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EVALUATION OF THE CHANGES IN THE NASAL CAVITY DURING THE MIGRAINE ATTACK

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Abstract

Objectives—There are some subjective symptoms involving the nasal cavity such as nasal congestion during a migraine attack. In this study, we aimed to evaluate the possible changes occurring in the nasal cavity, during headache in migraine patients.

Material and Methods—Subjects with migraine were studied. The control group was consisted with tension-type headache patients. The severity of the headache and accompanying complaints were assessed by visual analog scale, and nasal mucosa was assessed by anterior rhinoscopy and endoscopy. Resistance of the nasal cavity was evaluated with anterior rhinomanometry. The data obtained during the attack periods and attack free periods were compared.

Results—25 migraine patients and 15 tension-type headache patients were enrolled. It was found that 19 subjects (%76) of migraine group and 5 of tension-type headache group were suffering from nasal congestion during the attack, and that the differences between the groups were statistically significant ($p<0.05$). The average of total nasal resistance in migraine patients was $0,57\pm0,60$ kPa/L/sn during migraine attacks and $0,28\pm0,14$ kPa/L/sn during attack free periods. The average of total nasal resistance in tension-type headache patients was $0,32\pm0,14$ kPa/L/sn

during attack periods and $0,31 \pm 0,20$ kPa/L/sn during attack free periods. In the migraine group, the change of nasal resistance between during the attack and attack free periods was found statistically significant, while there was no statistically significant difference in the tension-type headache group.

Conclusion—According to the results of this study, complaining of nasal obstruction and nasal airway resistance increases during migraine attacks. Cause and effect relationship between nasal obstruction and pain is not clear and clinical trials are needed to determine the effect of nasal obstruction treatment (mucosal decongestion etc.) on the complaint of pain.

Keywords

Migraine; tension-type headache; nasal obstruction; rhinomanometry

Introduction

Migraine is a common primary episodic headache disorder accompanied by neurological, gastrointestinal and autonomic changes in various proportions ¹. In our country it is determined that the incidence of migraine is 21.8% in women and 10.9% is in men ². In recent studies, especially trigeminovascular system has been shown to play an important role on the formation of migraine pain ³. According to the trigeminovascular theory, neurogenic inflammation of the meninges during the migraine attack causes pain by the activation of the trigeminal nerve terminals. The release of neuropeptides in trigeminal nerve ending which provides the sensory stimulation of nasal cavity and sinuses, causes a number of changes resulting in frequent symptoms in nasal mucosa during the migraine attacks such as runny nose, nasal congestion, and a feeling of fullness on face, especially by increasing blood circulation through the effect of vasodilatation in turbinates.

In this study, we aimed to evaluate the possible changes emerged in nasal cavity during the headache attack of migraine objectively by rhinomanometry and to determine the role of the nasal cavity changes in migraine attack.

Material and Methods

This study was conducted between October 2010–April 2011 in Gulhane Training Hospital after the approval by The Ethical Committee of Gulhane Military Academy of Clinical and Laboratory Research.

Patients in follow up with a migraine, according to the year 2004 criteria of International Headache Society ⁴, and having no complaints related to nasal cavity except pain attacks were included in this study. As control group, cases with a diagnosis of tension-type headache were selected. Acute upper respiratory tract infections, deviated septum which causes mucosal contact, sinonasal inflammatory allergic diseases, diseases that can cause nasal congestion such as adenoid hypertrophy, and disorders that can cause chronic headaches such as cerebrovascular disease, trigeminal neuralgia and epilepsy were excluded.

The severity and frequency of the headache and accompanying complaints of the cases were assessed by applying visual analog scale, and nasal mucosa was evaluated by anterior

rhinoscopy and endoscopy. The nasal cavity resistance was measured by active anterior rhinomanometry (MasterScope-Rhino, Cardinal Health GmbH, Germany). All measurements were performed according to the recommendations of the International Standardization Committee for Rhinomanometry were followed⁵. Active anterior rhinomanometry for left and right unilateral nasal flow, total nasal airflow resistance was calculated by measurement program (JLAB Lab Manager Software, version 5.3.0) supplied with rhinomanometry setup. Inspection and measurements were repeated during the attack and attack free periods. The data were compared by using the Mann-Whitney U, Wilcoxon and Chi-Square tests.

Results

Twenty-five cases of migraine patients and 15 cases of tension-type headache patients, totally 40 cases were enrolled in the study. The demographic findings of the groups summarized in table 1. When the cases forming the migraine and tension-type headache groups were compared in terms of pain character; pain intensity, frequency and duration, there was no statistically significant difference ($p=0,61$; $0,25$ and $0,08$ respectively) (Table 2).

