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# Supraorbital and infraorbital nerve blockade in migraine patients: results of 6-month clinical follow-up

S. ILHAN ALP, R. ALP*

Department of Neurology, Corlu State Hospital, Tekirdag, Turkey
*Department of Neurology, Faculty of Medicine, Namık Kemal University, Tekirdag, Turkey

**Abstract. – BACKGROUND: Nerve blockades are used for the treatment of acute migraine episodes in emergency room conditions and beneficial results are obtained from this clinical use. Although this is the case, there are limited numbers of studies investigating the long-term effects of such an approach.**

**PATIENTS AND METHODS: In this investigation, we had 26 patients diagnosed as migraine based on the ICHD II criteria, these were injected with 1% lidocaine at supraorbital and infraorbital nerve localizations and clinical results were evaluated after 6 months of follow-up. All patients received 1.5 ml of 1% lidocaine bilaterally for supraorbital and infraorbital nerves with three day intervals for three times. Clinical evaluation was conducted by recording the number of migraine episodes per month together with migraine disability assessment scale (MIDAS) and visual analog scale (VAS) scores before and six months after the treatment.**

**RESULTS: Mean age of the patients recruited in the study was 31.1±10.2 years. Disease duration was 8.1±5.4 years, the duration of the headache was 28.4±18.4 hours, mean number of episodes before treatment was 9.9±5.2, mean MIDAS was calculated as 3.2±0.8, and VAS as 9.0±1.0. Six months after the treatment, mean number of attacks was 2.0±3.0, MIDAS was 1.4±0.9 and VAS was 3.5±3.6. There was a statistically significant difference between the results obtained before and after the treatment.**

**CONCLUSIONS: Injecting 1% of lidocaine to supraorbital and infraorbital nerve for three times prevents the acute migraine episodes effectively during the 6-months of follow-up without having any significant side effects**

*Key Words:*
Migraine, Supraorbital nerve blockade, Infraorbital nerve blockade, Lidocaine, Local anaesthetic blockade.

## Introduction

Migraine is a common, disabling, primary neurovascular disorder characterized by episodic, severe headaches and systemic or neurological symptoms. The molecular mechanisms of migraine have not been clearly explained yet[1]. Both central (cortex and upper brainstem) and peripheral (trigeminovascular) mechanisms are being held responsible for migraine attacks[2,3].

Migraine has a prevalence of 10% in the general population and has a considerably high cost to the society[4]. Based on the results obtained in the European Union, there is an indirect cost of 27 million euros resulting from migraine related treatment costs, decreases in work efficiency and loss of man power and the figures are quite striking. This means that the cost of migraine is equal to that of dementia and stroke while being higher than those of movement disorders, multiple sclerosis and epilepsy[5,6].

In this study, we investigated the long term effects of lidocaine injection to supraorbital and infraorbital nerves and this treatment option is easy to use on outpatients and has low cost.

## Patients and Methods

### Patients

The study included 26 patients diagnosed as migraine based on ICDH-II (2nd Edition of The International Headache Classification) criteria. The patients were admitted to Neurology Outpatient Unit and agreed to receive lidocaine treatment. The study was initiated after receiving the approval of local Ethics Committee and the patients were followed-up for 6 months.

The patients participating in the study did not receive any drug and did not use any treatment of prophylactic nature during the course of the study. They did not have any disease other than migraine and none of them were pregnant. None received a psychiatric treatment.

### Injection Procedure

All patients were injected with 1% lidocaine bilaterally at supraorbital and infraorbital nerves



*Corresponding Author:* Selen Ilhan Alp, MD, Ph.D.; e-mail: recepalp@hotmail.com

with three day intervals for three times. Supraorbital nerve blockade was performed at a position 2.5 cm lateral to the midline towards the supraorbital foramen located on the upper border of the orbita immediately below the eyebrow with a 23-25 gauge needle to receive 1.5 ml of 1% lidocaine. Upper lip was lifted to guide the injector parallel to the long axis of the second premolar, from the point where the mucosa met the gingival a 23-25 gauge needle was inserted towards the infraorbital foramen to inject 1.5 ml of 1% lidocaine.

