Psychogenic Nonepileptic Attacks

29

William O. Tatum

Case Presentation

A 45-year-old right-handed female nurse had mitral valve prolapse, fibromyalgia, obstructive sleep apnea, and depression and was being evaluated by neurology for "brain fog." She previously underwent hospitalization for a suicide attempt following a rape when she was 20 years old. She was, otherwise in her state of usual health until she was confronted by her husband with the fact that he had been having an affair with her best friend and wanted a divorce. She did not have a history of seizures and had no early risk factors for epilepsy. She did have a minor closed head injury and brief loss of consciousness when she fell off a skateboard at 9 years old but was otherwise normal. She was distressed about the realization that her life was about to change. Two weeks after receiving the information from her husband, she was involved in a minor car accident. She had accidentally driven over a curb at slow speeds in a grocery store parking lot and hit a tree. She did not hit her head, recalled the accident, and did not lose consciousness. Later that day, she was watching TV with a friend having just attempted to talk about the divorce when suddenly her friend witnessed the patient fall over on the couch, eyes roll backwards, before she "shook all over". She was unresponsive during the event which lasted for "5–10 minutes" and was tired and sore all over her body after the event. Her friend dialed 911, and she was transported to the local emergency department where she was loaded with levetiracetam (LEV). A CT brain, a 12-lead EKG, and electrolytes were normal. Recurrent episodes occurred within the week, and she was seen by a neurologist who increased the levetiracetam from 500 mg twice daily to 1000 mg twice daily. An

W. O. Tatum (✉)
Mayo Clinic, Department of Neurology, Jacksonville, FL, USA
e-mail: tatum.william@mayo.edu

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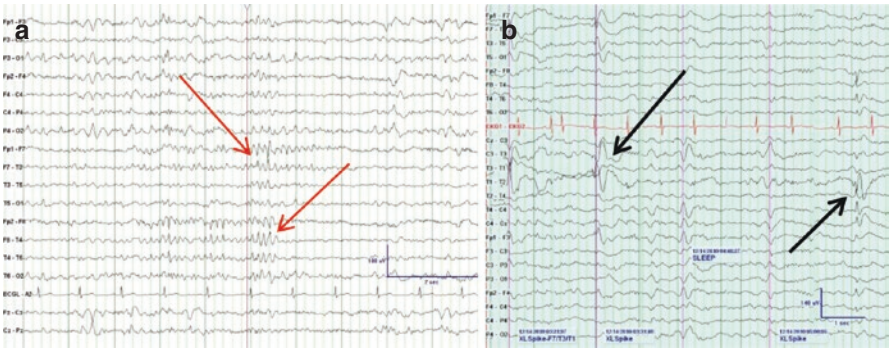

Fig. 29.1 (**a**) EEG demonstrating the patient's "abnormality" of bilateral wicket spikes which are a benign variant of uncertain significance unrelated to epilepsy (*red arrows*). Compare the patient's EEG in (**a**) with another EEG taken from a patient with focal epilpepsy (**b**) revealing pathological bilateral independent spike-and-slow wave discharges in the temporal regions (*black arrows*). Note the differences between the EEGs shown demonstrating bitemporal bursts of wicket spikes in (**a**) and isolated spike-and-slow waves in (**b**)

EEG was performed in the office and was abnormal due to "temporal spikes that are potentially epileptogenic" (Fig. 29.1). On LEV, the episodes continued, but she became very depressed and contemplated "not wanting to live." When she was evaluated at Mayo Clinic, her father provided an outpatient smart phone video of one of the events that was clearly a nonepileptic event.

Clinical Questions

1. What does the clinical history suggest as a diagnosis?
2. What are the EEG findings that help support the diagnosis?
3. What is the prevalence of episodes that mimic epileptic seizures?
4. What is the approach to treatment for patients with psychogenic nonepileptic attacks (PNEA)?
5. What is the overall prognosis for remission of in people with PNEA?

Diagnostic Discussion

1. The history of paroxysmal events with loss of consciousness and post-event fatigue is suspicious for the diagnosis of epilepsy especially when they are associated with an abnormal epileptiform EEG. However, the history of depression, fibromyalgia, and mitral valve prolapse have been associated with psychological conditions including anxiety and disordered mood. Minor head injuries without loss of consciousness are a minimal risk factor for post-traumatic epilepsy in the context of this patient's clinical history. The episodes themselves are frequent and daily without any response to antiseizure medication. The principal difference between PNEA and epileptic seizures (ES) are listed in the Table 29.1

Table 29.1 The clinical features of psychogenic nonepileptic attacks and epilepsy

Feature	PNEA	Epilepsy
Age involved	15–35 years	All ages
Seizure onset	Gradual	Abrupt
Population	80% female	Male = female
Semiology	Non-stereotyped Head side to side Eyes closed Limbs out of phase Opisthotonus On and off Variable	Stereotyped Head fixed unilateral Eyes open Limb in phase Body straight Evolution Stereotyped
Duration	Prolonged >2–3 min	Usually <1–2 min
Post-ictal	Rare and variable	Yes (confused)
Injury	Infrequent usually mild	Tongue biting and injury/burns
Suggestion	Reliable	Rare
EEG	Normal	Abnormal
Witnessed	Usually	Not always

