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Evaluation of a possible relation between dentinal hypersensitivity and migraine: A clinical interventional study

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Abstract---Objectives: this study aims to find out any possible role of dentine hypersensitivity as a triggering factor for migraine in susceptible individuals. Methods: This prospective clinical interventional study was done by enrolling 160 patients who were diagnosed as having migraine according to guidelines of 'International Headache Society 'Criteria for Migraine, who were followed for a period of 3 months and those 84 people who showed definite symptoms of

hypersensitivity were further investigated for next 3 months by dividing them into Group A (Group who had dentine hypersensitivity and migraine and received relevant dental treatment to stop dentine hypersensitivity) and Group B (People who had dentine hypersensitivity and migraine but no dental intervention was done to treat dentine hypersensitivity) both these groups were observed for frequency and intensity of migraine attacks for a period of three months. Results: the results showed that apart from priorly identified and documented triggering factors like alcohol, stress, sleep deprivation, fatigue, hormonal changes etc., dentine hypersensitivity has also had significant role and effected considerable number of people to initiate or aggravate the frequency and severity migraine attacks in susceptible individuals, the statistical analysis showed a significant increase in the risk for migraine attacks in the people suffering from dentine hypersensitivity. Conclusion: Dentine hypersensitivity should be further investigated in large samples for its possible role as a triggering factor for migraine attacks, as this study has clearly shown increased incidence of migraine attacks in people having concomitant dentine hypersensitivity.

Keywords---migraine, dentine hypersensitivity, triggering factors.

Introduction

Migraine is a complex neurobiological disorder that has been recognized since antiquity. The core features of migraine are headache, which is usually throbbing and often unilateral, and associated features of nausea, sensitivity to light, sound, and exacerbation with head movement. Migraine has long been regarded as a vascular disorder because of the throbbing nature of the pain. However, vascular changes do not provide sufficient explanation of the pathophysiology of migraine. Up to one-third of patients do not have throbbing pain. Modern imaging has demonstrated that vascular changes are not linked to pain.[1,2,3] Acute migraine attacks occur in the context of an individual's inherent level of vulnerability. The greater the vulnerability or lower the threshold, the more frequent attacks occur. Attacks are initiated when internal or environmental triggers are of sufficient intensity to activate a series of events which culminate in the generation of a migraine headache. Many migraineurs experience vague vegetative or affective symptoms as much as 24 hours prior to the onset of a migraine attack. This phase is called the prodrome and is not the same as aura phase.

The aura phase consists of focal neurological symptoms that persist up to one hour. Symptoms may include visual, sensory, or language disturbance as well as symptoms localizing to the brainstem. Within an hour of resolution of the aura symptoms, the typical migraine headache usually appears with its unilateral throbbing pain and associated nausea, vomiting, photophobia, or phonophobia. Without treatment, the headache may persist for up to 72 hours before ending in a resolution phase often characterized by deep sleep. For up to twenty-four hours after the spontaneous throbbing has resolved, many patients may experience malaise, fatigue, and transient return of the head pain in a similar location for a

few seconds or minutes following coughing, sudden head movement, or valsalva movements. This phase is sometimes called the migraine hangover.[4] A strong familial influence in migraine has long been apparent and this has been demonstrated in twin studies.

The concordance for migraine in monozygotic twins is greater than that for dizygotic twins.[5] Various external and internal stimuli can lead to migraine events in susceptible individuals, which can be considered as migraine triggers or precipitants.[6,7] The reported migraine triggers include stress, sleep, fatigue, fasting, physical exercise, hormonal changes, weather, sunlight, alcohol, and various sensory stimuli. [8–13] Most studies that examined migraine trigger factors were based on participant reports. These trigger factors are found in 73–80% of migraineurs. [14, 15] Many triggering factors were mentioned till date but there was no study which evaluated the dentinal hypersensitivity as a triggering factor for initiation and/ aggravating factor of migraine episodes. In this study we are trying to explore possible relation between dentinal hypersensitivity and migrane using regression analysis.

