

# A Case Study of Comorbid Psychogenic Non-Epileptic Seizures and Epileptic Seizures in an Adolescent Female with Depression

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## Abstract

Among patients with Psychogenic Non-Epileptic Seizures (PNES), about 10% to 30% have epilepsy, which brings some difficulties in diagnosis. Usually, psychogenic non-epileptic seizures and epileptic seizures occur independently at different points in time, and their performance is similar, but rarely seizures can occur several times in a row. This paper reports a case of adolescent depression in our hospital, who experienced psychogenic non-epileptic seizures followed by epileptic seizures.

**Keywords:** Epileptic seizures; Psychogenic non-epileptic seizures; Epilepsy.

## Case presentation

A 13-year-old female patient, currently attending junior high school, was admitted to our hospital on October 31, 2023, due to a two-year history of "poor mood" accompanied by limb twitching for two months. The patient first experienced low mood and irritability two years prior due to academic stress, presenting with decreased interest, withdrawal tendencies, auditory and visual hallucinations, along with headaches, palpitations, chest tightness, difficulty falling asleep, and easy awakenings. Additionally, she exhibited suicidal ideation and engaged in self-harming behaviors such as cutting and biting her hands. Two months before admission, she began experiencing episodic limb twitching, primarily affecting the upper limbs, with sudden onset and cessation lasting approximately 1-2 minutes, occurring several times a day. One month before admission, she was diagnosed with "depressive episode" at our outpatient clinic. Despite treatment with fluoxetine, risperidone, clonazepam, olanzapine, and thioridazine for over 20 days, her mood did not improve, and limb twitching persisted, leading to the decision for hospitalization. Additional medical history revealed that the patient was introverted and sensitive before the illness, with good academic performance and high self-expectations.

On mental examination, the patient appeared clear-minded but exhibited a distressed expression and passive interaction, displaying tension and unease upon contact. She demonstrated poor cooperation, providing brief responses with slow speech and reduced volume, lack of concentration, low mood with irritability, diminished interest, motivation, and confidence, alongside auditory and visual hallucinations. Cognitive processes were delayed, and she described feeling mentally sluggish with slowed movements, decreased energy, and easy fatigue. Suicidal ideation and self-harming behaviors were present, along with insomnia characterized by difficulty falling asleep and early awakening, with normal intelligence and full insight. Routine EEG on admission showed normal results, while polysomnography revealed frequent bursts of spike-slow wave complexes during NREM sleep. Subsequent extended video EEG demonstrated low-amplitude fast waves dominating bilateral hemispheres with asymmetrical slow wave activities during wakefulness. During seizure-free intervals, asymmetric epileptiform waves were observed bilaterally, predominantly anterior. During seizure events, hand tremors and eye blinking were noted, with simultaneous EEG changes indicative of non-epileptic seizures. However, abnormal clinical discharges were observed on wake EEG following two episodes of limb twitching, despite the

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absence of overt clinical symptoms during video EEG monitoring. According to the family, the patient experienced episodes of staring and unresponsiveness post-twitching, resembling absence seizures (and having experienced similar episodes outside the hospital). Psychological assessments revealed severe depression (82.5 on self-rating depression scale), anxiety (77.5 on self-rating anxiety scale), and overall poor mental health (SCL-90 score of 321, particularly high on hostility and other subscales). The diagnosis was severe depressive episode with psychotic symptoms, with a possibility of bipolar affective disorder given the patient's mood instability. Concurrent limb twitching was considered psychogenic non-epileptic seizures.

Treatment comprised fluoxetine 20 mg qd, sustained-release venlafaxine 150 mg qd, thioridazine 10 mg twice daily, quetiapine sustained-release tablets 150 mg qd, quetiapine tablets 100 mg at bedtime, and clonazepam 2 mg at bedtime to improve mood, cognition, stabilize psychiatric symptoms, aid sleep, and lamotrigine 25 mg qd as an antiepileptic, alongside suggestion therapy. Despite treatment, the patient reported improved but persistently low mood, daytime irritability, and self-harm impulses, along with nocturnal mood fluctuations and ongoing limb twitching. To address these symptoms further, medication doses were gradually increased to fluoxetine 40 mg qd, quetiapine tablets 200 mg at bedtime, sustained-release venlafaxine 150 mg twice daily, lamotrigine 50 mg qd, and alprazolam 0.2 mg twice daily as needed. After two weeks of treatment adjustment, the patient exhibited improved mood with reduced self-harm impulses and fewer episodes of limb twitching. No limb twitching occurred on the day before discharge. At one-month follow-up, the patient's mood remained stable, psychiatric symptoms including hallucinations had disappeared, she showed improved responsiveness to external stimuli, and limb twitching had not recurred, with no further reports of absence seizures by the family.

