Electroencephalographic changes of panic disorder patients in west Uttar Pradesh

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Abstract---80 panic disorder patients were diagnosed with electroencephalography (EEG). The study was electroencephalographic (EEG) changes of Panic Disorder patients in West Uttar Pradesh, India. The objectives 1) To examine the EEG of Panic Disorder patients. 2) To explore the clinical features for the diagnosis of panic disorder and their association with EEG. A large group of patients having panic disorder showed abnormal EEG associated with their clinical features. The pathophysiology behind is not clarified. The study was interpreted with 14 (17.5%) panic disorder patients and EEG abnormalities of 80 patients conflict with 13 symptoms in DSM-IV. The patients were assessed with Logistic regression analysis. EEG was selected as the dependent variable and age, sex, and with or without 13 symptoms as independent variables. The panic disorder patients showed abnormal EEG alpha frequency with repeatedly slow theta frequency. Nausea or abdominal distress (43.4% vs 67.7%, p =
derealization or depersonalization (37.7 % vs 82.4%, p = 0.005); or paresthesias (7.5% vs 47.1%, p = 0.001) were extracted by multivariate analysis as factors related to EEG abnormalities. The study indicated that physiological predispositions are closely related to panic disorder.

**Keywords**—panic attack, panic disorder, agoraphobia, DSM-IV, electroencephalography (EEG), alpha & theta frequency, nausea or abdominal distress, derealization or depersonalization, paresthesias.

**Panic disorder**

The neurobiological hypothesis of fear reported the awkwardness of stimuli from the cortex and brainstem causes an abnormal activation of the amygdale, with a behavioral, autonomic, and neuroendocrine stimulant. EEG changes in frontal and temporal cortices reported in Panic Disorder patients. A Panic Attack is a discrete period of sudden onset of intense apprehension, fearfulness, or terror, often associated with feelings of impending calamity with symptoms of breath, palpitations, chest pain or discomfort, choking or smothering sensations, and fear of "going crazy" or losing control are observed. The 13 somatic or cognitive symptoms are palpitations, sweating, trembling or shaking, sensation of shortness of breath or smothering, feeling of choking, chest pain or discomfort, nausea or abdominal distress, dizziness or lightheadedness, derealization or depersonalization, fear of losing control or going crazy, fear of dying, paresthesias and chills or hot flashes.

**Agoraphobia**

Agoraphobia is the anxiety of the humiliating situations of the unwanted environment of a person with a Panic Attack or panic-like symptoms. Panic Disorder without Agoraphobia is distinguished by repeated unanticipated Panic Attacks about which there is determine worry. Panic Disorder with Agoraphobia is distinguished by both repeated unwanted Panic Attacks and Agoraphobia. Agoraphobia without History of Panic Disorder is distinguished by the presence of Agoraphobia and panic-like symptoms without a history of unwanted Panic Attacks.

**Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)**

Diagnostic criteria of 13 symptoms in panic disorder patient is present in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The panic event causing panic attacks shown by symptoms are being consulted in hospitals of north India. 6.7% of primary care patients meet the diagnostic criteria for panic disorder and 28% of Panic Disorder patients consult emergency. The lifetime prevalence of Panic Disorder is 0.4% in Taiwan. According to National Comorbidity Survey (NCS) data, the lifetime prevalence of Panic Disorder in the United States is 3.5% & panic attacks is 7.3%. In Japan out of 4,000 subjects investigated cases, the prevalence of Panic Attack was 6.6% & Panic Disorder was 5%. The age of panic disorder is in early age and the risk in women is twice as
high as in men. In panic disorder, anticipatory anxiety and agoraphobia advance even between panic attacks and patients can be operationally diagnosed as having panic disorder from clinical symptoms. Recent studies on Panic Disorder in brain science suggest that lesions in the amygdala and hippocampus are deeply associated with the disorder and other cases blood flow decrease in the pre side frontal cortex is involved. Furthermore, genetic studies showed the hereditary panic disorder is in the range of 35-40% with similarity in identical twins being higher than that in fraternal twins. However, many studies report similarity in identical twins to be 50% or less.

**Materials and Methods**

**Study type**

The study was a hospital based cross sectional study carried out by the department of Psychiatry and Physiology present in S.M.M.H. Medical College Saharanpur district of Uttar Pradesh, North India.

