:1 allocation ratio, a 80% power, and a 95% confidence limit in the study. Two cases were added to each group to combat dropout.

Statistical analysis:

SPSS version 27 (IBM®, Chicago, IL, USA) was used for all statistical analyses. The Shapiro-Wilk test, in conjunction with histogram visualization, was used to determine normality in the data distribution. The unpaired Student's *t*-test was used to analyze the parametric data, which were presented as mean \pm standard deviation (SD). Group differences were examined using the Mann-Whitney *U* test, and non-parametric data were presented as the median and interquartile range (IQR). The Chi-square test was used to evaluate correlations between categorical variables, which were displayed as frequencies and percentages. Fisher's exact test was used for categorical variables when expected cell counts were less than 5 or for 2 \times 2 contingency tables with small sample sizes. Throughout the analysis, a two-tailed $P \leq 0.05$ was regarded as suggestive of statistical significance.

RESULTS

Out of 77 cases initially evaluated for eligibility, 11 did not fit the requirements for inclusion, and six declined to participate. The remaining 60 participants were randomly assigned into two equal groups of 30 cases each. All enrolled cases completed follow-up and were included in the final statistical analysis (Figure 1).

At baseline, the groups were comparable in terms of age, gender, migraine history, frequency, severity, or use of prophylactic medications (Table 1).

Pain intensity and MIDAS scores were also comparable at baseline. However, at 1-, 2-, and 3-months post-intervention, both intensity and MIDAS scores were greatly decreased in the GON Group as opposed to the SPG Group ($P < 0.05$). Duration of the headache was significantly extended in the GON Group at baseline and 1 month ($P < 0.05$), but comparable durations were observed between the groups at 2 and 3 months (Table 2).

The number of NSAID doses per day was similar between groups at baseline. At 1, 2, and 3 months, NSAID use was substantially reduced in the GON Group, with a greater reduction in daily NSAID intake as opposed to the SPG Group ($P < 0.05$) (Table 3).

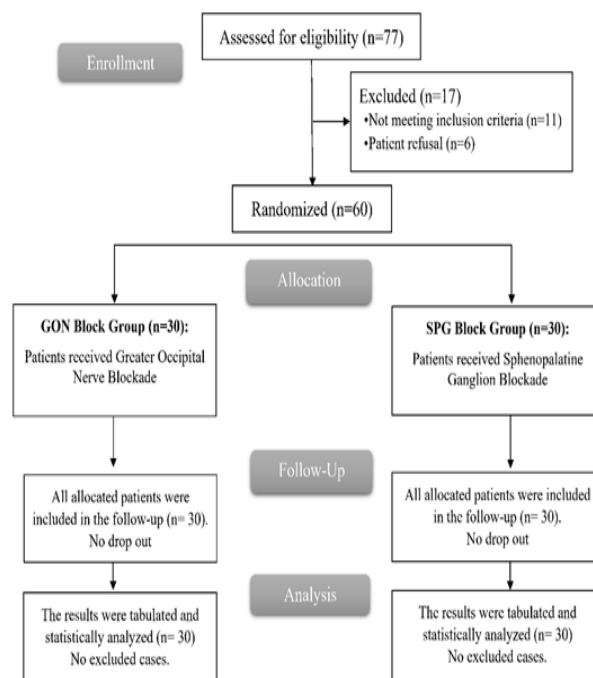


Fig. 1: CONSORT flowchart of the enrolled patients.

Table 1: Demographic data, migraine history, frequency, severity and prophylactic medication of the studied groups:

		GON Group (n= 30)	SPG Group (n= 30)	P
Age (years)		38.03 \pm 11.67	39.93 \pm 13.64	0.564
Sex	Male	10(33.33%)	12(40%)	0.592
	Female	20(66.67%)	18(60%)	
Migraine history		13(43.33%)	12(40%)	0.793
	One	8(26.67%)	6(20%)	
Migraine frequency/day	Two	10(33.33%)	15(50%)	0.424
	More	12(40%)	9(30%)	
	Mild	13(43.33%)	6(20%)	
Migraine severity	Moderate	11(36.67%)	15(50%)	0.150
	Severe	6(20%)	9(30%)	
Prophylactic medication	Beta-blockers	14(46.67%)	9(30%)	0.267
	Ca channel Blockers	11(36.67%)	14(46.67%)	
	TCA	5(16.67%)	6(20%)	
	SSNRI	7(23.33%)	5(16.67%)	
	VPA	3(10%)	4(13.33%)	1.000

Data are presented as mean \pm SD or frequency (%); GON: Greater occipital nerve; SPG: Sphenopalatine ganglion; Ca: Calcium; TCA: Tricyclic antidepressants; SSNRI: Selective serotonin-norepinephrine reuptake Inhibitors; VPA: Valproic acid; Statistical test: Chi-square test, Fisher's exact test, unpaired *t*-test; N.B. Patients may be on multiple prophylactic medications simultaneously.

