Psychogenic Non-Epileptic Seizures: A Case Report

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Abstract

Psychogenic non-epileptiform seizures (PNES) are a common condition that affects over 400,000 individuals within the United States. PNES are a challenging entity in modern medicine, for they are located at the interface between neurology and psychiatry regarding clinical presentation and pathophysiology. The experiences and symptomatology of the patients resemble those associated with epileptic seizure activity, however many patients present with co-occurring psychiatric comorbidity. A combination of video-electroencephalography and a concise, well-documented clinical history of the event helps in a definitive clinical diagnosis of PNES. Measuring the levels of serum prolactin may prove useful as an adjunctive laboratory test in diagnosing PNES. Management of PNES involves prompt patient education regarding the condition and treating the psychiatric comorbidity as well. A combined approach of both psychotherapeutic and pharmacological interventions helps in the optimal treatment of PNES.
Introduction

Epilepsy is a neurological condition which is defined as multiple unprovoked seizures due to abnormal electrical activity in the brain and is commonly associated with an anatomic seizure focus or impaired synchrony of neuronal impulse firing. In some patients, a psychiatric illness may be an etiological cause of seizure activity without focal neurological deficits; such patients experience what are known as psychogenic non-epileptic seizures (PNES) and are a common condition that affects over 400,000 individuals within the United States[1]. They usually present with symptoms that closely resemble an epileptic fit activity leading to a misdiagnosis. However, a significant percentage of the patients often present with co-occurring psychiatric comorbidity. Generalized anxiety disorder, conversion disorder, dissociative disorders, major depressive disorder, post-traumatic stress disorder, and personality disorders [2] are the most symbolic associations. Hence, PNES are a challenging entity in modern medicine, for they are located at the interface between neurology and psychiatry regarding clinical presentation and pathophysiology. Although the pathophysiology of PNES is unknown, they may act as a physical outlet for intense, overwhelming feelings such as anxiety and depression. Psychiatric comorbidities screening is always essential in PNES as the management involves prompt treatment of the underlying cause. Of the multiple psychiatric comorbidities associated with PNES, many patients show up with generalized anxiety disorder and/or major depressive disorder; therefore, use of the GAD-7 and PHQ-9 examination for every patient may be useful for screening for anxiety and depression, respectively. Herein, we report a young female, diagnosed with generalized anxiety disorder, presented with an episode of rapid alternating movements of her body and lack of neurological abnormalities on examination.

Case Presentation

A 32-year-old female patient presents to the primary care clinic accompanied by her roommate. Her roommate states that he noticed her having an episode of rapid, alternating movements of her body. This event lasted for 15 minutes, started gradually, and involved thrusting movements of her pelvis. After discussing with the patient, she denies the loss of bowel and bladder function, postictal confusion, and tongue biting. During
the episode, she remembers tearfully crying and breathing rapidly. Past medical history is relevant for generalized anxiety disorder, and she states that she is currently stressed with her new job as a security guard. Family history is not significant, and she denies tobacco, alcohol, and illicit drug use. Physical examination reveals an HR of 90, RR of 12, BP 128/83, Temperature 96.4 degrees F. Her serum prolactin level is 18 ng/L (normal < 20 ng/ml) at the time of visit. Lacerations and fractures are not appreciated, and the remainder of her neurological examination does not reveal any abnormalities.

**Discussion**

Though the usual presentation of PNES is in the third decade of life, it can present at any age. Due to the lack of population-based studies, it is quite difficult to measure the incidence of PNES. Approximately 300,000 to 400,000 individuals suffer from PNES in the United States [1]. Prevalence of PNES ranges from two to thirty-three cases per 100,000 individuals [3]. According to Gates, 5-10% of PNES patients present to outpatient epilepsy clinics, and 20-40% present to inpatient epilepsy clinics [4]. The clinical features of a classic PNES include pelvic thrusting, opisthotonus, out-of-phase limb movements and side-to-side head movements. Prolonged duration of asynchronous motor activity, gradual onset, incomplete loss of consciousness, and lack of postictal confusion are some of the suggestive features [5].

