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Efficacy of Diet Restriction on Migraines

Migrende Diyet Kısıtlamasının Etkinliği

Akçay Övünç ÖZÖN[1], Ömer KARADAŞ[2], Aynur ÖZGE[3]

[1]Department of Neurology, Private Liv Hospital, Ankara, Turkey
[2]Department of Neurology, Ankara Mevki Military Hospital, Ankara, Turkey
[3]Department of Neurology, Mersin University School of Medicine, Mersin, Turkey

ABSTRACT

Introduction: Migraine is a common type of headache, but its pathogenesis is still not fully understood. Triggering factors may vary in migraine patients with a particular importance of certain food intake. In this study, the efficacy of limiting certain migraine- triggering foods in the prevention of migraine attacks was investigated.

Methods: Patients diagnosed with migraine without aura according to the International Classification of Headaches were enrolled. Fifty migraine patients stating that migraine attack started after the intake of certain foods were evaluated. The patients were randomly divided into 2 groups. The migraine-triggering foods identified by the patients were excluded from the diet in both groups 1 (n=25) and 2 (n=25). Monthly attack frequency, attack duration, and attack severity (using the visual analogue scale) were recorded before starting the diet restriction and 2 months after the diet restriction. Diet restriction was relaxed in group 1 after the second month and continued in group 2. In the fourth month, the monthly attack frequency, attack duration, and attack severity (using the visual analogue scale) were determined in both groups.

Results: A total of 50 patients comprising 9 males and 41 females were evaluated in this study. In both the groups, in the second month after diet implementation, monthly attack frequency, attack duration, and attack severity were found to have decreased to a statistically significant extent compared to those in the period before diet implementation [group 1 (p=0.011, p=0.041, and p=0.003, respectively) and group 2 (p=0.015, p=0.037, and p=0.003, respectively)]. In the evaluation in the fourth month, it was observed that this significant decrease was maintained only in group 2.

Conclusion: The results of the study reveal that if migraine-triggering foods are identified by migraine patients, restricting their intake can be an effective and reliable method to reduce migraine attacks.

Keywords: Migraine, headache, food, diet, elimination

ÖZ

Amaç: Migren tipi baş ağrısı çok sık karşılaşılan bir baş ağrısı olup hala patogenezi tam olarak anlaşılmış değildir. Migreni tetikleyen faktörler migren hastalarında farklılık göstermekle birlikte, bazı yiyecek alımının kısmi önemi mevcuttur. Bu çalışmada migreni tetikleyici bazı gıdaların alımının kısıtlanmasının, migren ataklarının önlenmesi üzerindeki etkinliği araştırıldı.

Yöntem: Çalışmaya Uluslararası Baş ağrısı sınıflamasına göre aurasız migren tanısı konulan hastalar alındı. Migren atağının belirli gıdaların alımından sonra başladığını belirten 50 migren hastası değerlendirildi. Hastalar randomize olarak 2 gruba ayrıldı. Hem grup 1 (n=25) hem de grup 2 (n=25) hastaları için belirlenen tetikleyici gıdalar o hastanın diyetinden çıkarıldı. Diyet kısıtlamasına başlamadan önce ve diyet uygulama sonrası 2. ayda, bir ay içerisindeki atak sıklığı, atak süresi ve Görsel Analog Skala ile ağrı şiddetleri kaydedildi. İkinci aydan sonra grup 1 hastalarındaki diyet kısıtlaması kaldırıldı ve grup 2 için diyet kısıtlamasına devam edilerek 4. ayda her iki grupta, bir ay içerisindeki atak sıklığı, atak süresi ve Görsel Analog Skala ile ağrı şiddetleri saptandı.

