Psychogenic nonepileptic seizures: A case series

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Abstract

Manifestation of abnormal excessive synchronous cortical activity, which is what defines epileptic seizures, is typically absent in seizures with psychological etiology, a heterogeneous set of factors that have been identified. Distinguishing psychogenic nonepileptic seizures (PNES) from epileptic seizures may be difficult at the bedside. We report a case series of PNES which presented with diagnostic dilemma. Diagnostic delay of years with PNES is common. The exact choice of treatment is often difficult and should be based on individual differences in the underlying factors. Outcome can be measured in terms of seizure occurrence (frequency, severity), but other measures might be of greater importance for the patient. Prognosis is varied but studies consistently report that 1/3rd to 1/4th of these cases become chronic.

Keywords: Conversion, electroencephalography, nonepileptic seizure, seizure

Case 1

A 36-year-old lady, B.Ed. in English, married for 15 years, was brought to psychiatry OPD by husband with c/o episodes of altered consciousness with involuntary body movements, giddiness, low mood, and crying spells with disturbed sleep since a year increased for 2 months. She was apparently alright around 1 year back when she got the news of demise of one of her distant relatives and immediately after showed involuntary movements of limbs and altered consciousness. This was not associated with tongue bite, incontinence, or any physical injury. She recovered within 10–15 min without any intervention. She had two more episodes in the subsequent 1 month due to which she was referred to psychiatry OPD.

PNES are typically caused by psychological conditions. Psychogenic nonepileptic seizures (PNES) present as paroxysmal time-limited alterations in motor, sensory, autonomic, and/or cognitive signs but have no ictal epileptiform activity.

Distinguishing PNES from epileptic seizures may be difficult at the bedside. Diagnostic delay of years with PNES is common.[] The evolution of epilepsy monitoring units and the ability to utilize simultaneous video and electroencephalography EEG recordings may be a key to diagnosis. Video EEG of a typical event showing the absence of epileptiform activity during the spell with a compatible history is regarded as the gold standard for diagnosis.[4]
shown to a private practitioner who started her on tablet escitalopram 10 mg OD and tablet flupenthixol 0.5 mg. She was symptom free for 3 months and stopped taking medications on her own. About 3 months back, while teaching in school, she heard the news of her brother’s accident. Following this, she had an episode of loss of consciousness associated with tongue bite and an injury to the earlobe and involuntary passage of urine, along with postictal confusion and amnesia of the event. In the following month, she had 3 similar episodes, each one after interpersonal issues with the husband regarding their financial condition. All these episodes were associated with loss of consciousness and injuries wherein on one occasion, she hurt her knee and on another one, she injured her foot and fractured her toe. She had persistent sadness of mood, worrying thoughts about the financial condition at home as she had to leave her job due to these episodes. Sleep was disturbed with delayed onset of sleep and early morning awakening. On mental status examination (MSE), there were worrying thoughts, preoccupation with her illness of seizures, with ideas of helplessness. Due to injuries, and involuntary passage of urine, she referred to neurologist for evaluation. Initially, she was prescribed tablet lacosamide 100 mg BD; but after no significant finding on Video EEG,

### Table 1: Features of seizure and pseudoseizure and of the three patients

| Feature                              | Seizure                                | Pseudoseizure | Case 1                      | Case 2                      | Case 3                      |
|--------------------------------------|----------------------------------------|---------------|-----------------------------|-----------------------------|-----------------------------|
| Cause                                | Organic/metabolic                       | Generalized tonic-clonic movements | No organic cause seen       | Organic cause seen          | No organic cause seen       |
| Precipitant                          | May be seen                             | Usually seen  | Present                     | Present                     | Present                     |
| Occurrence in sleep                  | May be seen                             | Not seen      | Not seen                    | Not seen                    | Not seen                    |
| Onset                                | Abrupt                                  | Gradual       | Gradual                     | Gradual                     | Abrupt                      |
| Movements                            | Usually generalized tonic-clonic        | Nonsynchronous out of phase movements or lie motionless | Nonsynchronous out of phase movements or lie motionless | Both in different time frames | Nonsynchronous out of phase movements |
| Duration                             | Short, up to (1-2 min)                 | Prolonged, variable (10-15 min) | Prolonged                   | Initially short but later prolonged | Prolonged                   |
| Consciousness                        | Lost and unresponsive to pain           | Usually preserved | Preserved                   | Both in different episodes  | Preserved                   |
| Aura                                 | Usually present                         | Unusual except for symptoms of hyperventilation | Not seen                   | Not seen                    | Not seen                    |
| Injury                               | Frequent injuries, tongue bite          | Injuries absent | Present                     | Not present                 | Not present                 |
| Reflexes                             | Babinski reflex and pupillary constriction after seizure | No pathological reflexes | No pathological reflexes     | No pathological reflexes    | No pathological reflexes    |
| Postictal confusion or transient paralysis | Present                             | Minimal and patient unconcerned    | Minimal and patient unconcerned | Minimal and patient unconcerned | Minimal and patient unconcerned |
| Amnesia                              | Present                                 | May or may not be seen             | Not present                 | Not present                 | Not present                 |
| Witness                              | Independent of witness                  | Usually witness present            | Always present              | Always present              | Always present              |
| Induction by suggestion               | Not seen                                | Readily induced or stopped         | Always present              | Always present              | -                           |
| Induction by photic stimuli/sleep deprivation/hyperventilation | Often precipitated                     | Not present                     | Not present                 | Not present                 | Not present                 |
| Prolactin and creatine kinase levels after attack | Rises                                 | Normal                         | Normal                      | Normal                      | Normal                      |
| EEG                                  | Epileptic changes in majority (VEEG preferred) | No epileptiform discharges        | No epileptiform discharges  | No epileptiform discharges  | No epileptiform discharges  |
| MRI                                  | Changes seen                            | Usually normal                   | Within normal limits        | Gloss in left temporal lobe and FLAIR hyper intensity within left amygdala | Within normal limits        |
| Response to treatment                | Often present                           | Intractable despite adequate medication | Seen                       | Seen                        | -                           |
| Management                           | Anticonvulsants/treatment of the cause  | Psychiatric management            | Combined treatment          | Combined treatment          | Combined treatment          |

