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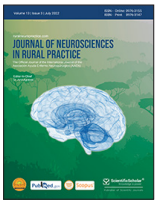

Original Article

Impact of comorbid psychogenic non-epileptic seizures on migraine: An observational study

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ABSTRACT

Objectives: There is a bidirectional and complex interplay between psychiatric comorbidities and migraine. Migraine has been observed in 50–60% of patients with psychogenic non-epileptic seizures (PNES). Studies describe migraine as a medical comorbidity in PNES. However, there are limited studies on impact of PNES on migraine. We aim to see the impact of PNES on migraine.

Materials and Methods: This cross-sectional and observational study was conducted at a tertiary-care center from June 2017 to May 2019. Fifty-two patients with migraine with PNES and 48 patients with migraine without PNES were included on the study. Migraine and PNES were diagnosed based on International Classification of Headache Disorders-3 (ICHD-3) and International League Against Epilepsy (ILAE) criteria, respectively. Headache intensity was assessed using visual analog scale. Comorbid depression, anxiety, and somatoform-symptom-disease were assessed using the Generalized Anxiety Disorder-7 Scale, Patient Health Questionnaire-9, and DSM-5 criteria, respectively.

Results: Females were common in both groups and the difference was statistically insignificant. Headache frequency was significantly more in patients with migraine with PNES ($P < 0.05$). However, headache intensity was similar in both groups. Patients with headaches and PNES identified triggers less commonly except for stress. Depression and somatoform symptom disorder were significantly more common in patients with migraine with PNES. Abnormal neurocircuitry involving frontal, limbic, and thalamic regions due to comorbid PNES may cause central sensitization, resulting in frequent migraine headaches which is further augmented by coexisting depression and somatoform-symptom-disease.

Conclusion: Migraine with PNES patients suffers more frequent headaches than patients with migraine without PNES. They differ in various headache triggers, with mental stress being the predominant trigger.

Keywords: Migraine, Psychogenic non-epileptic seizures, Depression, Somatoform symptom disease, Anxiety

INTRODUCTION

Migraine is frequently associated with a variety of comorbidities. These include vascular conditions, respiratory diseases, other pain syndromes, seizures, and psychiatric conditions.[1-3] Psychiatric comorbidities include anxiety, depression, post-traumatic stress disorders, and psychogenic non-epileptic seizures (PNES). Much information is available on the impact of psychiatric comorbidities such as anxiety and depression on migraine.[4-6] However, there is a lack of literature regarding the impact of comorbid PNES on migraine. Although PNES is much more commonly associated with migraine (19.2% in adult migraineurs) than epileptic seizures (1% in adult migraineurs), patients as well as treating physicians are usually more concerned about epileptic seizures.[7] Around 50–60% of PNES patients report migraine.[8] Migraine and PNES may have a complex interplay. Frequently, PNES patients do not get the desired attention and care due to the supposed non-organicity of their symptoms,[9] especially if they are not being treated at a specialized psychiatric facility. Several studies have shown the impact of associated chronic pain, headache, or migraine on patients with PNES.[10-12] However, studies looking into the effect of PNES on migraine characteristics are still few [Table 1]. Our study aimed to assess the impact of PNES on headache characteristics, triggers, and associated psychiatric comorbidities in patients with migraine.

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Table 1: Demographic and clinical characteristics in migraine with PNES and migraine without PNES.

Demographic profile	Migraine with PNES (*n*=52)	Migraine without PNES (*n*=48)	*P*-value
Age	24 (10–42)	30 (14–53)	<0.001
Gender			
Male	7 (13.7%)	10 (20.8%)	0.34
Female	44 (86.3%)	38 (79.2%)	
Duration of illness in months	24 (1–192)	24 (5–192)	0.93
Median headache frequency	17.5 (2–30)	7.5 (1–30)	0.001
Episodic: Chronic migraine	22:30	31: 17	0.03
Location of headache			
Hemicranial	20 (38.5%)	24 (50%)	0.24
Holocranial	32 (61.5%)	24 (50%)	
Pain characteristics			
Throbbing	45 (86.5%)	48 (100%)	0.013
Non-throbbing	7 (13.5%)	0	
Aura	3 (5.8%)	3 (6.2%)	1.00
Associated migrainous features			
Nausea	37	39	0.23
Vomiting	19	31	0.005
Photophobia	45	44	0.41
Phonophobia	45	46	1.04
Median pain severity	8 (5–10)	9 (4–10)	0.06
Headache hampering routine activity	49 (94.2%)	45 (93.8%)	1.00
Aggravation by physical activity	49 (94.2%)	45 (93.8%)	1.00