Material and Methods

Selection of participants and base line evaluation. The participants who met the inclusion criteria were recruited between September 2020 and May 2021. This study was conducted at the neurology outpatient clinics of private hospital based in Hyderabad, Telangana, India. The following inclusion criteria were applied: 1) age between 19 and 55 years and migraines with or without auras, as defined by the International Headache Society Criteria for Migraine (ICHD-3 beta).[16] 2) an episode of 2–14 headache days per month; 3) stable headache characteristics for at least 1 year prior to study entry. The following exclusion criteria were applied: 1) headaches attributed to secondary causes; and 2) inability to complete questionnaires. For the baseline survey, the participants were asked to choose their potential triggers on the basis of their previous experiences from a list of 18 trigger factors. Those factors were selected on the basis of the results of previous studies about migraine trigger factors to which additionally dental hypersensitivity was added as 19th entry, and included stress, excessive sleep, sleep deprivation, exercise, fatigue, hormonal changes, emotional changes, weather changes, sunlight, noise, odors, fasting, overeating, caffeine, smoking, alcohol, cheese/chocolate, traveling. [6,9,10,13,14] and dental hypersensitivity. The participants were also asked to complete the Hospital Anxiety and Depression Scale to determine their anxiety and depression levels [17]. The ethical approval for the study was granted by the SKS neuro hospital review committee, Hyderabad, Telangana, India (Approval number: 2019-28/18B). The participants received an explanation of the study's aims and procedures and provided written informed consent.

Data collection

All the participants were asked to record a voice message and pick up the triggering factors mentioned in the list given to them and post it through whatsapp message to the researcher who was the administrator of the whatsapp group every time they got an episode of migraine, this information was manually

entered into a register on a column created for each individual as and when they reported, these records pertaining to all the 160 individuals were collected after 3months into the study, after care full evaluation, 76 patients who reported no dentinal hypersensitivity were withdrawn from the study as correlatable etiological trigger was not there in these individuals remaining 84 individuals who reported dental hypersensitivity as one of the factors which triggered or aggravated the migraine episodes were retained for further participation in the study and the remaining who failed to correlate migraine with dental hypersensitivity were excluded from further participation.

The participants who were further involved as subjects in the study were divided into two groups Group A 42 patients and Group B 42 patients, both these group patients were thoroughly evaluated in dental chair for possible reasons of dentinal hypersensitivity, 42 patients belonging to Group A were given glass ionomer cement fillings (Gold label GC Corporation Fuji ,Tokyo Japan) and/or Dentine bonding agents(3M ESPE USA, single bond universal bonding agent) as per treatment needs in the areas where there were cervical abrasions, a blast of cold air was blown to test the integrity and to ascertain the completeness of the filling, on the confirmation of the same the patients were discharged and were asked to keep sending the information of number of episodes of migraine and possible triggering factor through voice message, However Group B patients were not given any treatment for dental hypersensitivity and were asked to keep sending voice messages mentioning the triggering factors which they perceived to be responsible for the migraine during each episode.

Data analysis

We analyzed the effect of trigger factor exposure on the headache occurrence using the daily records from the entries made from voice messages of the participants'. The frequency for each trigger factor was acquired by calculating the number of headache days with certain trigger factors divided by the total number of headache days. There were many terms for the occurrence of a headache, such as intensity or probability; we chose the likelihood of a headache. Likelihood of a headache during the presence of each trigger factor was obtained with the following equation:

$$Frequency = \frac{\text{The number of headache days with certain trigger factor}}{Total\ number\ of\ headache\ days}\ X\ 100$$

$$\text{Likelihood} = \frac{\text{The number of headache days with certain trigger factor}}{number\ of\ days\ with\ the\ presence\ of\ same\ trigger\ factor}\ X\ 100$$

Each headache was classified as a migraine or non-migraine headache, according to the diagnostic criteria B–D of item 1.1 of migraine without aura as per the International Classification of Headache Disorders (ICHD)-3 beta. As illustrated below,

Diagnostic criteria

- At least five attacks fulfilling criteria B-D

- Headache attacks lasting 4-72 hr (untreated or unsuccessfully treated)
- Headache has at least two of the following four characteristics:
  - unilateral location
  - pulsating quality
  - moderate or severe pain intensity
  - aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs)
- During headache at least one of the following:
  - nausea and/or vomiting

The categorical variables were presented as percentages, and the continuous variables were summarized using descriptive statistics, such as the means and standard deviations. The clinical variables for the headache were compared according to the presence or absence of trigger factors, using t-tests for continuous variables, and a chi-square test or Fisher's exact test for the frequency variables. The trigger factor frequency was compared between the migraine and non-migraine headaches by using a chi-square test or Fisher's exact test. The associations of the 18 trigger factors and migraine were examined using a stepwise multiple logistic regression analysis with 153 possible combinations of trigger factors. A variable must have had a p value of less than 0.15 to be entered into the regression model. SAS statistical software (SAS version 9.3, SAS Institute, Inc., Cary, NC) was used for all analyses. The statistical significance was set at $p < 0.05$.