When compared in terms of accompanying symptoms, the rate of the symptoms of nausea, vomiting, photophobia and phonophobia in migraine patients during headache was significantly higher than in those with tension-type headache (Table 2).

Anterior rhinoscopy findings during headache were compared. 19 cases of migraine group (76%) and 4 cases of tension-type headache group (27%) had the complaint of nasal congestion during the attack and the difference between the groups was found statistically significant ($p=0,02$) (Table 2). Hyperemia of the nasal mucosa and inferior turbinate hypertrophy was found significantly higher in migraine group ($p=0,01$ and $<0,001$ respectively) (Table 2). 7 cases of migraine group (28%) and 2 cases of tension-type headache group (13%) had the nasal oversecretion which was not statistically significant.

It was found that the average of total nasal resistance during migraine attacks and attack free periods were 0.57 ± 0.60 kPa/L/sn and 0.28 ± 0.14 kPa/L/sn respectively. It was found that the average of total nasal resistance during tension-type headache attacks and attack free periods were 0.32 ± 0.14 kPa/L/sn and 0.31 ± 0.20 kPa/L/sn respectively. During headache attack periods nasal resistance was significantly higher in migraine group than tension-type headache group while there was no statistically significant difference between nasal resistances in both group during attack free periods ($p=0,02$, $p=0,52$). Difference of nasal resistance in each group during headache attack and attack free periods compared. During headache attack periods, nasal resistance was significantly higher in migraine group ($p<0,001$) (Table 3).

Discussion

Migraine is an important health problem affecting a large part of society. As it is frequently seen in the community and directly affects the quality of life; the importance of the effective

and accurate treatment options increases. Migraine treatment will be possible by the clear understanding of the pathogenesis.

The molecular mechanisms and pathogenesis of migraine is not fully explained yet. Migraine headache is the result of the chain of neuronal-vascular events triggered by the endogenous and exogenous factors in people with genetic susceptibility ⁶. Activation of trigeminovascular system constitutes the essence of this chain.

The trigeminovascular inflammation firstly proposed by Moskowitz in 1984, is the most widely accepted theory of the pathogenesis of migraine today ⁷. According to this theory, the peripheral nociceptors at meninges are activated as the result of the release of ions and chemical agents close to the sensory fibers that innervate meninges. Peripheral nociceptors exposed to chemical agents, generate a secondary stimulus causing a feeling of pain ⁸. Trigeminal nerve is the first way for the conveying of pain. The second recipient neuron is in the brain stem. The activation and sensitization of the trigeminal system take the pain sense to the trigeminal ganglion and trigeminal nucleus caudalis ^{9,10}.

According to the axon reflex theory, the activation of peripheral trigeminal axons also causes the release of neuropeptides to the perivascular area contained in peripheral nerve endings. Calcitonin gene related peptide (CGRP), substance P (SP), and neurokinin A emerge from these neuropeptides and make vasodilatation in meningeal vessels ^{11,12}. In addition, when trigeminal ganglion is stimulated, it's shown that platelet aggregation, degranulation of mast cells occurs and then histamine secretion follows ¹³. By the secretion of histamine, rise of local blood flow and vascular permeability is observed, thus it leads to the rapid accumulation of other proteins, including antibodies, to this region. When mast cells are activated, a group of cytokines are secreted. As a result, neurologic inflammation occurs ¹⁴. This vasodilatation and neurogenic inflammation leads to formation of more stimulation of perivascular trigeminal axons and of more pain ³. Also, the presence of high levels of CGRP during attacks indicates the peripheral trigeminal activation. The increase of the level of CGRP by the activation of trigeminovascular system during migraine attack and existence of it in the jugular venous blood have been showed ^{12,15}.

Activation and sensitization of the trigeminal vascular system is responsible for headache and migraine accompanying symptoms. Two changes occur in the neurovascular junction; vasodilation of dural blood vessels and neurogenic inflammatory reaction. By making the first row neurons of the trigeminal nerve sensitive, neurogenic inflammation and enlargement of blood vessels, cause pain in vascular character that increases with non-nociceptive stimuli such as exercise, bending forward, coughing and sneezing that increase the intracranial pressure. Activated neurons in first row of the trigeminal nerve, transmits pain to the second order neurons in the nucleus of trigeminal nerve. By activating the centers responsible for nausea and vomiting such as solitarius tract, activated second row of the brain stem neurons cause vomiting and nausea frequently seen in migraine attacks. The pain spreads to the cortex here. The effect of the trigeminovascular system on the cortex causes major symptoms of migraine such as photophobia, phonophobia or osmophobia ¹⁶. In our study, in migraine patients during attacks, large quantities of vomiting and nausea are seen,

whereas these symptoms are very rarely seen in patients with tension-type headache and a significant difference between the two groups was seen.