PNES: Psychogenic non-epileptic seizures, NES: Non-epileptic seizure

MATERIALS AND METHODS

Participants

The study was a cross-sectional and observational study. We included consecutive patients with migraine with PNES and without PNES from our neurology outdoor and indoor from June 2017 to May 2019. Migraine was diagnosed as per ICHD-3 criteria and PNES was diagnosed as per ILAE Non-epileptic Seizures Task Force criteria[13,14] [Figure 1]. We excluded patients with a history of true seizures and a structural lesion on neuroimaging known to cause epilepsy. The Institutional Ethics Committee approved the study (Ref code: 84th ECM IIA/P4). Written informed consent was taken from each subject before enrolment into the study.

Clinical assessments

We assessed all patients for their demographic details, duration of illness, headache characteristics, frequency of headache in the previous month, severity, and triggers. For the severity of migraine, we used 0–10 visual analog scale. All patients were also assessed for comorbid anxiety, depression, and somatic symptom disorder (SSD) with the help of Generalized Anxiety Disorder-7 (GAD-7), Patient Health Questionnaire-9 (PHQ-9), and DSM-5 criteria for SSD, respectively.[15-17] The examiner used the scales, ensuring the patient's total privacy and data were recorded on the pro forma. All the patients were subjected to cranial MRI/CT scans to rule out structural lesions. Migraine patients with PNES underwent Video-EEG (v-EEG) recording. In most patients, we did the recording at the time of the headache. PNES was induced using verbal suggestion and photic stimulation, and the caregiver corroborated the semiology. Baseline v-EEG was done for 10 min and continued till 30 min after the patient was induced for PNES. v-EEG continued for a minimum of 4 h if PNES could not be recorded. Electrocardiogram was done on all the patients to rule out cardiac conditions.

Sample size calculation

After considering the mean frequency of headache days in migraine, 8.46 ± 8.8 days/month and in migraine with PNES 15.1 ± 9.8 days/month,[18-20] confidence interval (two-sided) 95%, power 90%, and the ratio of sample size 1, the sample size for each group was estimated to be 42.

Statistical analysis

Categorical variables were presented as percentages and continuous variables were presented as median with range. Categorical variables were compared using the Chi-square test and Fisher exact test while continuous variables were compared by the Mann–Whitney test. $P < 0.05$ (two-tailed) was considered statistically significant. SPSS version 24 was used for statistical analysis.

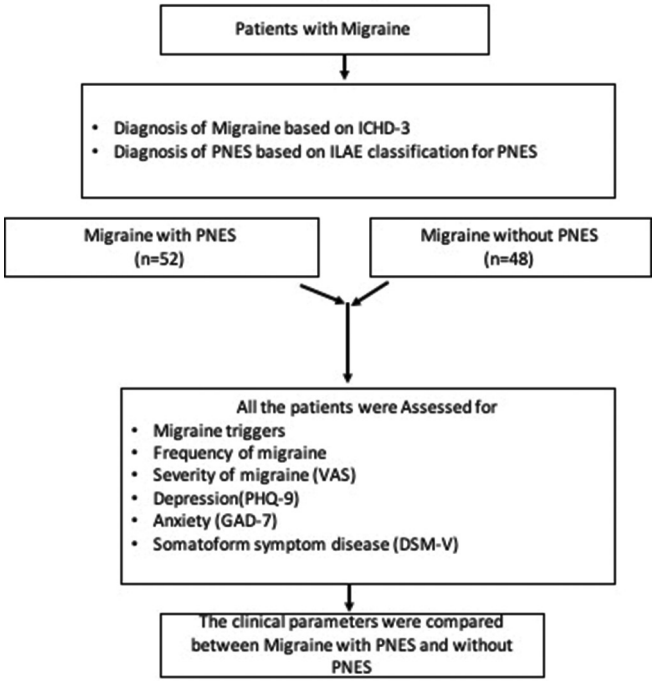

Figure 1: Flow diagram of study design.