EEG – Electroencephalography; MRI – Magnetic resonance imaging; VEEG – Video EEG; FLAIR – Fluid-attenuated inversion recovery
it was discontinued. Magnetic resonance imaging (MRI) brain showed a relatively small right hippocampus with prominent temporal horn suggestive of? Early signs of right medial temporal sclerosis. With a diagnosis of dissociative convulsions, the patient was started on fluoxetine 20 mg and clonazepam 0.25 mg sos, along with psychotherapy with improvement in her clinical status.

**Case 2**

A 37-year-old lady living with husband, educated up to 12th standard, housewife, K/C/O seizure disorder under regular treatment with tablet divalproate 1000 mg HS, tablet aripiprazole 5 mg OD, tablet escitalopram 10 mg OD, came to the psychiatry OPD with her husband, currently c/o episodes of involuntary body movements with uprolling of eyeballs, clenching of jaw, deviation of tongue to one side, dribbling of saliva from the corner of the mouth – lasting for 1–2 min. There was also low mood, with crying spells and reduced interest in previously pleasurable activities, apprehension with palpitations, worrying thoughts about financial constraints, irritability and anger outbursts, accompanied with aggressive behavior with a passive death wish but no suicidal ideas for a duration of 4 years, increased for 3 months. In the month of September, 2020, her father tested positive for COVID-19, which had an impact on her and she was crying for days after that would not eat or interact normally and would mostly keep to herself. She became disinterested in daily activities and socialization and would mostly lay back in her spot in the house, either sleepy or distractedly lost in her thoughts. She began complaining of dizziness, which would begin abruptly and independent of postural changes. On the 10th of November, 2020, she had an episode of involuntary body movements. There were uniform jerky movements in all four limbs, without any facial deviations. It was accompanied with uprolling of eyeballs, clenching jaw, and urinary incontinence. It was not associated with tongue bite. It lasted for 1–2 min, after which the patient was unconscious for 15–20 min. After waking up, she was very confused about her whereabouts and was complaining of nausea for around 30–40 min. Along with all of this, she was also saying that she wishes to die because her existence is futile and that it would be beneficial to everyone if only God were to take her life away. On MSE, worrying thoughts were present with a preoccupation with her illness, ideas of helplessness, with a passive death wish a fair judgment and insight.

