

“What Triggers You?” A Study of Migraine Triggers in a Tertiary Care Centre

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Abstract: Migraine is a primary headache that may be triggered by exposure to a potential ‘trigger’. **METHODS:** Migraine triggers in 111 patients with migraine who attended the headache clinic at the Madras Institute of Neurology were documented by a questionnaire approach. The questionnaire included 22 sets of migraine triggers. **RESULTS:** Stress (45%) was identified to be the most common trigger followed by weather changes (20.7%) and cold drinks (15.3%) as possible triggers. 10.8% were unaware of any possible trigger and most were not aware of other triggers. All patients were educated about all possible triggers and were advised to maintain a headache diary. **CONCLUSION:** It is important to identify these triggers and avoidance of these triggers reduces the frequency of attacks. Management of stress will help in reducing the morbidity of headaches.

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I. Introduction

Migraine may be best described as a disorder of pain modulation. This may include mechanisms that involve the Ventrolateral periaqueductal grey, Rostral ventromedial medulla, Locus coeruleus and Superior salivatory nucleus in the brainstem, as well as nociceptive traffic through higher centres, such as the hypothalamus, thalamus and cortex¹

Several theories have been put forward regarding the pathophysiology including Wolff's vascular theory, Cortical spreading depression, a role of platelet aggregation (peripheral serotonin), the opening of cerebral anastomoses, cerebral hypoxia, defective anti-nociceptive systems, and neurovascular abnormalities which involve central serotonin²

Migraine appears to be an interaction between environmental factors (triggers) and genetic susceptibility³. Cortical spreading depression (CSD) can be provoked by chemical, electrical, and mechanical stimuli. It also can occur in the setting of energy failure. CSD can be a migraine trigger

As the wave of depolarization moves across the cerebral cortex, NO, arachidonic acid, protons (H⁺), and potassium (K⁺) are released extracellularly. Meningeal nociceptors are activated. Mast cells are activated and degranulate. The trigeminovascular reflex is activated. Trigeminal neurons supplying the dural vessels release calcitonin gene-related peptide (CGRP), substance P, and neurokinin A. The vessels dilate and become inflamed, and plasma protein extravasation occurs (also known as sterile neurogenic inflammation).

The primary event that causes activation of the trigeminovascular system is the firing of first-order peripheral trigeminal neurons in response to nociceptive signals from the meninges; the firing of these neurons produces pain, which is then referred to the head. Continuous activation of these meningeal nociceptors may sequentially activate first-, second- and third-order trigeminovascular neurons, which in turn activate the many regions of the brainstem and fore-brain, resulting in migrainous symptoms⁴

Triggers of migraine, such as sleep and food deprivation, are homeostatic processes that are regulated by ‘on’-‘off’ cell firing in the ventrolateral periaqueductal grey and rostral ventromedial medulla⁵

The Superior salivatory nucleus converges bidirectional inputs from the trigeminovascular system and ventrolateral periaqueductal grey, resulting in vasodilation, the release of inflammatory mediators and activation of meningeal nociceptors⁶.

II. Material And Methods

STUDY POPULATION: 111 migraine patients who attended the headache clinic for more than 6 months. The study included migraine patients with or without aura. **STUDY CENTRE:** The head ache clinic at the Madras Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai -3. **SAMPLE SIZE:** 111 (n=111). **STUDY METHOD:** Questionnaire approach including 22 subset of options (triggers). More than one option were allowed to be selected as migraineurs may have more than one trigger. The results were tabulated and analyzed. **FOLLOW UP:** The patients were followed up on a monthly basis and were educated about the role of stress reduction, avoidance of triggers and the need to maintain a headache diary.

The questionnaire included the following list of triggers

Aged cheese/chocolates/dried fruits and nuts Lights Alcohol and spirits Menstruation Artificial sweeteners and food additives Medications Caffeine Noise Citrus fruits / onions Odors Cured meats Sleep excess or lack Dehydration Skipped meals Depression Stress Exercise (too much) Watching TV or movies Eye strain Weather changes Fatigue (extreme) Others

III. Results

Among the triggers the most common ones were stress (45%) followed by weather changes (20.7%). 17 patients have mentioned soft drinks (15.4%) as one of their triggers. Other noteworthy triggers were lack of or excess sleep (12.6%), menstruation (10.81%), noise (10.81%) and watching tv (9.01%). Exercise and fatigue when summed up accounted for 16.22% whereas flickering lights, eye strain and watching tv were triggers in 24.32% of the migraine population.