**Methods**

Panic disorder patients who visited in the Department of Psychiatry S.M.M.H. medical college, Saharanpur in the period from June 2022 to September 2022 was considered for study. The study participants were informed about the consent of the ethics of the medical significance of EEG diagnosed with panic disorder. Prevalence of Panic Disorder was 5% with 95% confidence interval. Sample size of the study was 80(48 males, age 35.6 ± 7.3 years and 32 females, age 38.9 ± 6.8 years). The sample size was further divided into normal(31 males and 35 females, age 31.6 ± 9.3 years) and abnormal (7 males and 7 females, age 31.9.9 ± 9.8 years) patients. No healthy volunteer was present as control group for the study.

**Inclusion criteria**

Patients were examined by Psychiatrist to confirm that panic disorder patients undergo 13 symptoms of DSM-IV.

**Exclusion criteria**

- Patients with physical diseases such as arrhythmia, angina, hyperthyroidism, chronic obstructive pulmonary disease (COPD), asthma, pheochromocytoma, circulatory, respiratory, digestive, endocrine and neurological disease.
- Patients with epilepsy, schizophrenia, severe depression, and personality disorders.
- Patients that regularly take psychotropic drugs and other medicines.
- Patients having alcoholism or drug abuse.

The participants were made aware of the purpose and methods of the study and assured that personal data would not be disclosed. They were given the choice to decline to participate in the study without fearing any consequences. Clearance
from ethical committee S.M.M.H. Medical College, Saharanpur was obtained prior to the study.

**EEG recording**

EEG was assessed by Brain Tech 40+ system. The high pass filter was 1 Hz and the low pass filter was 70 Hz. During recording session, the participants were sitting in semi-reclined position with their neck and arm supported.18

**Electrodes placements**

Nation to inion- 10% or 20% rule was followed to locate the electrode and located FP - FZ - CZ - PZ - O points. The right and left auricular points -A1- CZ - A2 and located T3 - C3- CZ -C4 -T4. The points FP -T3 -O -T4 -FP and locate the points FP1 -F7 -T3 -T5 -O1 -O2 -T6 -F8 -FP2. The FP1 -O 1 -C3 points, FP1 -F3 -C3 points, and O1 -P1 -C3 points.FP2 –C4 –O2, FP2 – F4–C4 and O2-P4-C4. FP -FP1 -FP2 electrodes were placed in the frontal pole region of the frontal cortex.F3 -F4 electrodes were placed in the frontal cortex.F7 –F8 electrodes were placed in the inferior part of the frontal cortex.T3 -T4 electrodes were placed in the mid temporal region of the temporal cortex.T5 -T6 electrodes were placed in the posterior temporal region.C3-C4 electrodes were placed in the central region. P3-P4 electrodes were placed in the parietal region. O1-O2 electrodes were placed in the occipital region. A1-A2 electrodes were placed in the left and right auricular regions. Zero electrodes were placed in the midline of the cerebral cortex. FZ electrode was placed in the mid-frontal region.CZ electrodes were placed in the mid-central region. PZ electrodes were placed in the mid-parietal region of the cerebral cortex.18

**EEG reading and interpretation**

The type of EEG patterns such as 14 & 6 Hz positive spike, small sharp spikes 6 Hz spike and slow waves, psychomotor variant, subclinical rhythmic electroencephalographic discharge of adults and wicket spikes were considered abnormal as long as they were not frequent and their basic activity’s localization, rhythmicity &consecutiveness were stable.17

**Statistical analysis**

EEG findings were considered as dependent variables and the subjects having or not having the 13 symptoms mentioned in DSM-IV criteria, age, and sex were considered independent variables. Statistical analysis of data was based on the calculation and the distribution into classes according to the nature of each variable. Logistic regression analysis was performed among the variables. A p value < 0.05 was considered statistically significant. The independent variables were selected stepwise and selection under the likelihood ratio testing was selected. A statistical software IBM-SPSS version 17.0 was used for statistical analysis.
**Table 1**
Characteristics of patients with panic disorder sub classified on the basis of EEG findings