Bulgular: Çalışmada 9'u erkek 41'i kadın olmak üzere toplam 50 hasta değerlendirildi. Her iki grupta hastalara diyet uygulama sonrası 2. ayda, bir ay içerisindeki atak sıklığı, atak süresi ve ağrı şiddetleri, diyet uygulama öncesine göre istatisiksel olarak anlamlı derecede daha düşük olarak saptandı. [grup 1 p değerleri sırasıyla (p=0,011, p=0,041, p=0,003), grup 2 p değerleri sırasıyla (p=0,015, p=0,037, p=0,003)]. Dördüncü ayda yapılan değerlendirmede sadece grup 2'de bu anlamlılığın devam ettiği görüldü.

Sonuç: Çalışma sonuçları migren hastalarında, migren ataklarını tetikleyici gıdaların olduğu tespit edildiğinde, bu gıdaların alımının kısıtlanması, migren ataklarının azaltılmasında etkin ve güvenilir bir tedavi yöntemi olabileceğini ortaya koymaktadır.

Anahtar Kelimeler: Migren, baş ağrısı, gıda, beslenme, eliminasyon

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Correspondence Address/Yazışma Adresi: Akçay Övünç Özön, Özel Liv Hastanesi, Nöroloji Bölümü, İstanbul, Türkiye • E-mail: akcayovunc@yahoo.com
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INTRODUCTION

The pathophysiology of migraine, which is a primary headache disorder, is still not fully understood. Migraine attacks emerge with various features such as autonomic, gastrointestinal, and neurological symptoms. Symptoms such as increasing desire to consume some foods, mental slowdown, sleepiness, difficulty in concentrating, tiredness, and uneasiness occur primarily in the premonitory phase (prodrome) and can also be seen in the post-headache phase. Migraine is a common type of trigeminovascular pain that negatively affects the activities of daily living of individuals and has a high social impact. Migraine appears more often in the first three decades with a female preponderance that is based on hormonal changes (1,2,3,4,5).

Many factors triggering migraine attacks have been identified. The commonly reported ones include environmental factors, menstruation, sleep disorders, changing stress levels, changes in the air, smells, and foods (6,7,8,9). There are non-pharmacological therapy approaches for migraine treatment, such as medical treatment, psychotherapy, biofeedback, and cognitive behavior treatment. On the other hand, pharmacological treatment is used for acute attacks and as a prophylactic measure. Although simple analgesics, non-steroid anti-inflammatory agents, ergot alkaloids, triptans, and opioids are used to treat acute attacks, propranolol, metoprolol, flunarizine, amitriptyline, valproic acid, topiramate, and melatonin are preferred for the prophylactic treatment. The side-effect profile of the drugs used in the acute attack and prophylactic treatment is quite wide. Apart from their side effects, they interact with drugs in a potentially dangerous way and new treatments are needed (4,5,10,11,12).

It has been discussed for years whether food factors play a triggering role in migraines, and the first few studies on this issue reported that restricting the intake of some foods is effective in preventing migraine attacks and symptoms. Although some foods are known to be triggering in migraine patients, this issue is still being discussed (13,14). The objective of this study was to investigate whether restricting the intake of foods that trigger migraine attacks prevents such attacks.

METHODS

The patients who were diagnosed with migraine without aura according to the International Classification of Headache Disorders 2004 were included in this study (15). Patients, with normal physical and neurological examinations, who were aged between 18 and 60 years, and who had a migraine attack frequency of 4 or more times a month were consecutively selected. Patients with secondary headaches were excluded. In case of patients using prophylactic medication, on admission, the medication was not changed during the study period. According to the International Classification of Headache Disorders 2004, the following patients were excluded from the study: ones with medication overuse headache, who started therapy in addition to their current migraine therapy in 6 months before the beginning period of the study, who underwent the Botulinum Neurotoxin Type A treatment for a medical purpose in 6 months before the beginning period of the study, who underwent non-pharmacologic treatment, who had other primary headache history apart from migraine, who had a malignancy history and had undergone cranial surgery, who were pregnant or in the breast-feeding period, who had uncontrolled hypertension, who had uncontrolled diabetes mellitus, who had suffered chronic liver failure, who had suffered chronic renal failure, who had suffered congestive heart failure, who had hypophyseal and hypothalamic dysfunction, who had consumed 500 mg/day or more caffeine in the last 1 month, and who had a major psychiatric disorder.