### Following Patient Data

Demographic data, the history of the headache, MIDAS (Migraine Disability Assessment scale) and VAS (Visual Analogue Scale) was documented for all patients. They were provided with follow-up charts and were asked to record the number of episodes together with their intensities. The patients first had weekly and monthly controls for a period of six months. Clinical follow-up was performed with the number of monthly episodes, monthly VAS scores, MIDAS scores before and six months after treatment.

### Statistical Analysis

Results were expressed as mean ± standard deviation. Differences were considered significant at a probability level of $p < 0.05$. Student's *t*-test was performed using SPSS for Windows, Release 11.5 computer program (SPSS Inc., Chicago, IL, USA).

## Results

Of the 26 patients, (19 women and 7 men) 5 had migraine with aura and 21 without aura. Mean patient age was 31.1±10.2 years. Mean disease duration was 8.1±5.4 years, mean duration of headaches 28.4±18.4 hours, and mean number of episodes before treatment was 9.9±5.2. Mean MIDAS was calculated as 3.2±0.8, and VAS as 9.0±1.0. Mean number of attacks at six months after treatment was 2.0±3.0, MIDAS was 1.4±0.9 and VAS was 3.5 ± 3.6. When the parameters before treatment were compared with those after treatment there was statistical significance (t = 9.2 %95 confidence interval 6.1-9.6, $p = 0.0001$; t = 8.4 %95 confidence interval 1.3-2.2, $p = 0.0001$; t = 8.1 %95 confidence interval 4.1-6.9, $p = 0.0001$ respectively) (Figure 1). No side effects were observed.

## Discussion

The pathophysiology of migraine is not fully understood. Both clinical and physiological considerations suggest that this syndrome is intimately linked to the meningeal tissues, the trigeminal ganglion, trigeminal brainstem nuclei and to the descending inhibitory systems[1,7,8]. Trigeminal fibbers innervating cerebral vessels arise from neurons in the trigeminal ganglion that contain substance P and calcitonin gene-related peptide (CGRP), both of which can be released when the