Nasal symptoms frequently accompany migraine attack. Sensory stimulation of intranose and sinuses is via branches of the trigeminal nerve. After growing out of Gasserian ganglions, ophthalmic (V1) and maxillary (V2) nerves, responsible for stimulation of the region, form a common branch and a complex network of anastomoses with other sensory, sympathetic and motor nerves ¹⁷. The trigeminovascular system activation that leads to an attack of migraine and release of neuropeptides, seen in the central and peripheral branches of the trigeminal nerve; are also seen inside the nose and sinuses (Axon reflex theory). Neuropeptides such as SP and CGRP, widely released in the mucosa of the nose containing the end of the trigeminal nerve, cause the increase of the blood flow by causing vasodilation and as a consequence leads to congestion and hypersecretion. In addition, as described above, the release of neuropeptides causes mast cell degranulation and the release of other pro-inflammatory substances, and thus causes the formation of neurogenic inflammation. As a result, symptoms such as a feeling of pressure on face, runny nose, nasal congestion, sinus headache, tearing and itching of the nose can be seen in migraine attack ¹⁸. Barbanti and his colleagues reported that at least one of nasal symptoms in 46% of patients was seen during a migraine attack ¹⁹. In our study, possible complaints in the nose that cause subjective symptoms during the migraine attack were evaluated by an objective method, anterior rhinomanometry. In the group consisting of migraine patients, a significant decrease at the air flow values and a significant increase at the nasal resistance values were detected during the attack compared to the non-attack period (Table 3). In contrast, in patients with tension-type headache, airflow and nasal resistance values were not significantly different during episodes of pain and non-attack period. These results show that the nasal changes that cause subjective symptoms in the nose, decrease air flow and increase nasal resistance during the migraine attack. This is because, in accordance with the above-described mechanisms, we suppose that there is congestion occurring in the nasal mucosa as the result of neurogenic inflammation and vasodilatation in the attack of migraine. This congestion manifest itself by nasal obstruction which is one of the most important nasal symptoms of migraine attacks. In our study, we determined that patients with migraine headache had a statistically significant level of nasal congestion complaint and inferior turbinate hypertrophy (Table 2).

Contacting of nasal mucosal surfaces with each other, causes pain ²⁰. The mutual distance between the mucosal surfaces in the nasal cavity is a few millimeters. Congestion may easily cause the contact on the mucosal surfaces. Pain sensation, emerging from the nose and sinuses, comes to the trigeminal ganglion by afferent ways. This sense can increase the severity of migraine pain or trigger migraine by increasing the activation of trigeminovascular system ¹⁷. Surgical applications eliminating the contacts of the nasal cavity mucosa are known to be effective in the treatment of pain ²¹. The results of this study suggest that the elimination of structural problems can lead to pain-reducing effect on the frequency and severity of the pain in patients with migraine, having structural problems that facilitate contact with the nasal mucosa.

Conclusion

Migraine attacks often occur together with nasal symptoms. Whether the symptoms occur due to migraine or them cause migraine is controversial. If mucosal congestion or contact of nasal mucosa is considered to trigger migraine or to exacerbate pain, the reduction in frequency and intensity of migraine pain will be possible by the treatment of pathologies of the nose. Additional clinical studies are needed on this subject.

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Table 1

Demographic findings of groups

		Migraine (n=25)	Tension-type headache (n=15)
Gender	Male	2	4
	Female	23	11
Mean age$\pm$SD(min/max) years		28 $\pm$ 3(22/53)	27 $\pm$ 7(18/53)

Table 2

Comparison of groups for symptoms and findings

	Migraine (n=25)	Tension-type headache (n=15)	P	
Pain	Intensity (VAS)	8	9	0,61
	Frequency (monthly)	4	4	0,25
	Duration (year)	9,5	2	0.08
Nausea	20	3	<0,001	
Vomiting	18	0	<0,001	
Photophobia	20	0	<0,001	
Phonophobia	21	0	<0,001	
Nasal congestion	19	4	0,02	
Nasal over-secretion	7	2	0,19	
Nasal mucosal hyperemia	13	2	0,01	
Inferior turbinate hypertrophy	18	2	<0.001	

Table 3

Comparison of groups for anterior rhinomanometry

	Migraine (n=25)	Tension-type headache (n=15)	P
Total nasal resistance * (mean±SD)	Attack periods	0,57±0,60	0,32±0,14
	Painless	0,28±0,14	0,31±0,20
P	<0.001	0,63	

*
It's unit is kPa/L/sn.