RESULTS

Fifty-two patients with migraine with PNES and 48 patients with migraine without PNES were included in the study. Demographic and clinical characteristics are presented in [Table 1]. The median age was significantly lower in migraine with the PNES group (24 years) in comparison to migraine without the PNES group (30 years) ($P < 0.001$). Females outnumbered males in both groups. Median headache frequency in migraine with the PNES group (17.5/month) was significantly higher than migraine without PNES (7.5/month) ($P = 0.001$). The pain was throbbing in 86.5% and non-throbbing in 13.5% of migraine with PNES, while all patients without PNES had throbbing pain ($P = 0.013$). Vomiting was significantly more commonly associated with migraine without PNES ($P = 0.005$).

All the patients with migraine with PNES had normal v-EEG. The episode of PNES was clinically established in all PNES patients. In eight patients, an attack of PNES could be induced during v-EEG and further corroborated by the caregivers. The most common semiology was swoon (67.3%), often associated with hyperventilation and clenched teeth. Other semiologies included jerking of limbs, stiffness of limbs, isolated tongue protrusion, head-banging associated with flail limb movements, and opisthotonus in different combinations.

The number of triggers was significantly more in patients with migraine without PNES (1.62 ± 1.37) when compared with migraine with PNES (1.05 ± 0.99) ($P = 0.03$). Apart from this, some triggers include noise ($P < 0.001$), patients with migraine commonly reported sun exposure ($P = 0.003$), and weather change ($P = 0.02$) without PNES. On the contrary, mental stress as a headache trigger was more common in patients with migraine with PNES ($P = 0.009$) [Table 2].

The median PHQ-9 score was higher in migraine with PNES (14) in comparison to migraine without PNES (9) ($P = 0.03$). The proportion of patients with severe depression (PHQ-9 score ≥ 20) was more in patients with migraine with PNES ($P = 0.03$). There was no significant difference in GAD-7 scores between the two groups. SSD was more commonly associated with migraine with PNES (75%) than migraine without PNES (39.6%) ($P = 0.001$) [Table 3].

DISCUSSION

Our study underscores the impact of comorbid PNES on migraine characteristics. There are few studies which highlight the impact of co morbid PNES on migraine characteristics [Table 4]. In this study, we found that PNES significantly increases headache frequency. Although both groups had a similar duration of illness, the proportion of chronic migraine was more in migraine with PNES.

In PNES patients, increased metabolism in the thalamus has been demonstrated in a positron emission tomography study.[21] Hyperactivity of the thalamus due to comorbid PNES may cause central sensitization in these patients, leading to increasing headache episodes. Thalamic hyperactivation can also be further augmented by comorbid depression, which may contribute to the higher frequency of headaches.[22]

We concluded that despite of increased frequency of headache episodes, patients with migraine with PNES reported a lesser number of migraine triggers in comparison to migraine without PNES. It seems very difficult for migraine patients to identify and remember the specific triggers when headache frequency is high and overwhelming. Instead, a decrease in headache frequency with treatment may result in better identification of triggers, which has been observed by other experts, too.[23]

Although migraine with PNES patients reported an overall lesser number of migraine triggers, they reported mental stress as a headache trigger significantly more commonly than migraine without PNES. Patients with PNES usually remain in the state of hypervigilance which cause rapid hyperactivation in response to emotional cues and thus make these patients more prone to be adversely affected by mental stress.[24] We also found SSD more common in migraine with PNES. More robust connectivity between the insula and frontoparietal cortex has been demonstrated in patients with somatization, enhancing emotions' impact on perception, memory, and judgment capability.[25] Comorbid somatization further makes these patients susceptible to mental stress and has higher somatosensory sensitivity. Patients having comorbid anxiety and depression may have higher

Table 2: Headache triggers in migraine with PNES and migraine without PNES.