Treatment failure rates were comparable (at 1, 2, or 3 months) between the two groups (Table 4).

AEs—such as nasal irritation, temporary swallowing difficulty, and vasovagal reactions—as well as patient satisfaction, were comparable across both groups (Table 5).

Table 2: Intensity, duration and MIDAS of the studied groups:

	GON Group (n= 30)	SPG Group (n= 30)	P
Intensity (NRS)			
Baseline	6(6-7)	6(5-7)	0.150
1m	4(4-5)	5.5(5-6)	0.001*
2m	4(3-5)	4(3.25-5)	0.047*
3m	4(3-4)	5(3-5)	0.001*
Duration (days)			
Baseline	29(18-34.5)	16.5 (11.25-29)	0.022*
1m	14(7.25-17)	7(2.25-13.75)	0.016*
2m	7.5(2.25-9.75)	4(2-8)	0.286
3m	6(2-8)	3(2-4.75)	0.155
MIDAS			
Baseline	17.5(12-23.75)	19(14-24.75)	0.514
1m	11(6-18.5)	17(11.25-22)	0.010*
2m	4(2.25-8.75)	11.5(6.25-19)	<0.001*
3m	2(1.25-5)	8.5(3.25-15.75)	<0.001*

Data are presented as median (IQR); *: Significant when *P* value ≤0.05; GON: Greater occipital nerve; SPG: Sphenopalatine ganglion; NRS: Numerical rating scale; MIDAS: Migraine disability assessment score; Statistical test: Mann-Whitney *U* test .

Table 3: Number of NSAID per day and reduction in number of NSAID/day of the studied groups:

	GON Group (n= 30)	SPG Group (n= 30)	P
Number of NSAID/day			
Baseline	5(4.25-6)	5(4-6)	0.768
1m	2.5(2-3)	3 (3-4)	0.003*
2m	2(1.25-2)	2(1.25-4)	0.046*
3m	1(1-2)	2(1-3)	0.034*
Reduction in number of NSAID/day			
1m	2.53±0.78	1.4±0.62	<0.001*
2m	3.13±0.94	2.1±1.27	<0.001*
3m	3.4±1.07	2.6±1.28	0.011*

Data are presented as median (IQR); *: Significant when *P* value ≤0.05; GON: Greater occipital nerve; SPG: Sphenopalatine ganglion; NSAID: Non-steroidal anti-inflammatory drugs; Statistical test: Mann-Whitney *U* test, unpaired *t*-test.

Table 4: Failure rate of the studied groups:

	GON Group (n= 30)	SPG Group (n= 30)	P
1m	3(10%)	6(20%)	0.472
2m	5(16.67%)	7(23.3%)	0.519
3m	4(13.33%)	5(16.67%)	1

Data are presented as frequency (%); GON: Greater occipital nerve; SPG: Sphenopalatine ganglion; Statistical test: Fisher's exact test.

Table 5: Adverse events and patient satisfaction of the studied groups:

	GON Group (n= 30)	SPG Group (n= 30)	P
Adverse events			
Nasal irritation	0(0%)	2(6.67%)	0.491
Temporary difficulty in swallowing	0(0%)	2(6.67%)	0.491
Vasovagal reaction	1(3.33%)	0(0%)	1
Patient satisfaction			
Extremely dissatisfied	0(0%)	1(3.33%)	0.743
Dissatisfied	1(3.33%)	3(10%)	
Neutral	4(13.33%)	4(13.33%)	
Satisfied	9(30%)	8(26.67%)	
Extremely satisfied	16(53.33%)	14(46.67%)	

Data are presented as frequency (%); GON: Greater occipital nerve; SPG: Sphenopalatine ganglion; Statistical test: Fisher's exact test.

DISCUSSION

The present study demonstrated that cases who received GON blocks exhibited significantly greater reductions in multiple headache-related parameters (intensity, duration, frequency, and scores on the MIDAS scale) when compared with those who received SPG blocks. Furthermore, the GON group demonstrated a substantial decline in the daily use of NSAIDs. Importantly, both treatment modalities were well-tolerated, with no statistically significant differences observed between the groups in terms of AEs or levels of patient-reported satisfaction.

These findings are consistent with the results reported by Unal and colleagues [5]. who observed that GON block recipients experienced more pronounced reductions in headache intensity, duration, frequency, and MIDAS scores relative to SPG block recipients. They also reported a significant decline in NSAID consumption in the GON group, while both treatment groups demonstrated comparable safety profiles and levels of patient satisfaction.

