Open Access Macedonian Journal of Medical Sciences. 2014 Sep 15; 2(3):494-498.
http://dx.doi.org/10.3889/oamjms.2014.089
Brief Communication

Specific Triggers of Migraine Headache in Adolescents

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Citation: Knezevic-Pogancev M, Jovic N, Stojadinovic A. Specific Triggers of Migraine Headache in Adolescents. OA Maced J Med Sci. 2014 Sep 15; 2(3):494-498. http://dx.doi.org/10.3889/oamjms.2014.089

Key words: migraine; adolescents; headache; trigger factors; therapy.

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Received: 29-Apr-2014; **Revised:** 09-Aug-2014; **Accepted:** 10-Aug-2014; **Online first:** 03-Sep-2014

Competing Interests: The authors have declared that no competing interests exist.

Abstract

AIM: Purpose of the study was to recognize specific migraine triggers in adolescents.

MATERIAL AND METHODS: Study was conducted on 20,917 adolescents in Serbia.

RESULTS: Lack of sleep, passive tobacco smoking, alcohol intakes, and "not eating in time" are triggers that provoke migraine in adolescents.

CONCLUSION: Avoiding migraine triggers in 68% of adolescents reduced drug therapy for 75%.

Introduction

Adolescent's migraine, as a very complex syndrome, is still a matter of deduction. It seems to be a complex disorder caused by influence of multiple genes and environmental factors [1]. Exact cause of migraine is unknown. There are no laboratory based diagnostic tests to identify acute migraine attack, as well as no tests to recognize migraine headache sufferers [2]. There is not enough knowledge of age dependent specific signs and symptoms, to clearly distinguish all clinical entities of adolescent migraine syndrome. It is largely underestimated and misdiagnosed, because of the lack of anatomical changes, specific biological markers and specific research tools or brain imaging techniques. Migraine phenomena: sensory, vegetative and affective, found only in humans, with marked age dependent quantitative and qualitative variations which differ from adolescent to adolescent, are outlining the adolescent migraine. In the opinion of a great number of authors today, adolescent migraine, as a very complex syndrome, is still a matter of deduction. According to Barlow's definition, migraine reflects an inherited vulnerability and vasomotor instability [3]. Family and twin studies show that there is a genetic component to migraine, but no genes predisposing to common forms of the disorder have been identified, and the actual types of responsible genes are still not fully understood [4]. No empirical study has explicitly examined how genetic and environmental factors influence the adolescent's migraine headache [5, 6].

Various exogenous triggering factors, alone or in combination with other exogenous or endogenous factors, have high tendency to provoke migraine headache in people who are prone for migraine headache. Trigger factors are exclusive occurrences, which decompensate the nervous system. They do not cause headaches. Certain triggers do not induce headaches in everyone. A specific trigger may not cause head pain each time even in the apparent migraine sufferer. The current studies have identified over 300 different migraine triggers but the

mechanisms by which they produce migraine attacks are mostly unknown [7, 8]. In descending order of frequency they were cited as: sensorial stimuli (75%), sleep deprivation (49%), hunger (48%), environmental factors (47%), food (46%), menses (39%), fatigue (35%), alcohol (28%), sleep excess (27%), caffeine (22%), physical exertion (20%), head trauma (20%), trips (4%), sexual activity (3%), medications (2%), neck movements (2%), smoking (1%) and the use of a low pillow (1%) [9]. Annequin D. found that migraine headache episodes in children are frequently triggered by several factors: emotional stress (school pressure, vexation, excitement, upset), hypoglycemia, lack or excess of sleep (weekend migraine), sensorial stimulation (loud noise, bright light, strong odor, warmth or cold), sympathetic stimulation (sport, physical exercise) [10]. Spierings et al., reported stress/tension, not eating in time, fatigue, and lack of sleep as the most common triggers of migraine headache [11]. The weather, smell, and smoke have been reported as factors that distinguished migraine from the tension-type headache [12].

Since the time of Hippocrates and Areteus of Cappadocia, there have been numerous anecdotes about foods that can trigger headaches in susceptible individuals. There is an amazing similarity between their observations and current knowledge of dietary triggers of migraine headaches [13]. Despite a series of experimental studies demonstrating that food and odors cause headache their role remains unclear. The importance of chocolate has been doubted seriously, and scientific evidence for cheese as a precipitating factor is lacking. In spite of series of experimental studies, it is suggested that subjective sensitivity to certain foods should be examined critically, and proven trigger factors should be avoided. General dietary restrictions have not been proven to be useful.