Results

Demographic characteristics initially, 160 patients were recruited from the centre. However, 77 patients were withdrawn from the study after they were diagnosed to not having any existing dental hypersensitivity symptoms; therefore, 84 patients finished the study. Of these, we analysed the headache data from 84 patients of these two groups Group A and Group B (S1 File). 38 patients had a migraine without aura, and 4 had a migraine with aura in Group A. In Group B only 1 patient reported migraine with aura whereas 41 reported migraine without aura. The participants' mean age was 38.9 ± 8.6 years of age, with 67% of participants being women. The mean illness duration was 10.4 ± 8.2 years (Table 1). The required recording time per day was 2.1 ± 1.2 (1–5) minutes

Table 1
Demographic data showing study results

parameter	Group A	Group B
Age in years	43.2± 7.2	38.9 ± 8.6
Female	72%	67%
Duration of illness in years	6.8±3.5	8.6±4.2
Pain intensity VAS	8.2±2.4	7.8±3.2
Monthly head ache days	7.4±2.8	9.2±3.0
People who resorted to medication for relief	38%	56%
Expression of satisfaction towards relief given by dental treatment on VAS on a scale	Highly satisfied 34%	Highly satisfied 18%

of 10 (8-10: highly satisfied;5-7: Moderately satisfied; 1-4:Not satisfied)	Moderately satisfied 53% Not satisfied 13%	Moderately satisfied 28% Not satisfied 54%

Mean ± standard deviation; VAS, visual analogue scale;

Of the 7560 days (42patients x 180 days) that included a trigger, 1332 days records (17.6%) were recorded as headache days in Group A and 21.9% in Group B. The following triggers were likely to trigger a headache: 68.5% for alcohol, 41.8% for odor, 58.6% for emotional change, 56.2% for hormonal changes, and 67.7% for stress, 35.1% for sleep deprivation, 58.5% for fatigue and 14.6% for dental hypersensitivity in Group A whereas it was 58.5% for alcohol, 61.3% for odor, 53.4% for emotional change, 36.3% for hormonal changes, and 47.7% for stress, 45.8% for sleep deprivation, 61.1% for fatigue and 22.5% for dental hypersensitivity in Group B.

Discussion

The main findings of the current study were as follows: 1)the frequent trigger factors on headache days were stress, fatigue, and sleep deprivation; the likelihood of a headache was 67.7% for stress, 35.1% for sleep deprivation, 58.5% for fatigue, and 14.6% for dental hypersensitivity: 2)the headaches with trigger factors were more severe relative to those without trigger factors, 3)traveling, hormonal changes, noise, alcohol, overeating ,stress increased the risk of migraines; and 5) hormonal changes and noise increased the risk of migraine regardless of preventive medication, whereas stress, overeating, alcohol, and traveling increased the risk of migraine in situations without preventive medication. Irrespective of remaining triggering factors dental hypersensitivity has significantly increased the intensity and frequency of migraine attacks in Group B when compared to that of Group A.

The study had some limitations. First, the temporal sequence and relationships between the triggers were not evaluated. The changes from the previous levels and associations between the trigger factors may have influenced the headache onset and severity [18]. The differentiation from the premonitory symptoms with functional imaging may be promising[19] Second, we relied on the participants' judgment for recording the triggers and cannot rule out the possibility of selection bias from the clinical setting and recall or confirmation bias by participant [7]. The merits of this study were analyzing dental hypersensitivity as an additional triggering factor apart from those 18 triggers that had already been identified by ICHD, and evaluating the possible role of dental treatment to alleviate this trigger factors thereby decreasing the frequency of headache occurrence, headache features, and the influence dental treatment as a of preventive medication to provide relief to the patients.

Conclusion

This study has paved a path for more intense speculation and analysis of the role of dental hypersensitivity as a potential triggering factor in some susceptible individuals for initiation or aggravation of migraine episodes and possible relief

offered by dental treatment towards providing relief to those migraine episodes triggered by dental hypersensitivity, since the Headaches with trigger factors had greater severity or migraine features. The type of triggers and the presence of preventive treatments may influence headache features, so the investigation of trigger factors is helpful in understanding the pathophysiology of migraines and developing a preemptive strategy for trigger factors.

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