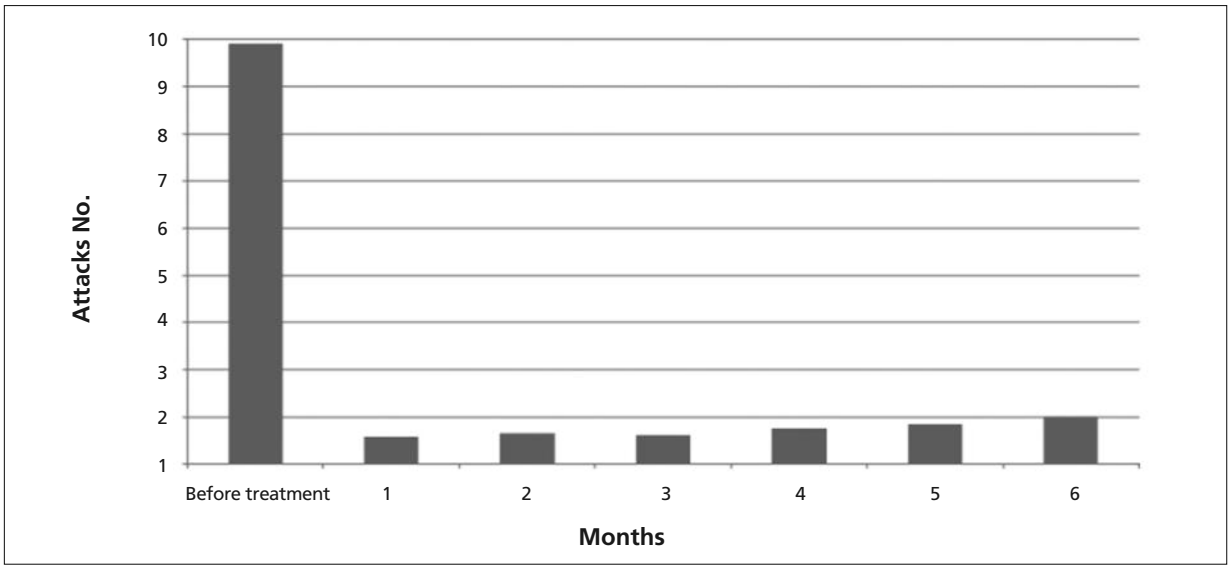


**Figure 1.** Monthly distribution of headache episodes.

trigeminal ganglion is stimulated either in humans or in cats[9,10]. Substance P and neurokinin A cause vasodilation and promote the extravasation of plasma proteins and fluid from nearby meningeal blood vessels. Although CGRP does not promote plasma extravasations, it is a potent vasodilator. Together, these neuropeptides produce in the area around the innervated blood vessels. An inflammatory response called sterile neurogenic perivascular inflammation[11]. Migraine involves dysfunction of brain stem pathways that normally modulate sensory input. The key pathway of pain is the trigemino-vascular input that originates from meningeal vessels and passes to second order neurons in trigemino-cervical complex via trigeminal ganglion and synapses[12,13].

Use of local anaesthetics with the aim of alleviating headaches was initiated in 1940s by injecting lidocaine to Greater Occipital Nerve (GON)[14]. Several studies referred successful results after injecting local anesthetics to GON and trigeminal nerve branches during acute migraine episodes[15,16]. However, there are a limited number of studies investigating long-term effects of such applications on pain. In these reports, GON and supraorbital nerve have been approached together.

Saadah and Taylor[14] in 112 headache patients injected 1% lidocaine and 12 mg of betamethasone into multiple tender points in the vicinity of the occipital nerves. 65% of the patients had lengthened pain alleviation. Caputi and Firetto[17] had in 27 migraine patients; the GON and supraorbital nerves of these patients were approached on alternate days with 5% bupivicaine injections at a maximum of 10 blockades. During the 6-month follow-up they had beneficial effects in 85% of the subjects[17]. Piovesan et al[18] in 37 migraine patients injected GON with 9% physiological saline and 0.5% bupivacaine, with successful results during 2 months of follow-up. Afridi et al[14] treated 101 primary headache patients, whose 54 had migraine. They made a single injection of 2% lidocaine and 80 mg of methylprednisolone to GON obtaining a total response in 9 and partial in 17 patients[14].

Acute phase effects of 2% lidocaine injection with adrenaline to supraorbital nerve has been investigated and reported as effective[15]. Different than the attempts in these studies, we injected the supraorbital and infraorbital branches of the trigeminal nerve with 1% pure lidocaine for 3 times and documented results at 6 months. The data we obtained reveal that supraorbital and infraorbital trigeminal nerve blockade is effective in the treatment of migraine based on long term follow-up.

How can we explain the long-lasting positive effects on pain once membrane stabilizing anaesthetic effect subsides? It is stated that in an attempt to inhibit continuous trigeminal excitability that is present during the headache, supraorbital and infraorbital nerve blockade becomes effective by both blocking the harmful stimulus and blocking the antidromic flow of substance P and CGRP. These substances are the mediators of the axone reflex that provides the basis for perivascular neurogenic inflammation[7,19]. Factors locally strengthening the vasodilatation, extravasations and algogenic stimulation of these peptides are intercepted by the normalization of the threshold response to nociceptor stimulation[17].

Caputi and Firetto[17] reported that there was a continuous mechanical hyperalgesia at the exit points of epicranial nerves in all migraine patients even during the interictal phase and that central hypersensitivity occurred once extracranial perivascular nociceptors were sensitized. This condition is the main trigger in migraine episodes. Elimination of the focus harboring the active nociceptors with local anesthetics alleviates the mechanical hyperalgesia. That is why they recommend the continuation of the injections until nociceptor sensitization decreases and neuronal sensitivity returns to normal. Thus, we performed three repetitive injections in all patients and followed-up the six month prognosis of the patients. There was no significant side effect of this technique. It is easy to use and has low cost. The effect appears in a short interval.

## Conclusions

We believe that this treatment will have an increasing importance among options available to migraine patients in the near future.

**Conflict of Interest**

None declared.

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