Nowadays, as the most common trigger factors of adolescent migraine are reported: certain dietary factors, kinetosis, emotional stress, sleep deprivation, physical activity and exertion, mild hypoglycemia due to skipping meals, excessive sun exposure, high external temperature, starvation, febrile illness, different medications and various chemical substances, photo-stimulation, noise, intensive odors, smoke, alcohol abuse etc. Although sleep problems are a common complaint in migraine patients, the role of sleep habits and hygiene, as triggers of head pain, have been poorly analyzed. Only a small number of them is well studied and adequately documented in adolescents [14]. Karl N. et al., suggested that similar trigger factors may trigger similar mechanisms and may cause common pre-headache signs and symptoms in all headache types [15].

Trigger factors are important for two main reasons: firstly, they may provide some clues to the pathogenesis of migraine; secondly, by avoiding them, migraine headache drug therapy may be obviated [16].

Material and Methods

This research was carried out in North Serbian Province, Vojvodina, with total population of 2,031,992. Study was carried out from 1988 to 2008. Participants were selected by multi-study random sampling procedure in 9 cities. Total of 20,917 adolescents, aged 10-18 have been included. Adolescents were selected according to their month and year of birth, and the first 3 letters of their first name – applying a multistage, stratified, clustered sampling procedure. This ensured that adolescent could not enter the study twice during the long research period.

The semi structured screening questionnaire, developed for this study by the author, and was designed according to the International Headache Society criteria (IHS). The questionnaire was developed in 3 phases. First, semi structured interviews with pediatricians and researchers were organized to select relevant domains. The domains for the section about headaches were selected based on the International Classification of Headache Disorders – 2nd criteria [17]. More than 150 possible items were identified. Precise, comprehensive and appropriate items were included in the first form. The possible responses were open-ended options or categorical judgments. In the second phase, the questionnaire was pretested in the semi-structured interviews with a small group of adolescents who either did or did not suffer from headaches (16 families were included). This phase aimed to evaluate the questionnaire's interface and validate the content. Additionally, the sensitivity was evaluated by correlating the data from the questionnaire and the medical records of the adolescents who had headaches. This phase resulted in a revised version, which was cross-evaluated only on healthy adolescents. Fifty adolescents have completed the questionnaire twice in 3 weeks. The nonresponse rate, response distributions, graphical response presentation (response inconsistency) and questionnaire burdens (time to complete, formatting, etc.) were analyzed. A number of items were modified or eliminated and the final form included 93 items which required 20 min to complete.

The inclusion criteria were: age 10–18, attending school, and informed consent signed by both, parents and adolescents. The exclusion criterion was a prior diagnosis of a disease that has a headache as a symptom. The World Health Organization (WHO) defines adolescence as the period of life with psychological and social transition between childhood and adulthood, i.e. from the age of 10 to the age of 19. We included ages from 10 to 18 according to the Health Care rules in Serbia. The mean age of the participants was 15 years and 2.5 months (range 10–18 years, SD 3.02).

The study was approved by Ethical Committees of the Institute and the University of Novi Sad. All patients and their parents have signed the

informed consent before entering the study.

The study was conducted in 3 phases. The first phase of the study was completing a questionnaire to narrow down the number of patients to those who had at least two headaches per month over the past year. The second phase was a face-to-face interview, as well as physical and neurological examination. After diagnosing migraine, adolescents with migraine were asked to keep a headache diary over the period of 6 months. In addition, they were asked to strictly avoid potential triggers, and continue keeping the headache dairy. The third phase was re-interviewing them one year after they started the headache and medication diary.

Due to the possible duplicate interviews (1.5%) and due to the incomplete questionnaires - 5.5% of findings have been biased. Questionnaires completely answered by adolescents who had RH were analyzed separately. Comparing characteristics of individual headaches in adolescents with migraine and non-migraine headaches, specific age dependent features of migraine headaches were established.

The demographics, clinical and social characteristics were described by age and sex according to headache presence and type. The Hi2 test, Levin test and ANOVA were used as statistical methods. A significance level of 5% was used ($p<0.05$). All statistical analyses were performed with SPSS 15.0 (SPSS Inc., Chicago, Ill., USA).

Results

Adolescents filled out the questionnaire (20,917), 4,376 (20.9 %) of them reported recurrent headaches, and 2,008 (9.4%) of them reported migraine (1020 girls and 988 boys).