In 2012, she began experiencing frequent episodes of dizziness, occurring once or twice a month for which she took medication. She claims to have become irregular with her medication from the year 2013 because she apparently started feeling well and healthy. Earlier in 2015, she got divorced, and toward the end of 2015, she remarried. On her first visit to the hospital, she had an episode of dizziness and fell while waiting at the queue to the medicine OPD. Over there, on OPD basis, she was started on tablet escitalopram 10 mg 1-0-2, tablet haloperidol 0.25 mg 1-0-1, tablet olanzapine 300 mg 1-0-1, tablet clonazepam 0.5 mg 0-0-1, tablet clobazam 5 mg 1-0-1, and tablet folic acid 5 mg 0-0-1. She took the above medication regularly and would maintain regular follow-ups. After that, the frequency of involuntary body movements increased to 1–2 per month, on an average. From the year 2016, she started becoming irritable toward people around her, including her husband. She also developed features of low mood, accompanied with multiple crying spells. From March 2019, the patient started visiting a private practitioner, where she was prescribed tablet oxcarbazepine 300 mg BD, tablet escitalopram 10 mg HS, tablet haloperidol 0.25 mg OD, tablet etizolam 0.5 mg, and tablet propranolol 20 mg OD. She was under the above regimen, when in the month of August in 2020, when the patient had an episode of involuntary body movements that lasted for 1 h, with occasional relaxations in between. Such episodes continue till she visited the psychiatry OPD. The patient is a known case of hypothyroidism under treatment of tablet thyroxine 50 mcg OD. With normal EEG findings, MRI showing gloss in left temporal lobe and fluid-attenuated inversion-recovery hyperintensity within left amygdala? Postseizure transient abnormality, a diagnosis of seizure with pseudoseizure was made, and tablet oxcarbazepine was stopped, tablet valproate increased to 1000 mg HS, and tablet escitalopram increased to 15 mg HS. The number of episodes gradually started declining and from multiple times, a day came down to once in 10 days within the 1st month of treatment. Supportive psychotherapy and family-oriented therapy for positive expressed emotions and need for compliance with medications. After 2 months of pharmacotherapy with psychotherapy, she is maintaining well with no episodes of involuntary body movements.

**Case 3**

A 19-year-old MBBS student was brought to the neurology OPD, after she had an episode of altered consciousness, with uprolling of eyeballs an evening before the day she was brought to the OPD after an altercation she had with her parents over a phone call. It was not associated with any involuntary body movements, frothing at mouth, postictal confusion, aura, and she recalled the event faintly. After complete neurological examination and evaluation, with MRI, EEG, and other tests within normal limits, she was referred to Psychiatry OPD. She was at home for 8 months, during which she lost her grandmother whom she was very close to. After returning back to college a few days back, she was informed that her semester examinations will be held soon and she was not prepared for the same because of the difficult circumstances at home. On the day of the episode,
she had a verbal altercation with her parents regarding her studies after she told them that she is not able to adjust after coming back. On MSE, she was fidgety, repeatedly crackling her knuckles, communicative, oriented to time, place and person, maintaining eye-to-eye contact with normal psychomotor activity, revealing worrying thoughts regarding real-life stressors regarding examinations, and her relationship with her parents, with a fair insight and judgment. She was started on tablet escitalopram 5 mg OD which will gradually be uptitrated as per response along with psychotherapy, focusing on her coping skills. She has maintained improvement on follow-up.

**DISCUSSION**

Pseudoseizure is a term for events that appear to be seizures but do not represent the manifestation of abnormal excessive synchronous cortical activity, which is what defines epileptic seizures. They are of psychiatric origin and are not a variation of epilepsy. Other terms used in the past include hysterical seizures, psychogenic seizures, and others. The current most standard terminology is PNES. Some still use other terms such as psychogenic functional spells or psychogenic nonepileptic spells, events, or attacks. These terms reinforce that the events are not epileptic in origin.[5-7]

It is observed that the common etiologies associated with pseudoseizures are most often related to personal conflicts of emotions, which can be parental misbehavior, strained inter-personal relationships, sudden death of a loved one, scholastic pressure, etc. In simple non-Freudian terms, dissociation is an unconscious expression of emotional conflicts in the form of physical symptoms. It is this unconscious expression that differentiates hysteria from malingering or hypochondriasis.

Correct diagnosis is necessary for successful treatment. Patients with psychogenic nonepileptic spells have frequently been misdiagnosed as having epilepsy and have been prescribed multiple medications. Consultation with neurology may be helpful. Admission to a monitoring unit may be in order if the diagnosis is uncertain. Long-term video EEG monitoring is the most important diagnostic test.[8] Laboratory testing is of limited utility. Serum prolactin levels have long been noted to increase shortly after a generalized epileptic seizure but not after PNES. A lactic acidosis commonly follows a generalized convulsion. However, a rise in lactate levels is not specific for convulsions of epileptic origin; elevated lactate levels occurred in volunteers simulating generalized seizures.[9] Elevated creatine kinase levels after generalized convulsive status epilepticus were observed compared to patients with psychogenic nonepileptic status epilepticus and may be useful in distinguishing psychogenic status epilepticus from generalized convulsive status epilepticus.[10] In challenging cases, admission to an epilepsy monitoring unit or similar facility with combined video EEG monitoring may be needed to secure the diagnosis [Table1]. The best treatment is not known but may consist of a combination of medication if depression or anxiety exists and cognitive behavioral therapy. An honest and clear discussion of the patient’s diagnosis is of utmost importance. In cases of conversion disorder, it is important to acknowledge that the spells are real and cause distress to the patient, family, and friends. It should be articulated that the episodes are not seizures. A respectful approach and the reassurance that supportive therapy will most likely decrease or even eliminate the frequency of spells should be outlined.

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There are no conflicts of interest.

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