<table>
<thead>
<tr>
<th>Division of subgroups of panic disorder</th>
<th>EEG normal (Male/Female)</th>
<th>EEG abnormal (Male/Female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panic disorder With agrophobia</td>
<td>66(31/35)</td>
<td>14(7/7)</td>
</tr>
<tr>
<td>Panic disorder with depression</td>
<td>33(23/10)</td>
<td>8(3/5)</td>
</tr>
<tr>
<td>Panic disorder with somatoform disorder</td>
<td>24(14/10)</td>
<td>5(2/3)</td>
</tr>
<tr>
<td>Panic disorder with past depression</td>
<td>11(8/3)</td>
<td>2(2/0)</td>
</tr>
<tr>
<td>Panic disorder with past somatoform disorder</td>
<td>27(14/13)</td>
<td>9(3/6)</td>
</tr>
</tbody>
</table>

**Table 2**
Abnormal EEG findings of 14 cases

<table>
<thead>
<tr>
<th>No.</th>
<th>age</th>
<th>sex</th>
<th>Basic activity</th>
<th>Abnormal findings</th>
<th>Focus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22</td>
<td>female</td>
<td>9-10 Hz irregular</td>
<td>Slow wave burst (θ)</td>
<td>PO-diffuse</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>female</td>
<td>10-12 Hz irregular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>3*</td>
<td>42</td>
<td>female</td>
<td>10 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>Right CPO</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>female</td>
<td>9 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>Diffuse</td>
</tr>
<tr>
<td>5</td>
<td>31</td>
<td>female</td>
<td>8-10 Hz irregular</td>
<td>Slow waves (7 hz)</td>
<td>intermingled at basic activity</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>female</td>
<td>10-11 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>7</td>
<td>31</td>
<td>female</td>
<td>10-11 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>8</td>
<td>36</td>
<td>male</td>
<td>9 Hz irregular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>9</td>
<td>33</td>
<td>male</td>
<td>10-12 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>Diffuse</td>
</tr>
<tr>
<td>10</td>
<td>23</td>
<td>male</td>
<td>9-11 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>11</td>
<td>33</td>
<td>male</td>
<td>10-11 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>CPO</td>
</tr>
<tr>
<td>12*</td>
<td>40</td>
<td>male</td>
<td>8-10 Hz irregular</td>
<td>Slow wave burst (θ)</td>
<td>Diffuse</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Right Fp</td>
</tr>
<tr>
<td>13</td>
<td>34</td>
<td>male</td>
<td>8-10 Hz irregular</td>
<td>Slow waves (7 hz)</td>
<td>intermingled at basic activity</td>
</tr>
<tr>
<td>14</td>
<td>38</td>
<td>male</td>
<td>10-11 Hz regular</td>
<td>Slow wave burst (θ)</td>
<td>Left CPO</td>
</tr>
</tbody>
</table>
Fig 1. EEG Result: Basic activity is 10 Hz 20–50 μV with regular alpha waves. 6-7 Hz θ wave activities appear in the posterior and central channels occasionally. Judgment of this EEG is slightly abnormal.

Fig 2. EEG Result: Basic activity is 8-10 Hz 20–50 μV with irregular alpha wave. 6-7 Hz θ wave activities appear in the posterior and central channels occasionally. Judgment of this EEG is slightly abnormal.
Table 3
Comparison of 13 symptoms with the EEG findings of panic disorder patient

