Co-occurring psychogenic nonepileptic seizures and possible true seizures

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Psychogenic nonepileptic seizures (PNES) are a physical manifestation of a psychological disturbance. They are characterized by episodes of altered subjective experience and movements that can resemble epilepsy, syncope, or other paroxysmal disorders, but are not caused by neuronal hypersynchronization or other epileptic semiology.[1] Asynchronous movements, closed eyes, crying, stuttering, side-to-side head movement, and pelvic thrusting may be observed, all of which are atypical of epileptic seizures.[1] PNES, a syndrome of "pseudo-seizures," is recognized in 11% of convulsive seizure cases presenting to the emergency department (ED).[2] PNES can co-occur with epilepsy; in 2 population-based studies, the pooled rate of EEG-confirmed comorbid epilepsy in PNES was 14%.[3]

Patients with PNES may present to multiple clinicians and hospitals for assessment. Access to outside hospital records can be limited, which can lead to redundant testing and increased health care costs and burden. Additionally, repeat presentations can increase stigmatization of the patient and delay or prevent appropriate therapeutic management, which might exacerbate a patient's underlying psychiatric condition and could be dangerous in a patient with a co-occurring true seizure disorder. Though obtaining and reviewing external medical records can be cumbersome, doing so may prevent unnecessary testing, guide medical treatment, and strengthen the patient-doctor therapeutic alliance.

In this article, I discuss our treatment team's management of a patient with PNES who, based on our careful review of records from previous hospitalizations, may have had a co-occurring true seizure disorder.

Case report

Ms. M, age 31, has a medical history of anxiety, depression, first-degree atrioventricular block, type 2 diabetes, and PNES. She presented to the ED with witnessed seizure activity at home.

According to collateral information, earlier that day Ms. M said she felt like she was seizing and began mumbling, but returned to baseline within a few minutes. Later, she demonstrated intermittent upper and lower extremity shaking for more than 1 hour. At one point, Ms. M appeared to be not breathing. However, upon initiation of chest compressions, she began gasping for air and immediately returned to baseline.

In the ED, Ms. M demonstrated multiple seizure-like episodes every 5 minutes, each lasting 5 to 10 seconds. These episodes were described as thrashing of the bilateral limbs and head crossing midline with eyes closed. No urinary incontinence or tongue biting was observed. Following each episode, Ms. M was unresponsive to verbal or tactile

stimuli but intermittently opened her eyes. Laboratory test results were notable for an elevated serum lactate and positive for cannabinoids on urine drug screen.

Ms. M expressed frustration when told that her seizures were psychogenic. She was adamant that she had a true seizure disorder, demanded testing, and threatened to leave against medical advice without it. She said her brother had epilepsy, and thus she knew how seizures present. The interview was complicated by Ms. M's mistrust and Cluster B personality disorder traits, such as splitting staff into "good and bad." Ultimately, she was able to be reassured and did not leave the hospital.

The treatment team reviewed external records from 2 hospitals, Hospital A and Hospital B. These records showed well-documented inpatient and outpatient Psychiatry and Neurology diagnoses of PNES and other conversion disorders. Her medications included 2 anticonvulsants—topiramate 200 mg every 12 hours and oxcarbazepine 300 mg every 12 hours—as well as clonazepam 0.5 mg as needed for seizures and anxiety.

Ms. M's first lifetime documented seizure occurred in May 2020, when she woke up with tongue biting, extremity shaking (laterality was unclear), and urinary incontinence followed by fatigue. She did not go to the hospital after this first episode. In June 2020, she presented and was admitted to Hospital A after similar seizure-like activity. While admitted and monitored on continuous EEG (cEEG), she had numerous events consistent with a nonepileptic etiology without a postictal state. A brain MRI was unremarkable, and Ms. M was diagnosed with PNES.

She presented to Hospital B in October 2020 reporting seizure-like activity. Hospital B reviewed Hospital A's brain MRI and found right temporal lobe cortical dysplasia that was not noted in Hospital A's MRI read. Ms. M again underwent cEEG while at Hospital B and had 2 recorded nonepileptic events. Interestingly, the cEEG demonstrated right temporal spikes that were consistent with the dysplasia location on the MRI. Ms. M was discharged and instructed to keep a seizure journal until outpatient follow-up.

Ms. M documented 3 seizure-like events between October and December 2020. She documented activity with and without full-body convulsions, some with laterality, some with loss of consciousness, and some preceded by an aura of impending doom. Ms. M was referred to psychotherapy and instructed to continue topiramate 100 mg every 12 hours for seizure prophylaxis.

Ms. M presented to Hospital B again in March 2022 reporting seizure-like activity. A brain MRI found cortical dysplasia in the right temporal lobe, consistent with the MRI at Hospital A in June 2020. cEEG was also repeated at Hospital B and was unremarkable. Oxcarbazepine 300 mg every 12 hours was added to Ms. M's medications.

Ultimately, based on an external record review, our team (at Hospital C) concluded Ms. M had a possible true seizure co-occurrence with PNES. To avoid redundant testing, we did not repeat imaging or cEEG. Instead, we increased the patient's oxcarbazepine to 450 mg every 12 hours, for both its effectiveness in temporal seizures and its mood-stabilizing properties. Moreover, in collecting our own data to draw a conclusion by a thorough record review, we gained Ms. M's trust and strengthened the therapeutic alliance. She was agreeable to forgo more testing and continue outpatient follow-up with our hospital's Neurology team.

Take-home points

Although PNES and true seizure disorder may not frequently co-occur, this case highlights the importance of clinician due diligence when evaluating a potential psychogenic illness, both for patient safety and clinician liability. By trusting our patients and drawing our own data-based conclusions, we can cultivate a safer and more

Clinical Point

In 2 large studies, the prevalence of comorbid PNES and EEG-confirmed epilepsy was 14%

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satisfactory patient-clinician experience in the context of psychosomatic disorders.

Clinical Point

By conducting a thorough record review, we gained Ms. M's trust and strengthened the therapeutic alliance

References

1. Bajestan SN, LaFrance WC Jr. Clinical approaches to psychogenic nonepileptic seizures. Focus (Am Psychiatr Publ). 2016;14(4):422-431. doi:10.1176/appi.focus.20160020
2. Dickson JM, Dudhill H, Shewan J, et al. Cross-sectional study of the hospital management of adult patients with a suspected seizure (EPIC2). BMJ Open. 2017;7(7):e015696. doi:10.1136/bmjopen-2016-015696
3. Kutlubaev MA, Xu Y, Hackett ML, et al. Dual diagnosis of epilepsy and psychogenic nonepileptic seizures: systematic review and meta-analysis of frequency, correlates, and outcomes. Epilepsy Behav. 2018;89:70-78. doi:10.1016/j.yebeh.2018.10.010