All of the trigger factors were age dependent (Table 1). Trigger factors sensitivity in adolescent's migraine was stable during the adolescent age for "lack of sleep, odors, and weather changes". It increased for "different food and psychic stress", and decreased for "day activity disturbance, passive tobacco smoke, and food deprivation" (Table 1).

Lack of sleep as a direct headache trigger was equally reported by adolescents with migraine (90.6%) and those with non-migraine headaches (94.5%). Particular food was indicated as a trigger in 72.4% of migraine as well as in 32% of non-migraine headaches ($p<0.05$). The most common dietary triggers implicated in migraine attacks were: meat (32.9%), eggs (30.5%) and aged cheeses (27.7%). Odours, as triggers were reported in 80.9% of migraine and in 10% of the non-migraine headaches ($p < 0.05$). Usual daily routine disturbance as a trigger of headache was reported in 50.0% of migraine and in 75.2% of non-migraine headaches ($p < 0.05$). Physical activity proved to trigger non-migraine (85.3%) more often than migraine headaches (45.3%) ($p < 0.05$). Tobacco smoking triggered 65% of migraine and 75% of non-migraine headache. Passive tobacco smoking triggered migraine more often than non-migraine headaches (81.6% vs. 23.9%). The same results were obtained for alcohol consumption as a trigger (80.2% vs. 68%) ($p < 0.05$). Psychical stress was equally reported as a trigger of headaches by adolescents with non-migraine (99.6%) and by adolescents with migraine headaches (96.9%). Sixty-five per cent of adolescents with migraine reported that "not eaten in time" was the trigger for migraine attacks, whereas 32% of non-migraine sufferers reported the same triggers for non-migraine headache. Non-migraine headaches were, in general, more susceptible to weather changes than migraine headaches (78.8% vs. 21.28%) ($p < 0.05$).

Using Canonical Discriminate Analysis (coefficient over 0.3) migraine headaches were clearly distinguished from non-migraine headaches according to the headache triggers. The canonical discriminate coefficient was 0.77 for lack of sleep, 0.68 for passive tobacco smoking, 0.43 for odours, 0.58 for fasting, 0.45 for particular food, and borderline 0.336 for disturbance of daily routine activity (Table 2). Trigger factors were dependent even in the adolescent age. They appeared as kinetosis, daily activity disturbance, food deprivation, and odors in younger adolescents (10-11 years old) and as odors, food type, and psychological stress in older adolescents (16-17 years old) without significant difference (Table 3).

Table 1: Migraine headache age dependent trigger factors.

Trigger factor	age groups (years)	10-11		12-13		14-15		16-17		Sum total	
	children with migraine	n	%	n	%	n	%	n	%	n	%
Lack of sleep		188	100	294	98.99	390	98.73	458	97.65	1330	98.6
Kinetosis		167	88.83	289	97.31	382	96.71	373	79.53	1211	89.8
Different food		145	77.13	252	84.85	380	96.20	425	90.62	1202	89.1
Psychic stress		71	37.77	239	80.47	393	99.49	469	100	1172	86.9
Odors		126	67.02	242	81.48	346	87.59	415	88.49	1119	82.9
Day activity disturbance		188	100	297	100	377	95.44	239	50.96	1101	81.62
Physical activity		164	87.23	246	82.83	322	81.52	273	58.21	1005	74.4
Passive tobacco smoke		126	67.02	194	65.32	201	50.89	178	37.95	699	51.8
Food deprivation		122	64.89	176	59.26	104	26.33	25	5.33	437	32.3
Weather changes		64	43.04	83	27.95	87	22.02	53	11.30	287	21.28

Migraine: non-migraine headache; Lack of sleep – Pearson Chi Square: V 59610, DF 1, p<0.0001; Kinetosis –- Fisher Exact Test: V 735.350, DF 1, p<0.0001 ; Different food - Linear-by-Linear Association: V 1203.032, p<0.0001; Chronic stress - Fisher Exact Test: V 94063. DF 2, p<0.0001; Odor - Linear-by–Linear Association: V 1889.054, DF 1, p<0.0001; Physical activity - Pearson Chi Square: V 69510, DF 1, p<0.0001; Passive tobacco smoke - Fisher Exact Test: V 532.965, DF 1, p<0.0001; Fatigue - Pearson Chi Square: V 55.445, DF 2, p<0.0001; Psychological stress - Likelihood Ratio: V 1935.861, DF 1, p<0.0001; Other - Pearson Chi Square: 2827.431, DF 1, p<0.0001

Table 2: Canonical discriminating analysis of migraine headache//non-migraine headache trigger factors in adolescents.