<table>
<thead>
<tr>
<th>Symptoms mentioned in DSM-IV</th>
<th>EEG frequency of normal patients (percent)</th>
<th>EEG frequency of abnormal patients (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>palpitations, pounding heart, or accelerated heart rate</td>
<td>16 (24.2%)</td>
<td>5 (35.7%)</td>
</tr>
<tr>
<td>sweating</td>
<td>23 (34.8%)</td>
<td>11 (78.5%)</td>
</tr>
<tr>
<td>trembling or shaking</td>
<td>11 (16.6%)</td>
<td>7 (50%)</td>
</tr>
<tr>
<td>sensations of shortness of breath or smothering</td>
<td>40 (60.6%)</td>
<td>13 (92.8%)</td>
</tr>
<tr>
<td>feeling of choking</td>
<td>20 (30.3%)</td>
<td>4 (28.5%)</td>
</tr>
<tr>
<td>chest pain or discomfort</td>
<td>38 (57.5%)</td>
<td>11 (78.5%)</td>
</tr>
<tr>
<td>nausea or abdominal distress</td>
<td>29 (43.4%)</td>
<td>9 (64.7%)</td>
</tr>
<tr>
<td>feeling dizzy, unsteady, lightheaded, or faint</td>
<td>23 (34.8%)</td>
<td>11 (78.5%)</td>
</tr>
<tr>
<td>derealization or depersonalization</td>
<td>25 (37.7%)</td>
<td>12 (82.4%)</td>
</tr>
<tr>
<td>fear of losing control or going crazy</td>
<td>23 (43.4%)</td>
<td>11 (64.7%)</td>
</tr>
<tr>
<td>fear of dying</td>
<td>16 (30.2)</td>
<td>5 (29.4%)</td>
</tr>
<tr>
<td>paresthesias</td>
<td>5 (7.5%)</td>
<td>7 (47.1%)</td>
</tr>
<tr>
<td>chills or hot flushes</td>
<td>11 (20.8%)</td>
<td>7 (41.2%)</td>
</tr>
<tr>
<td>Age(years)</td>
<td>31.6 ± 9.3 years</td>
<td>31.9 ± 9.8 years</td>
</tr>
<tr>
<td>Sex(number)</td>
<td>Male:31</td>
<td>Female:35</td>
</tr>
<tr>
<td></td>
<td>Male:7</td>
<td>Female:7</td>
</tr>
</tbody>
</table>

Table 4
EEG findings of panic disorder patients by the three factors extracted by multiple regression analysis

<table>
<thead>
<tr>
<th>Symptoms mentioned in DSM-IV</th>
<th>EEG normal patients = 53(%)</th>
<th>EEG abnormal patients = 14(%)</th>
<th>Wald</th>
<th>OR</th>
<th>95%CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>nausea or abdominal distress</td>
<td>29 (43.4)</td>
<td>9 (64.7)</td>
<td>6.261</td>
<td>7.928</td>
<td>1.567-40.082</td>
<td>0.01</td>
</tr>
<tr>
<td>derealization or depersonalization</td>
<td>or</td>
<td>25 (37.7)</td>
<td>6.261</td>
<td>9.102</td>
<td>12.489</td>
<td>2.422-64.426</td>
</tr>
<tr>
<td>paresthesias</td>
<td>5 (7.5)</td>
<td>7 (47.1)</td>
<td>9.369</td>
<td>13.919</td>
<td>2.579-75.083</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Results

First the Panic Disorder patients were examined with EEG. 31 males and 35 females panic disorder patients was evaluated with normal basic activity but 7 males & 7 females was interpreted with abnormal EEG wave patterns. Then the Panic Disorder patients were divided into normal and abnormal groups. Each group further divided into 7 subgroups. With agrophobia subgroup 23 males & 10 females showed normal basic activity but 3 male and 5 female showed abnormal EEG. 14 males & 10 females reported normal but 2 male and 3 female showed abnormal EEG in Panic disorder with depression sub group. EEG in Panic disorder with somatoform disorder subgroup 8 males and 3 females basic activity was normal but 2 male showed abnormal EEG. In Panic disorder with
past depression sub group 14 males and 13 females were normal but 3 male and 6 female evaluated abnormal EEG. 4 males reported normal but 2 female reported abnormal EEG in Panic disorder with past somatoform disorder group. (Table 1) Abnormal EEG finding was reported in 14 cases of panic disorder patients.

Case 1 displayed 9-10 hz irregular alpha frequency and slow wave burst theta frequency with PO-diffuse. 10-12 hz irregular alpha frequency and slow wave burst theta frequency with CPO in Case 2 was detected. Case 3 was evaluated with slow wave burst theta frequency and right CPO & 10 hz regular alpha frequency (Fig 1). Case 4 showed 9 hz regular alpha frequency and slow burst theta frequency as well with diffuse pattern of wavelength. Case 5 reported intermingled with basic activity slow wave frequency 7hz & 8-10 hz irregular alpha frequency. Case 6 & Case 7 both revealed 10-11 hz regular alpha frequency & slow burst theta frequency with CPO activity. Case 8 exhibit 9 hz irregular alpha frequency & slow burst theta frequency with CPO. Case 9 displayed 10-12 hz regular alpha frequency and slow burst theta frequency with diffuse type. Case 10 was involved in regular 9-11 hz alpha frequency & sharp wave of 6 hz with CPO. Case 11 focus on slow burst theta frequency with CPO 10-11 hz & regular alpha frequency. Case 12 patient reported 8-10 hz regular alpha frequency & slow burst theta frequency & positive spike wave with diffuse right front parietal (Fig 2). Case 13 was examined slow wave frequency of 7hz intermingled at basic activity & 8-10 hz irregular alpha frequency.