According to the inclusion and exclusion criteria of the study, a total of 57 patients comprising 10 males and 47 females among 247 migraine

Table 1. Food sensitivity questionnaire

Veal	**Cabbage**
Lamb	Cauliflower
Turkey meat	Broccoli
Fowl	Artichoke
Chicken	Apple
Sardine	Pear
Salmon	Mandarin
Shrimp	Orange
Oyster	Plum
Anchovy	Banana
Tuna fish	Apricot
Sujuk	Grape
Sausage	Raisin
Salami	Strawberry
Cows'	milk Kiwi
Goat's milk	Melon
Yoghurt	Pine apple
Butter	Mango
Cheese	Avocado
Cream cheese	Date
Parmesan cheese	Pumpkin
Kosher cheese	Coconut
Mozzarella cheese	Huckleberry
Roquefort cheese	Mushroom
Egg	Olive
Honeydew honey	Sesame
Flower honey	Thyme
Thyme honey	Cumin
Wheat	Curry powder
Rice	Clove
Lentil	Saffron
Corn	Starch
Oat	Vanilla
Soy	Ginger
Potato	Carob
Onion	Maple Syrup
Garlic	Sunflower seed
Tomato	Nut
Pepper	Peanut
Cucumber	Almond
Carrot	Walnut
Radish	Chocolate
Lemon	Pickle
Parsley	Tea
Mint	Turkish coffee
Dill	Nescafe
Garden Rocket	Sage tea
Eggplant	Fennel
Zucchini	Fruit juice
Leek	Alcohol
Other foods :	

Table 2. Foods identified to trigger migraine attacks

Type of food	The number of people affected by the food		
	Group 1	Group 2	Comparison of groups, p value*
Wheat	13 (52%)	14 (56%)	0.8125
Orange	10 (40%)	12 (48%)	0.6281
Egg	11 (44%)	9 (36%)	0.6274
Nescafe	9 (36%)	8 (32%)	0.8115
Cheese	9 (36%)	7 (28%)	0.6257
Chocolate	7 (28%)	7 (28%)	0.9920
Milk	7 (28%)	8 (32%)	0.8111
Sujuk	6 (24%)	7 (28%)	0.8104
Sugar (beet)	6 (24%)	6 (24%)	0.9920
Red Meat	6 (24%)	5 (20%)	0.8094
Pickle	6 (24%)	5 (20%)	0.8094
Alcohol	5 (20%)	5 (20%)	0.9220
Mushroom	4 (16%)	5 (20%)	0.8087
Corn	3 (12%)	4 (16%)	0.8076
Tea	3 (12%)	3(12%)	0.9919
Onion	2 (8%)	1 (4%)	0.8045
Garlic	1 (4%)	1 (4%)	0.9917

*Mann–Whitney U test (nonparametric test). If p value is >0.05, the difference between the medians is not significant.

patients evaluated and whose migraine attacks were determined to be related to the intake of certain foods by implementing the food sensitivity questionnaire form (Table 1) in migraine patients were included in the study. Informed consent was obtained from all participants. Examining 1-month headache diaries of all patients before treatment, the monthly attack frequency, attack duration (hourly), and pain severity (using the visual analogue scale) were recorded. The patients were randomly divided into 2 groups. The identified triggering foods were excluded from the diet of the patients in groups 1 (n=29) and 2 (n=28). Because 4 patients in group 1 and 3 patients in group 2 could not maintain the inclusion criteria during the study, the study was completed with 50 patients. Before diet implementation and in the second month after diet implementation, the monthly attack frequency, attack duration (hourly), and attack severity (using the visual analogue scale) were recorded. After the second month, the diet restriction was relaxed in group 1 patients but continued in group 2 patients. In the fourth month, the monthly attack frequency, attack duration (hourly), and attack severity (using the visual analogue scale) were recorded in both groups. All data were obtained by evaluating headache diaries.