Migraine triggers	Migraine with PNES (*n*=52)	Migraine without PNES (*n*=48)	*P*-value
Number of patients reporting triggers	35 (67.3%)	38 (79.2%)	0.26
Number of triggers			
Mean±SD	1.05±0.99	1.62±1.37	0.03
Median (range)	1 (0–4)	1 (0–5)	
Triggers			
Mental stress	21 (40.4%)	8 (16.7%)	0.009
Noise	4 (7.7%)	38 (79.2%)	<0.001
Strong smell	4 (7.7%)	7 (14.6%)	0.34
Fasting	3 (5.8%)	8 (16.7%)	0.11
Reading	5 (9.6%)	1 (2.1%)	0.20
Sleep deprivation	3 (5.8%)	4 (8.3%)	0.70
Physical exertion	2 (3.8%)	3 (6.2%)	0.66
Sun exposure	1 (1.9%)	10 (20.8%)	0.003
Travelling	1 (1.9%)	4 (8.3%)	0.19
Weather change	1 (1.9%)	7 (14.6%)	0.02
Cold	0	2 (4.2%)	0.22
Cheese intake	0	1 (2.1%)	0.48
Smoke	1 (1.9%)	1 (2.1%)	1.00
Crowded space	6 (11.5%)	11 (22.9%)	0.18
Watching screen	3 (5.8%)	1 (2.1%)	0.61

PNES: Psychogenic non-epileptic seizures

Table 3: GAD-7, depressive symptoms (PHQ-9), and SSD in migraine with PNES and migraine without PNES.

Psychiatric comorbidities	Migraine with PNES (*n*=52)	Migraine without PNES (*n*=48)	*P*-value
Depression (PHQ-9)			
Median score (range)	14 (1–26)	9 (0–27)	0.03
Mild (5–9)	10 (19.2%)	15 (31.2%)	0.03
Moderate (10–14)	13 (25%)	7 (14.6%)	
Moderately severe (15–19)	8 (15.4%)	7 (14.6%)	
Severe (20–27)	15 (28.8%)	8 (16.7%)	
Generalized anxiety disorder (GAD-7)			
Median (range)	8 (1–21)	6 (0–21)	0.11
Minimal (0–4)	12 (23.1%)	19 (39.6%)	0.16
Mild (5–9)	17 (32.7%)	12 (25%)	
Moderate (5–9)	10 (19.2%)	7 (14.6%)	
Severe (15–21)	13 (25%)	10 (20.8%)	
Somatic symptom disorder (DSM-V)	39 (75%)	19 (39.6%)	0.001
Mild	21 (40.4%)	9 (18.8%)	0.004
Moderate	15 (28.8%)	9 (18.8%)	
Severe	3 (5.8%)	1 (2.1%)	

GAD-7: Generalized anxiety disorder, SSD: Somatic symptom disorder, PHQ-9: Patient Health Questionnaire-9, PNES: Psychogenic non-epileptic seizures

stress susceptibility. Several neurobiological correlates of this increased stress susceptibility are reported such as dysregulated gene expression, impaired neurocircuitry, and dysregulation in neuroimmunoendocrine functioning.[26] This higher stress susceptibility may lead to a pathological response to mental stress, enhanced vulnerability for PNES, and a higher propensity for migraine.[27] Comorbid SSD and depression negatively affect the quality of life in PNES patients.[28]

The pathophysiological connection between PNES and migraine is unknown. Hypersensitivity to visual, auditory, olfactory, and somatosensory stimuli is a well-recognized phenomenon in migraine.[19] Although sensory hypersensitivity in PNES patients has not been studied, many experts suggest that dysfunctional tolerance to sensory arousal is one of the important pathophysiological factors. Both hyperarousal and hypoarousal responses have been shown.[19,24] This can be the

Table 4: Summary table of studies on impact of PNES on migraine.