Case 14 present 10-11 hz regular alpha frequency and slow waves burst theta frequency with left CPO type. (Table 2) Comparing 13 symptoms with the EEG findings of panic disorder patient announced that palpitations, pounding heart, or accelerated heart rate was offered by normal 24.2% & 35.7% patients. Normal 34.8% & abnormal 78.5% patients felt Sweating.16.6% normal & 50% abnormal patients disclosed Trembling or shaking sensations. Sensations of shortness of breath or smothering was expressed with 60.6% normal & 92.8% abnormal patients. 30.3% normal but 28.5% abnormal patients was evaluated with Feeling of choking symptom. 57.57% normal & 78.57% abnormal patients was examined with Chest pain or discomfort. Nausea or abdominal distress symptom was depicted by 43.4% normal & 64.7% abnormal patients. Feeling dizzy, unsteady, lightheaded, or faint symptom was present in 34.8%normal & 78.57% abnormal patients. Derealization (feelings of unreality) or depersonalization (being detached from oneself) behaviour was judged in 37.7% normal vs 82.4% abnormal patients. 43.4% normal & 64.7% abnormal patients experienced fear of losing control or going crazy. 30.2% normal & 29.4% abnormal patients experienced fear of dying group. 7.5% normal but 47.1% abnormal patients sensitized paresthesias (numbness or tingling sensations). Chills or hot flushes was felt by 20.8% normal but 41.2% abnormal patients.(Table 3) Nausea or abdominal distress (43.4% vs 64.7%, OR = 7.93, 95%CI: 1.567-40.082, p = 0.01); derealization (feelings of unreality) or depersonalization (being detached from oneself) (37.7 %vs82.4%, OR = 12.49, 95%CI: 2.422-64.083, p = 0.005); or paresthesias (numbness or tingling sensations) (7.5%vs47.1%, OR = 13.92, 95%CI: 2.58-75.083, p = 0.001) were extracted by multivariate analysis as factors related to EEG abnormalities (Table 4).
Discussion

The hypothesis that panic disorder patients enlist in decorated memory processing of threat-related stimuli. The studies shows that amygdale activation may be carried confusion of sensory information and the outpouring of events that is learned as a Panic Attack. Decreased activity in the pre side frontal cortex showed impaired control over the amygdale flammable and control of fear response.\textsuperscript{19} The low P300 wave amplitude in panic disorder patients may be confirmation of directed attention and short-term memory deterioration.\textsuperscript{20} The alpha and beta frequency in anxiety point is to a lower alpha frequency and beta rhythms are greater rational with the hypothesis of the fear neurocircuitry.\textsuperscript{20} Alpha event-related synchronism is affected to follow top-down and inhibitory control processes & decreased alpha frequency is closely associated with active cognitive processing, introspective excitatory brain processes shows dysfunction in thalamic-cortical circuits identical with an inability to inhibit insignificant information, a role played especially by the pre side frontal cortex.\textsuperscript{21} Abnormal improvement of beta frequency is leading to an abnormal endurance of the status and losses identical behavioral and cognitive control.\textsuperscript{21} Marked depression in the alpha rhythm activity, combined to a significantly increased beta activity in patients with agoraphobia may clue: 1) a decrease in the thalamo-cortical system synchronization; 2) an increase in extra thalamic element from the mesencephalic reticular formation; 3) a relationship with high levels of the conscious and domestic anxiety and depression of patients. The hypothesis states that inhibitory signals of the pre side frontal cortex that controls the sub cortical regions generate autonomic, neuroendocrine, and behavioral anxiety symptoms.\textsuperscript{21}

Studies showed increased right frontal side hemisphere activation or like decreased frontal alpha frequency granted in panic disorder patients shows acute emotional reaction associated with withdrawal behaviour.\textsuperscript{22} Phillips et al.\textsuperscript{23} states ventral network which including the amygdale, insula, ventral striatum, ventral anterior cingulate gyros and prefrontal cortex is involved in physiological or psychological production of emotional states and the automatic regulation of emotional responses. The dorsal network is compensated the regulation of behavior according to the situation. The left side pre frontal cortex guides and control the information processing needed to produce voluntary movement ie tendency of fight and flight reaction.\textsuperscript{24}