## Discussion

Psychogenic Non-Epileptic Seizures (PNES), also known as "pseudo-seizures" or "psychogenic epilepsy," manifest clinically similar to epileptic seizures but do not involve abnormal cortical discharges. Approximately 10% to 30% of PNES patients also suffer from epilepsy, making the diagnostic process relatively challenging [1]. Typically, PNES and epileptic seizures occur independently at different times, with successive occurrences of both being relatively rare [2].

The current understanding of the pathogenesis of epilepsy co-occurring with PNES mainly includes the following aspects: 1) Influence of emotionally unstable personality: Most susceptible individuals with PNES tend to exhibit features of emotionally unstable personality, preferring to express emotions through somatic symptoms [3]. Epileptic seizures may induce anxiety, depression, and other emotions in patients, thereby promoting the occurrence of PNES; 2) Epilepsy as a "symptom scaffold": Epilepsy itself may act as a "symptom scaffold" in patients, playing a role in the formation of repeated conditioned responses to stimuli. That is, on the basis of epileptic seizures, patients initiate abnormal preconscious activation of psychological representations, gradually solidifying this process through cognitive-emotional-behavioral progression into the subconscious [4]. 3) PNES as an alternative symptom of epilepsy: PNES may serve as an unconscious alternative symptom of epilepsy, whereby patients subconsciously benefit from epileptic seizures, such as receiving less attention and care from others after a decrease in seizure frequency [5].

In most cases, the occurrence of PNES in patients with epilepsy occurs after epileptic seizures. These patients may mimic the symptoms of epileptic seizures at the subconscious level to express internal, inexpressible emotions and concepts. Therefore, the clinical manifestations of PNES often resemble those of concomitant epileptic seizures [6]. Due to the similarity in clinical presentation, differentiation often requires the use of video Electroencephalography (EEG). Previous studies have indicated that patients diagnosed with psychogenic non-epileptic seizures may exhibit nonspecific abnormal discharges on EEG recordings during interictal periods [7]. Interestingly, in some patients diagnosed with PNES, subsequent invasive EEG examinations have captured epileptic waves [8]. This finding raises concerns about the possibility of misdiagnosis of some PNES cases, as the EEG detection devices used may not fully capture their underlying epileptic discharges.

Adolescence is a sensitive period for socio-psychological development, during which individuals undergo significant physiological, emotional, and social functional changes, making adolescents more susceptible to the influence of external environmental pressures. They are more prone to emotional disorders and depressive symptoms, which are also potential triggering factors for psychogenic non-epileptic seizures. In this case, the patient presents with two clinically distinct manifestations of PNES and epileptic seizures. Although we cannot completely rule out coincidence due to the higher frequency of epileptic seizures, supplementary medical history from the patient's family suggests that the patient often experiences epileptic seizures after PNES episodes outside the hospital. Therefore, we still need to consider the possibility of a certain association between these two conditions. Combined with the higher frequency of abnormal EEG discharges in the patient, which tends to occur after a PNES episode, we believe that in this case, PNES episodes may to some extent trigger epileptic seizures, or PNES may serve as a prodromal symptom of epileptic seizures. This temporal association may help us further understand the relationship between psychogenic non-epileptic seizures and epilepsy.

In future similar cases, we need to pay attention to the following aspects: 1) When the patient's EEG does not show abnormal discharges in the cerebral cortex during seizures, even if psychological treatment targeting PNES is conducted, attention should still be paid to other symptoms of the patient to avoid misdiagnosis; 2) When PNES symptoms alleviate, the patient's epileptic symptoms also alleviate. This may indicate that PNES can act as a triggering factor for epileptic seizures because the patient's limb movements and the resulting emotional tension during PNES episodes may serve as stimuli for epileptic seizures; 3) Despite the video EEG showing numerous subcortical abnormal discharges, other brain disorders cannot be ruled out, thus further examinations such as cranial MRI are recommended; 4) We emphasize the importance of video EEG, especially in distinguishing psychogenic non-epileptic seizures from epileptic seizures. However, we also need to be aware of the limitations of technology; sometimes, EEG may not fully capture the patient's epileptic discharges. Therefore, when interpreting test results, physicians need to be cautious and consider multiple aspects of information, including clinical symptoms, other auxiliary examinations, and patient history, to arrive at an accurate diagnosis.

## Declarations

**Conflict of interest:** The authors declare that the research was conducted in the absence of any commercial or financial

relationships that could be construed as a potential conflict of interest.

**Ethics statement:** In this study involving human subjects, ethical standards from the 1964 Declaration of Helsinki were followed. Informed consent was obtained from the participant for publication, with privacy safeguards. Ethical approval was granted by an Institutional Review Board, ensuring compliance with ethical guidelines and participant rights protection. This study did not involve animal subjects.

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**Data availability statement:** The data generated during this study are not publicly available due to privacy concerns but are available from the corresponding author upon reasonable request and subject to ethical approval. This research respects participant confidentiality and anonymity, with personal data redacted for privacy protection. Methodological details are provided in the manuscript for study replication purposes. Data access requests will be considered in line with ethical standards and participant rights.

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