Our study results were consistent with similar studies done by Spierings et al⁷ and Lawrence Robbins et al⁸. The factors indicated most frequently as precipitating headache by the patients with migraine were stress/tension, not eating on time, fatigue, and lack of sleep in the study by Spierings et al. The leading trigger factors for migraine were stress, weather changes, premenstrual, and sunlight in the latter study by Lawrence et al.

IV. Discussion

Stress has been reported as the most common trigger of migraine. Psychological stress contributes to the prolongation and exacerbation of an existing pathophysiological predisposition for migraine attacks. Stress might influence the occurrence of a migraine attack is through its effects on mast cells in the Dura. It may be a factor in the precipitation of new onset migraine, it can act as a trigger for individual migraine attacks, and it may play a role in the progression of migraine to a chronic migraine syndrome.

Geomagnetic activity may play a role in weather-related migraine, as may ionic and serotonergic changes in the bloodstream. Platelet serotonin has been experimentally decreased by the inhalation of negative ions.⁹ Soft or cold drinks may trigger migraine due to artificial agents or caffeine which are known migraine triggers. Estrogen stimulates NO synthase activity and these levels fluctuate with the menstrual cycle. Changes in light intensity may exacerbate the processing of dural nociceptive inputs to the thalamus and TCC.

US Headache Consortium established that there is “Grade A” evidence for the use of relaxation training, thermal biofeedback combined with relaxation, electromyographic feedback, and cognitive behavioral therapy as preventative therapy for headaches. Also, “Grade B” evidence was found for combining pharmacotherapy and behavioral therapy as preventative treatment for headache¹⁰

V. Figures and Tables

The questionnaire included the following list of triggers

Aged cheese/chocolates/dried fruits and nuts	Lights
Alcohol and spirits	Menstruation
Artificial sweeteners and food additives	Medications
Caffeine	Noise
Citrus fruits / onions	Odors
Cured meats	Sleep excess or lack
Dehydration	Skipped meals
Depression	Stress
Exercise (too much)	Watching TV or movies
Eye strain	Weather changes
Fatigue (extreme)	Others

VI. Conclusion

Stress has been reported as the most common trigger of migraine. Patient education about possible triggers and management of stress may play a vital role in the long term management of migraine.

References

Journal Papers:

- [1]. Simon Akerman*, Philip R. Holland*‡ and Peter J. Goadsby Diencephalic and brainstem mechanisms in migraine *Nature Reviews Neuroscience* 12, 570-584 (October 2011) doi:10.1038/nrn3057
- [2]. Passchier J. A critical note on psychophysiological stress research into migraine patients. *Cephalalgia* 1994; 14:194-8. Oslo. ISSN 0333-1024
- [3]. Thomas N. Ward, MD, FAAN, FAHS Migraine Diagnosis and Pathophysiology *Continuum Lifelong Learning Neurol* 2012;18(4):753–763.
- [4]. Penfield, W. & F., M. Dural headache and innervation of the dura mater. *Arch. Neurol. Psychiatry* 44, 43–75 (1940).
- [5]. Foo, H. & Mason, P. Brainstem modulation of pain during sleep and waking. *Sleep Med. Rev.* 7, 145–154 (2003)
- [6]. Burstein, R. & Jakubowski, M. Unitary hypothesis for multiple triggers of the pain and strain of migraine. *J. Comp. Neurol.* 493, 9–14 (2005).
- [7]. Egilius L.H. Spierings, MD, PhD; Anniek H. Ranke, BSc; Peter C. Honkoop, MSc Precipitating and Aggravating Factors of Migraine Versus Tension-type Headache *Headache* 2001;41:554-558)
- [8]. Silberstein S. Practice parameter: Evidence-based guideline for migraine headache (an evidence-based review). *Neurology.* 2000;55:754-763

Books:

- [9]. Lawrence Robbins, M.D Precipitating Factors in Migraine: A Retrospective Review of 494 Patients . (*Headache* 1994; 34: 214-216)

Chapters in Books:

- [10]. Anthony M. The effect of negative ions on platelet serotonin in normal and migrainous subjects. In: F. C. Rose, ed. *Advances in Migraine Research and Therapy*, Raven Press, New York, 1992; pp. 139-143.

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