The understanding of the PNES pathophysiology is still in the primitive stage. Complex interactions between genetic, environmental, and psychosocial factors could be the probable cause of seizure activity [6]. According to van der Krujis et al., emotional instability in PNES patients, whether expressed or unexpressed, triggers the motor symptomatology. In these patient’s, using Magnetic Resonance Imaging (MRI), they demonstrated high functional connectivity between the regions of the brain involved in emotion (insula), executive control (parietal cortex and inferior frontal gyrus) and movement (pre-central sulcus). This connectivity provides a correlation between neurology and physiology for the underlying psychiatric comorbidities and motor symptomatology [7].

Due to the heterogeneity of symptoms among the patients, diagnosing PNES is always a challenging entity. A comprehensive medical history along with diagnostic studies and clinical features of the witnessed event by a caregiver or by a clinician is paramount. Information regarding precipitants of seizure activity, frequency and duration of episodes, and factors which reduce seizure frequency must be obtained from the patients and
Eye witnesses. V-EEG (Video Electro Encephalography) remains the gold standard in diagnosis and is recommended in all patients suspected of PNES [8]. Usual findings are an absence of ictal EEG changes and presence of normal awake EEG rhythms before, during and after the event. An elevation in the serum prolactin levels is seen within 30 minutes after the seizure episode; however, lack of its increase doesn’t exclude the diagnosis of PNES [9]. Along with the rise in serum prolactin, elevation of other markers like serum cortisol, creatine phosphokinase, neuron-specific enolase, are seen in PNES and helps to differentiate it from epilepsy. A combined approach of electroclinical analysis and clinical pattern of the event helps in a definitive diagnosis. (Table 1)

Table (1): Diagnostic Studies for PNES

<table>
<thead>
<tr>
<th>Diagnostic Studies</th>
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<tr>
<td>Lab</td>
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<tr>
<td>• Absolute or relative increase of serum prolactin (which is twice the normal level) from blood drawn 10-20 min after seizure helps to differentiate epilepsy from PNES</td>
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<tr>
<td>• Serum elevation of cortisol, creatine phosphokinase, neuron specific enolase is seen with epilepsy and not in PNES</td>
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<tr>
<td>• Brain derived neurotrophic factor levels have been shown to be lower in patients with PNES.</td>
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<tr>
<td>Video EEG</td>
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<tr>
<td>• Gold standard of for diagnosis of PNES. It is recommended in all patients suspected of PNES. Usual findings are absence of ictal EEG changes and presence of normal awake EEG rhythms before, during and after the event.</td>
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<tr>
<td>Neurological Imaging</td>
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<tr>
<td>• MRI - Occasionally helpful and common change seen is mesial temporal sclerosis. Also, changes can be seen in cortical and cerebellar regions. MRI is not indicated for all patients unless focal neurological signs are evident.</td>
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</table>
PNES is initially managed with antiepileptic medications until the diagnosis is confirmed with v-EEG. After establishing the diagnosis, pharmacological therapy should be tailored towards the underlying psychiatric condition and withdrawal of antiepileptic drugs is highly recommended due to their dynamic adverse effects. These patients often require long-term psychiatric and neurological intervention. Education and support from a psychiatrist and neurologist are essential for treatment. Between 33-44% of patients remain seizure free six months post-diagnosis with appropriate education and therapeutic interventions [10]. Selective serotonin reuptake inhibitors (SSRIs), such as citalopram, escitalopram and, sertraline is the mainstay in the treatment of the major depressive disorder and generalized anxiety disorder. Treatment of post-traumatic stress disorder, conversion disorder, dissociative disorders, and borderline personality disorder requires the use of psychotherapy. Psychotherapeutic interventions have proven efficacious in reducing the frequency of seizures and improving measures of mental health in the PNES [11,12]. Cognitive behavioral therapy (CBT) plays a critical role along with the medications in the treatment of the major depressive disorder and generalized anxiety disorder. A recent randomized case-control study by Goldstein et al. demonstrated the efficacy of CBT in the treatment of PNES [13]. Compared to patients receiving standard medical care, patients receiving CBT demonstrated significantly lowered seizure activity post-twelve-week CBT treatment. Hence, a combination approach of psychotherapy and pharmacology is needed to improve the quality of life, function, recurrence of seizures in PNES patient population.

References