Our study participants were 80 compared to Hayashi et al.\textsuperscript{17} with 70 participants. Our study did not find any Paroxysmal abnormality interpreted as epileptiform that was found in 2 cases. The EEG abnormalities were slow waves break out in temporally repeated, found in all cases compared to 11 out of 17 cases. No localization of alpha frequency & no difference between right and left, 1 case dominant in right and 1 in left side compared to 4 cases were dominant in the right side. No slow wave complex was found and amplitudes were low or medium, were not considered as epileptiform discharges. In 8 of the 14 cases, found hyperventilation, although all patients recovered in 30 seconds compared to 10 out of 17 cases. EEG findings of panic disorder patients (43.4\% vs 64.7\% p = 0.01) compared to (37.7\% vs 82.4\%p = 0.003); derealization or depersonalization 37.7 \%vs82.4\% p = 0.005) compared to (7.5\% vs 47.1\%, p = 0.002); or
paresthesias (7.5% vs 47.1% p = 0.001) compared to (43.4% vs 64.7%, p = 0.012) were EEG abnormalities.

In a study, there were 25% of EEG abnormal patients, and 15% of the patients had slow wave activity in the temporal regions with occasional bursts of sharp waves identified as epileptiform discharges and 10% of them had nonspecific increases in generalized slow wave activity support. Stein et al. the sample size was only 35 could not find many EEG abnormalities in their patients with panic disorder (14.3%) not support. Another study with 54 patients the EEGs in non-medicated conditions and to compare the EEG with Computed Tomography (CT) results 13 patients displayed abnormal EEG recordings, and 6 patients exist abnormal CT scans support. The study of 120 subjects in which 35 patients (29.2%) examined, showed EEG abnormalities. The study of 21 panic disorder patients with 20 healthy volunteers 5 patients (23.8%) showed EEG abnormalities and less alpha frequency in the right temporal region support. The EEG abnormality rate reported for healthy people is in the range of 4.9-10% support. Our prosecution also showed a high EEG abnormality rate 17.5% among patients with panic disorder, although we had no control subjects.

Based on the findings, it was considered that i). Many panic disorder patients had EEG abnormalities. ii). Increased rate of EEG abnormalities in panic disorder patients might have some accord with physiological burden that easily cause panic attacks. In this study, nausea or abdominal distress, derealization or depersonalization and paresthesias were examined by multivariate analysis related to EEG abnormalities. 15 patients found EEG changes during panic attacks, but could not extract any specific symptoms which might be related to EEG abnormalities support. Stein et al. could not find any relation between EEG abnormalities and derealization, depersonalization, visual or auditory perceptual disturbances, and forced thinking not supported. Subjects with EEG abnormalities might be conscious to nausea with direct stimulations of the vomiting center in the medulla. Gibbs & Gibbs expressed that patients with paroxysmal slow activities, the episodes of nausea or vomiting is higher than that of a normal subject group support. Edlund reported that 4 of 6 patients with atypical panic attacks involving aggression, anger, severe demoralization, and social withdrawal with temporal EEG abnormalities that could not be clearly considered epilepsy support. Ietsugu et al. reported that Paresthesias is a good indicator of severe panic attacks supported. Nishimura et al, panic disorder patients with first-degree familial history are originally younger at onset, show more symptoms, and have more constant attacks with paresthesias and chills or hot flashes at first panic attack correlated with patients without first-degree familial history.

**Study limitations**

The study was investigated with a small group of participants and was necessary to compare with healthy individual. We grouped all patients together with abnormal EEG findings and thus could not justify the relationships between specific symptoms and specific EEG findings. Further studies with a larger number of participants can analyze our findings.
Conclusions

In this study, we observed 14 EEG abnormalities in 80 panic disorder patients. Most abnormal findings were slow wave bursts. These abnormalities were non-specific. Nausea or abdominal distress, derealization or depersonalization and paresthesias were extracted as factors related to EEG abnormalities in panic disorder.

Competing interests

The authors declare that they have no competing interest.

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