Statistical Analysis

Data analysis was performed using the Statistical Packages for the Social Sciences (SPSS Inc; Chicago, IL, USA) for Windows 15.0 package. Descriptive statistics were shown as a mean Å} standard deviation for continuous variables and as case number and percentage for nominal variables. Whether the distribution of continuous variables was near normal was investigated using the Kolmogorov–Smirnov test. In comparison with the pre-treatment, whether statistically significant changes occurred in the second and fourth months after treatment were compared using the Wilcoxon signed-rank test. The differences between groups were examined using the Mann–Whitney U test. Operating power was found to be 80%. Results showing p<0.05 were accepted to be statistically significant.

RESULTS

In this study, 247 migraine patients were evaluated, and it was completed with 50 patients. The foods triggering migraine attacks were identified (Table 2). The average age of the patients was 36.28 years in group 1 and 36.48 years in group 2. There were 21 (84%) female and 4 (16%) male patients in group 1 and 20 (80%) female and 5 (20%) male patients in group 2. There was no statistically significant difference between groups with regard to the average age and distribution of women and men (p=0.969 and p=0.987, respectively).

Although there was a statistically significant decrease in the monthly attack frequency, attack duration, and attack severity in the second month after treatment compared to that during pre-treatment in group 1 (p=0.011, p=0.041, and p=0.003, respectively), there was no statistically significant decrease in the monthly attack frequency, attack duration, and attack severity values in the fourth month after treatment compared to that during pre-treatment in group 1 (p=0.426, p=0.758, and p=0.128, respectively). There was a statistically significant decrease in the monthly attack frequency, attack duration, and attack severity in the second and fourth months after treatment compared to those during pre-treatment in group 2 (p=0.015, p=0.037, and p=0.003 and p=0.007, p=0.022, and p=0.003, respectively) (Table 3).

Comparing group 1 and group 2, the decrease in both groups was identified to be proportionately similar in terms of the monthly attack frequency, attack duration, and attack severity values in the second month. It was statistically insignificant (p=0.806, p=0.829, and p=0.868, respectively). Although the proportional decrease in the attack frequency and attack severity was observed to be statistically significant in group 2 in the fourth month compared to group 1 (p=0.013 and p=0.006, respectively), the proportional decrease in pain duration was statistically insignificant (p=0.138) (Table 3).

Table 3. The evaluation of the changes in clinical measurements before and after treatment in groups and between groups

	Group 1		Group 2		Group 1 vs. Group 2
	Mean±SD	p[a]	Mean±SD	p[b]	p[c]
VAS	13 (52%)	14 (56%)	0.8125		
Beginning	89.80±5.7		90.20±8.5		0.517
2 months later	72.80±20.9	0.003* α	72.20±19.9	0.003* α	0.868
4 months later	86.80±9.6	0.128 β	71.40±20.1	0.003* β	0.006*
Number of attacks	9 (36%)	7 (28%)	0.6257		
Beginning	6.08±1.7		5.96±1.7		0.759
2 months later	4.84±1.9	0.011* α	4.68±1.9	0.015* α	0.806
4 months later	5.96±1.7	0.426 β	4.64±1.8	0.007* β	0.013*
Pain duration	6 (24%)	6 (24%)	0.9920		
Beginning	29.44±21.8		30.56±22.3		0.953
2 months later	22. 2±15.2	0.041* α	23.52±18.1	0.037* α	0.829
4 months later	28.96±20.0	0.758 β	22.88±18.4	0.022* β	0.138

[a]Paired test (Wilcoxon) ffor comparisons in Group 1 between pre-treatment (beginning) and post-treatment (2 and 4 months later), [b]Paired test (Wilcoxon) for comparisons in Group 2 between pre-treatment (beginning) and post-treatment (2 and 4 months later), [c]Unpaired test (Mann-Whitney U test) for comparisons of the changes between Group 1 and 2, VAS: visual analog scale; SD: standard deviation

DISCUSSION

According to the findings of the study, more than one triggering factors related to food were identified for all the patients. This situation was observed in approximately 1/5 patients with migraine. The foods identified as triggering were restricted from the diet of all patients, and in the second month, migraine attacks and symptoms of all patients decreased. Food restrictions applied to group 1 were relaxed in the second month to test the effectiveness of this method. The patients continued their diet as it was before the implementation, and the well-being was observed to continue in this group. On the other hand, food restrictions were continued in group 2. The restrictions were found to be effective in decreasing the attack frequency, attack duration (hour), and attack severity of migraine after treatment at the end of the fourth month compared to that during the pre-treatment and that in group 1.