Migraine trigger factors	Triggers reported in questionnaire %	Canonical discriminative analysis coefficient Migraine//non-migraine headaches
Psychical stress	96,9	-0,56
Lack of sleep	90,6	0,77
Tobacco passive smoke	81,6	0,68
Alcohol intake	80,2	0,63
Fasting	75	0,58
Odours	80,9	0,43
Tobacco smoke	72,3	0,43
Weather changes	78,8	-0,56
Specific type of food	72,4	0,45
Daily activity disturbance	50	0,336
Oversleeping	45,3	-0,38

We discussed only triggers that could be avoided (sleep disturbance, food, odors, tobacco smoke, alcoholic beverages). Six months later adolescents with migraine reported having 2.8 headaches per month, and having used 5.7 medications per month. Six months later, after having strictly avoiding potential triggers, adolescents have reported having 1.6 headaches per month and using 2.4 medications per month.

Table 3: Age dependent canonical discriminating analysis of migraine headache//non-migraine headache trigger factors.

Migraine trigger factors	Age groups (years)				
Migraine//non-migraine headaches	10-11	12-13	14-15	16-17	SCORE
Daily activity disturbance	0.77	0.74	0.73	0.68	0.772
Lack of sleep	0.64	0.51	0.53	0.50	0.541
Odors	0.35	0.43	0.45	0.45	0.428
Foods	0.36	0.42	0.41	0.40	0.406
Fatigue	0.35	0.32	0.28	0.31	0.236
Psychological stress	0.22	0.18	0.28	0.36	0.216
Travel sickness (kinetosis)	0.36	0.43	0.10	0.09	0.159
Weather changes	0.18	0.16	0.16	0.19	0.152
Tobacco smoke	0.32	0.01	-0.10	-0.08	-0.081
Acute stress	-0.02	-0.02	-0.02	-0.15	-0.058
Food deprivation	-0.12	-0.04	0.03	0.02	-0.057
Oversleeping	-0.00	-0.01	-0.02	-0.05	-0.020

Discussion

Our study results are very similar to European Population Studies. Wober Bingol's [18] Population Study encountered prevalence of 3-17.6 % of migraine in adolescents. Describing pediatric headache, as a common health problem, Hershey found migraine in 10.6% of children between the age of 5 and 15 and in up to 28% of adolescents between the ages of 15 and 19 [19].

Clear triggers as direct causes of migraine headache were occasionally proved in up to 10.4% of adolescents. They were highly significant for occurrence of migraine without aura. Chabriat H. et al., suggested that similar triggers could precipitate headache of different type [20].

The age of children was of importance when identifying foods as direct triggers of migraine headaches. Some foods were indicated as direct triggers of migraine attacks (mean age 11 years and 8.9 months), whereas adolescents (mean age 10 years and 9 months), did not indicate food as a direct trigger of migraine attacks. They reported various odors as trigger factors of migraine attacks. This was in favor of slight, but still existing, higher susceptibility of younger children to odors as trigger factors of migraine attacks, and vice versa in regard to foods.

Fatigue, mental stress, and lack of sleep are the main migraine headache triggers in most reports. There were three different combinations of sleep involvement, possibly due to more than one pathophysiological mechanism. Thirty percent of patients had migraine attack triggered by sleep; 24% by sleep deprivation and 6% by excess sleep. Sleep was also associated with the relief of migraine attacks. Oversleeping could not have been considered a trigger of headaches generally speaking. Although sleep problems were not a common complaint in migraine patients, the role of sleep habits and hygiene, as triggers of head pain, have been poorly analyzed. Bruni evaluated the effect of modifying bad sleeping habits across several headache parameters as very successful [21].

Apparently, there are trigger factors differentiating migraine from non-migraine headache but not vice versa. Identification of migraine trigger factors is extremely important in adolescents. Their elimination directly provides prevention of migraine headaches in adolescents. Typical migraine triggers in adolescents are lack of sleep, tobacco passive smoking, alcohol intakes, and "not eating at time". By avoiding these triggers, in 68% of adolescents 75% reduction of drugs used in headache therapy can be achieved.

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