The current literature offers substantial support for the clinical efficacy of GON blocks. For instance, Chowdhury and colleagues [6]. found a notable decline in the number

of days with headaches over three months following weekly administration of 2% lidocaine GON blocks for four weeks in cases with chronic migraine. Similarly, Inan and colleagues [7], demonstrated that a GON block protocol—consisting of weekly injections for one month followed by monthly administrations for an additional two months—resulted in markedly decreases in headache intensity, frequency, and duration. Complementary findings were reported by Ulusoy and colleagues [8], who observed meaningful improvements in case functionality over the course of a three-month treatment period. A longer-term benefit was also observed in another trial by Okmen and collaborators [9], which documented sustained therapeutic benefits persisting up to six months after the final GON injection, including continued reductions in headache intensity, frequency, and associated disability.

These findings are further corroborated by a meta-analysis conducted by Zhang and colleagues [10], which concluded that GON blocks are effective in mitigating headache intensity and reducing the number of days requiring analgesic use. However, no statistically significant impact was found in the investigation on headache duration, suggesting some variability in treatment outcomes.

While SPG blocks have received comparatively less empirical attention, their use is underpinned by a well-established anatomical and pathophysiological basis. Anatomically, the SPG is located posterior to the middle nasal turbinate and is the largest extracranial parasympathetic ganglion in the cranial cavity. It serves as a key component of the trigeminal-parasympathetic reflex arc, which is implicated in the pathogenesis of migraine through its mediation of neurogenic inflammation and cranial vasodilation [11,12]. Acetylcholine, nitric oxide, and vasoactive intestinal peptide (VIP) are among the vasoactive and pro-inflammatory mediators released when the SPG is activated, and these mediators all play a part in the migraine cascade [13-15]. By administering a local anesthetic to the SPG, this pathophysiological process can be interrupted, thereby attenuating migraine-inducing stimuli such as olfactory triggers, sleep disturbances, and psychological stress.

Multiple techniques are available for performing SPG blocks, including subzygomatic, intraoral, and transnasal approaches. The subzygomatic route, while potentially more precise, is also technically demanding and invasive. In contrast, the transnasal technique is relatively straightforward and cost-effective, often preferred in outpatient settings. Anesthetic delivery can be achieved using specialized devices such as the Spenocath or by utilizing simple cotton-tipped applicators to apply the anesthetic across the thin (1–2mm) nasal mucosa overlying the SPG [11,16].

Kim and colleagues [17], carried out a study comparing topical and drip application methods. They found that the cotton swab technique more effectively inhibited parasympathetic activity, which is why this method was chosen in the present study.

In contrast, GON blocks target the trigeminocervico vascular system by inhibiting nociceptive input at the trigeminal nucleus caudalis, thus reducing the release of neuroinflammatory mediators and preventing subsequent vasodilation and inflammation [18,19]. The procedure can be performed using either a proximal approach at the C2 vertebral level under ultrasound guidance or a distal technique using anatomical landmarks. Karaoglan and colleagues [20], noted that the proximal method may be more effective in reducing headache days; however, the distal approach was selected in our study due to its practicality in outpatient settings and lack of requirement for ultrasound guidance.

While the full therapeutic effect of preventive migraine medications may take 2–6 months to become apparent [21], our study included cases that had already failed at least one prophylactic therapy. This strengthens the likelihood that the observed improvements in headache frequency and severity were attributable to the interventional block treatments rather than delayed pharmacological effects.

LIMITATIONS

This study has several notable limitations. The modest sample size limited the ability to perform detailed subgroup analyses, and the single-center, single-blind design may restrict generalizability and introduce bias, especially in patient-reported outcomes. The short three-month follow-up period prevents assessment of long-term efficacy and safety. Additionally, the absence of a placebo or sham control group means true treatment effects cannot be clearly separated from placebo responses, so results should be interpreted with caution.

FUTURE DIRECTIONS

Future studies should use double-blind, sham-controlled designs, include larger and more diverse patient populations across multiple centers, and extend follow-up to 6–12 months or longer. Objective outcome measures and cost-effectiveness analyses should complement subjective reports, and patient stratification may help identify those most likely to benefit from each treatment.

CONCLUSIONS

GON blockade is a superior short-term intervention for cases unresponsive to standard prophylactic therapy,

offering greater reductions in headache intensity, frequency, and disability as opposed to SPG blockade. Over 3 months, cases receiving GON blockade reported significantly improved pain scores, lower MIDAS scores, and reduced reliance on acute medications.

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