Authors	Country	Purpose	Sample size	Mean age (years), M: F	Study design	Target population	Summary of findings
Chakravarty *et al.* 2010[7]	India	To assess epileptic seizures and psychogenic non-epileptic seizures in subjects with migraine	1000 (200 children and 800 adults)	Children-11.8, 118:82 Adults-26, 144:656	Observational study	Children and adults with migraine	The study highlights the occurrence of PNES during acute attack of migraine. Almost one fourth of adult females with migraine had PNES at the time of headache. Semiology was observed as "swoon" in all patients.
Shepard *et al.* 2015[19]	USA	To investigate migraine characteristics in PNES	43 PNES with migraine and 29 Epilepsy with migraine	PNES-47.0±13.2, 8:35 Epilepsy-40.2±13.4, 4:25	Retrospective Study	Epilepsy and PNES patients with migraine	Patients with PNES reported more severe migraine (frequency and duration) compared to patients with Epilepsy
Gazzola *et al.* 2012[34]	USA	To assess chronic pain including migraine in PNES	85 PNES and 85 Idiopathic generalized epilepsy (control)	PNES-38.08, 17:68 Epelipsy-28.48, 38:47	Retrospective Case-control study	Epilepsy and PNES patients with chronic pain and migraine	Non-cephalic pain was frequent in patients with PNES. However, the prevalence of migraine did not differ between the two. More number of patients with PNES used opioid for pain compared to IGE patients.
Ettinger *et al.* 1999[35]	USA	To assess the frequency, location, and severity of pain in NES patients	56 NES patients	33±12. 16:40	Observational study	Patients with NES	About 77% of patients suffered moderate-to-severe pain, most commonly headache (61%) Pain is more frequent in patients with persistent NES than those with resolved NES.

PNES: Psychogenic non-epileptic seizures, NES: Non-epileptic seizure

reason that the presence of PNES affects migraine differently. Patients report a higher frequency of headache episodes but say migraine-specific throbbing headaches less commonly. Anxiety, depression, somatization, neuropathic pain, and fibromyalgia are associated with migraines and PNES.[5,29-32] The prevalence of migraine in PNES is 50–60%, much higher compared to the general population (~15%).

In addition, both conditions have common risk factors such as female gender, trauma, and childhood sexual abuse.[29,33] All the above factors may indicate the common pathophysiological link between the two disorders. However, our study is cross-sectional, observational, and experimental studies looking into the pathophysiological connection between two conditions are lacking.

CONCLUSION

Patients with migraine with PNES suffer more frequent headaches than those without PNES. They differ in various

headache triggers, mental stress being the predominant headache trigger in patients with migraine with PNES. Comorbid depression and somatization are more frequent in migraines with frequent headaches. Treating migraine with PNES requires a comprehensive approach, and including a neurologist, psychiatrist, and clinical psychologist may help improve the outcome.

Availability of data and material

The datasets generated during and or analyzed during the present study are not publicly available but are available from the corresponding author on reasonable request.

Authors' contributions

Ravi Uniyal – Conception, designing, analysis, interpretation of data, and drafting of the manuscript. Shweta Pandey – Conception, designing, analysis, interpretation of data, and drafting of the manuscript. Neeraj Kumar – Conception, designing, analysis, interpretation of data, and drafting of the manuscript. Ravindra Kumar Garg – Conception, designing, analysis, interpretation of data, and drafting of the manuscript. Hardeep Singh Malhotra – Analysis, interpretation of data, and drafting of manuscript. Imran Rizvi – Analysis, interpretation of data, and drafting of manuscript. Adarsh Tripathi – Analysis, interpretation of data, and drafting of manuscript.

Ethical approval

The study was duly approved by our Institutional Ethics Committee. (Ref code: 84th ECM IIA/P4).

Declaration of patient consent

The authors certify that they have obtained all appropriate consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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