2. While the sensitivity of an EEG in patients with epilepsy is low to moderate, the specificity of an epileptiform EEG in patients with epilepsy is high. Only about 1–2% of adult patients with abnormal interictal epileptiform discharges do not have clinical evidence of epilepsy. However, there are many variations of normal and benign variants of uncertain significance that may mimic pathological epileptiform discharges leading to misdiagnosis from a misinterpreted EEG. In this case, the EEG contained wicket waves that were present and misinterpreted as "abnormal." These benign waveforms are the most common misinterpreted "normal" variant [2]. They consist of intermittent bursts or sporadic monophasic arciform "spiky" waveforms located in the temporal regions maximal during drowsiness that may be mistaken for an abnormal temporal epileptiform discharge. However, wickets are typically not associated with after-going slow waves, do not distort the background activity, and have a similar frequency within the bursts though they may be bilaterally independent and asyncrhonoous similar to patients with epilepsy yet are benign.
3. Approximately 20–30% of patients (range 10–50%) will have a different diagnosis other than epilepsy when evaluated for spells with video-EEG monitoring [3, 4]. Video-EEG monitoring is the gold standard for the diagnosis of PNEA [1–4]. Ninety percent of these nonepileptic events will be due to a psychogenic cause [3]. Induction or activation using a placebo has been used to demonstrate suggestibility associated with psychogenic etiologies. Ten percent of the time physiologic episodes (predominately syncope) will be present [1, 3]. The diagnosis hinges on recording the typical event with a normal or unchanged "ictal" EEG. When consciousness is impaired, the EEG will exhibit abnormal electrocerebral activity that reflects loss of cerebral blood flow with slowing and attenuation of the background rhythms to the end point of a "flat" recording. Convulsive syncope is a common physiologic nonepileptic event manifest as multifocal myoclonic jerks or less often tonic stiffening that can mimic epileptic seizures.

4. The treatment of psychogenic nonepileptic attacks begins with the delivery of the diagnosis. Patient reactions to accepting a psychological cause portend the response to follow through with recommendations and the overall benefit of realizing the diagnosis. It is important to present the diagnosis with a strong and positive attitude to introduce the new diagnosis. PNEA reflects a conversion disorder with seizures that are mimics of epileptic seizures in people with epilepsy. Many patients are "disabled" by their "seizures" given their unpredictability and lack of ability to control them [5, 6]. Pseudostatus epilepticus may occur in approximately one-third of individuals. Identifying a mental health practitioner experienced in the treatment of PNEA to providing a bridge between neurology and psychiatric management. Antidepressants such as the selective serotonin reuptake inhibitors are helpful for depression but may have less impact on resolution of the episodic behavior. Cognitive behavioral therapy tailored to the individual needs by psychology is a treatment of choice that may lead to reduction of the events [6].
5. The prognosis for patients with PNEA is variable. Children have a much better likelihood of remission than adults. Like adolescents they have different stressors than adults and may have their episodes for a shorter period of time. Most PNEA diagnoses are delayed by 1–7 years from onset. Overall prognosis for remission of in people with the attacks is limited by the relatively limited number of people following through and completing treatment with Psychiatry and/or Psychology as many patients are lost to follow-up or do not attend sessions to undergo cognitive behavioral therapy. Long-term outcomes suggest that after years of PNEA, nearly 50% of patients continue to have the attacks despite a definitive diagnosis [5]. Many patients are not working and are reliant on social security disability. Outcome may be better for those people with higher levels of education, less severe motor involvement (one-third of patients), and short times to diagnosis in addition to those individuals with limited somatoform complaints and those who lack significant psychiatric diagnoses [5].

Pearls of Wisdom

1. PNEA is most common in women. They account for approximately 20–30% of patient hospitalizations for diagnostic video-EEG evaluation of uncontrolled paroxysmal attacks. Notable delays (years) are often present prior to arriving at a definitive diagnosis.
2. Abnormal EEGs should be reviewed when the clinical diagnosis of epilepsy is suspect to ensure over-interpretation of an abnormality is absent.
3. When waveforms on EEG are reviewed in patients with PNEA, variations of normal features in the EEG and benign variants of uncertain significance account for majority of misinterpreted EEGs that resulted in treatment.

4. The attacks in patients with PNEA represent a conversion disorder, and a history of abuse is common. Treatment with antiseizure medication is ineffective and should be discontinue when no psychiatric or neurological comorbidity is present. No psychological "intent" exists in people with PNEA, and patients do not "fake" seizure-like activity in the vast majority of cases. The opportunity for remission starts at delivery of the diagnosis though is guarded for long-term remission.
5. Always screen for psychiatric comorbidity in people with PNEA to identify a primary diagnosis and stressors with the purpose of considering medical treatment. An Axis 1 diagnosis is commonly anxiety-depression in patients with PNEA yet it is also a frequent comorbidity in patients with epilepsy. Cognitive behavioral therapy in conjunction with medication used to treat a comorbid psychological disorder can improve daily function and may lead to reduction in the attacks when the patient accepts the diagnosis of PNEA and complies with treatment recommendations.

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