In this study, the elimination of foods identified as triggering factors in migraine patients gave good results in reducing migraine attacks and symptoms. In recent studies, the importance of food intolerance and food sensitivity in many painful cases, such as primary headaches, especially migraine, were concentrated upon and treatment strategies were developed. Treatment strategies were the methods used to prevent food restriction and other triggering factors in the treatment of migraine and other primary headaches (16).

In the study conducted by Grant (16), the relationship between food allergies and migraine was examined, and the foods causing common reactions were excluded from the normal diet of 60 migraine patients for 5 days. A significant decrease was detected in the monthly number of headaches and full painlessness was observed in 85% of the patients. It was stated that immunological and non-immunological mechanisms could play a role in migraine pathogenesis resulting from food intolerance. Egger et al. (17) reported in their double-blind controlled studies including 88 children that there was a serious decrease in migraine attacks using an oligoantigenic diet.

In their prospective studies, including 61 episodic and chronic migraine patients and completed with 39 patients, Rees et al. (18) reported what the decrease in migraine attacks was, with a 6-point scale (0=no benefit, 5=high benefit), when they excluded the foods with IgG increases by monitoring serum food-specific IgG levels of the patients within a 2-month period. At the end of the second month, 38.2% of the patients indicated a serious benefit (4–5 points) in terms of migraine symptoms, 32.4% of the patients saw few to no benefit (0–1 points). When they started their normal diet again, a serious increase in strong migraine attacks was observed in 26.9% of the patients.

In their randomized double-blind studies, including 35 patients diagnosed with migraine without aura, Alpay et al. (19) studied the IgG levels developed against 266 food antigens after 6-weeks of normal diet using the ELISA method, and reported a decrease in the number of painful days, attack number, and rate of drug use (30% decrease in the number of attacks, 50% decrease in the number of painful days) after 6-weeks since they had excluded foods with high IgG levels from the diet; but, they reported no change in attack severity and attack duration. After a 2-week normal diet, the patients were given a provocative diet generating high IgG levels, and all the parameters of the patients during the provocative diet were at similar levels with the base values. Thirty patients completed the study. It was stated that diet restrictions based on IgG antibodies could be used as an effective strategy to minimalize migraine attacks (especially for the attacks resistant to therapy) (19).

Mitchell et al. (20) divided 167 patients with migraine and intolerance to one or more foods into 2 groups; they gave a normal diet to one group and gave a diet to another group created by excluding the intolerant foods as observed with the ELISA method. While a significant decrease was detected in the number of migraine pains in the fourth week, no significant decrease (as in the fourth week) was observed in the 12th week. In these studies, the elimination of the foods identified as triggering factors in migraine was used as a treatment method, and it was observed that variable results related to its effectiveness were obtained (20).

In this study, a wide food intolerance evaluation was performed with the food questionnaire form including foods triggering migraine attacks and other triggering foods apart from them, without using the ELISA method. The antibodies for a specific number of foods were determined with ELISA methods. The food questionnaire form enables evaluation of a larger number of foods. The treatment efficacy obtained with diet

restriction applied to the patients as evaluated with a food questionnaire form was similar to the treatment efficacy obtained from food restriction with food intolerance identified using high-cost methods such as ELISA.

As a result, some foods can both show individual differences and trigger migraine attacks. When personal food triggers are determined, restricting the intake of these foods contributes to the prevention and minimization of migraine attacks. As detected in group 2 in this study, an effective response obtained with continuing the diet implementation is possible over the long-term. Results show that this method can be used as an additional strategy in migraine prevention treatment and decreasing symptoms. Randomized, double-blind, placebo-controlled, and long-followed studies that include a large number of patients are necessary to accurately reveal the efficacy and power of this strategy in migraine treatment.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Gülhane Military Medical Academy.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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REFERENCES

1. Bussone G. Pathophysiology of migraine. Neurol Sci 2004; 25(Suppl 3):S239-41.
2. Ferrari MD. Migraine. Lancet 1998; 351:1043-1051. [CrossRef]
3. Martin VT, Behbehani M. Ovarian hormones and migraine headache: Understanding mechanisms and pathogenesis-part 1. Headache 2006; 46:3-23. [CrossRef]
4. Silberstein SD, Sapel JR, Freitag FG. Migraine: diagnosis and treatment. Wolff's headache. In: Silberstein SD, Lipton RB, Dalessio DJ, eds. 7thed. Oxford university pres. 2001. p. 201-203.
5. Karadaş ., Odabaşı Z. An Open-Label Clinical Study on the Efficacy of Melatonin Prophylaxis in Migraine: A Preliminary Report. Arch Neuropsychiatry 2012; 49:44-47.
6. Kelman L. The triggers or precipitants of the acute migraine attack. Cephalalgia 2007; 27:394-402. [CrossRef]
7. Martin PR, Reece J, Forsyth M. Noise as a trigger for headaches: relationship between exposure and sensitivity. Headache 2006; 46:962-972. [CrossRef]
8. Goadsby PJ. Recent advances in the diagnosis and management of migraine. BMJ 2006; 332:25-29. [CrossRef]
9. Vaughan TR. The role of food in the pathogenesis of migraine headache. Clin Rev Allergy 1994; 12:167-180.
10. Loder E, Biondi D. General principles of migraine management: the changing role of prevention. Headache 2005; 45(Suppl 1):S33-47.
11. Karadaş ., Inan LE, Ulaş ., Odabaşi Z. Efficacy of local lidocaine application on anxiety and depression and its curative effect on patients with chronic tension-type headache. Eur Neurol 2013; 70:95-101. [CrossRef]
12. Inan LE, Inan N, Karadaş ., Gül HL, Erdemoğlu AK, Türkel Y, Akyol A. Greater occipital nerve blockade for the treatment of chronic migraine: a randomized, multicenter, double-blind, and placebo-controlled study. Acta Neurol Scand 2015; 132:270-277. [CrossRef]
13. Van den Bergh V, Amery WK, Waelkens J. Trigger factors in migraine: a study conducted by the Belgian Migraine Society. Headache 1987; 27:191-196. [CrossRef]
14. Downing D, Davies S: "Allergy: Conventional and alternative concepts". A critique of the Royal College of Physicians of London's report. Journal of Nutritional Medicine 1992; 3:331-349. [CrossRef]
15. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia 2004; 24(Suppl 1):1-160.
16. Grant EC. Food allergies and migraine. Lancet 1979; 1:966-9. [CrossRef]
17. Egger J, Carter CM, Wilson J, Turner MW, Soothill JF. Is migraine food allergy? A double-blind controlled trial of oligoantigenic diet treatment. Lancet 1983; 2:865-869. [CrossRef]
18. Rees T, Watson D, Lipscombe S, Speight H, Cousins P, Hardman G, Dowson AJ. A Prospective Audit of Food Intolerance among Migraine Patients in Primary Care Clinical Practice. Headache Care 2005; 2:105-110.
19. Alpay K, Ertas M, Orhan EK, Ustay DK, Lieners C, Baykan B. Diet restriction in migraine, based on IgG against foods: a clinical double-blind, randomised, cross-over trial. Cephalalgia 2010; 30:829-837. [CrossRef]
20. Mitchell N, Hewitt CE, Jayakody S, Islam M, Adamson J, Watt I, Torgerson DJ. Randomised controlled trial of food elimination diet based on IgG antibodies for the prevention of migraine like headaches. Nutr J 2011; 10:85. [CrossRef]