Epileptiform Discharges During Slow Wave Sleep on Polysomnogram

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This is a case of a 30-year-old right-handed African American man who was referred for evaluation of abnormal nocturnal behaviors. The patient developed paroxysmal nocturnal attacks as a teenager, which consisted of tongue movements, swallowing, and gasping a few times each month. On rare occasions, the patient would sit up in bed, call out loudly to his bed partner, and exhibit tonic posturing of the left upper extremity. Most events last less than one minute with the patient quickly returning to sleep and having no recollection the next morning. There is no history of either witnessed snoring or witnessed apnea in the patient.

Prenatal and perinatal histories were normal. The patient denied febrile seizures as a child or meningitis. Development milestones were unremarkable. In his teenage years, the patient noted an insidiously progressive left upper extremity pain with weakness that was more severe in the morning and has progressed to fixed weakness.

There is no family history of seizures. Social history is negative for illicit drug abuse, alcohol, or smoking. The patient is in the final year of an engineering program, earning B-C grades. He is engaged and lives with his fiancé.

Neuropsychological examination revealed mild cognitive, memory, verbal, and visual deficits. Strength was 4/5 in left shoulder abduction, left elbow flexion, left elbow extension, left wrist extension, and left hand grip. Reflexes were brisk in the left biceps tendon. The remainder of the physical and neurological examination was normal; and Mallampati score was 2

Magnetic resonance imaging (MRI) of the brain with and without gadolinium contrast was normal. MRI of the left shoulder and brachial plexus showed no plexus inflammation and no joint abnormalities. Nerve conduction studies and concentric needle electromyography showed no electrophysiologic evidence for a left brachial plexopathy, left cervical radiculopathy, or left upper extremity neuropathy.

A diagnostic polysomnogram (PSG) with 16-channel EEG was performed while on extended release formulation levetiracetam 1,000 mg twice per day. The patient was on no other medications. A sample from the EEG is displayed in **Figure 1**.

QUESTION: What is the patient's diagnosis?

Figure 1—Nocturnal epileptiform discharges.

(A) 30-second epoch with 16-channel bipolar longitudinal electroencephalogram (EEG) montage showing epileptiform discharges during slow wave sleep. Time base: 20 mm/sec. Sensitivity: 7 μ V/mm. (B) 14-second segment with 19-channel contralateral ear referential EEG montage. Time base: 30 mm/sec. Sensitivity: 7 μ V/mm. *Individual epileptiform discharges.

ANSWER: Nocturnal fronto-temporal epilepsy.**DISCUSSION**

The PSG showed a normal background during wakefulness. During N1 and N2, occasional right frontal-temporal derivation spikes were noted. Upon entering slow wave sleep (SWS) the frequency of right frontal-temporal spikes, poly-spikes, and slow 1.5-2.5 Hz spike-and-wave epileptiform discharges dramatically increased with an average epileptiform discharge frequency per second of SWS of 0.15 (4.59 epileptiform discharges per epoch). Video monitoring did not reveal any abnormal movements.

In the International 10-20 system, the F7 and F8 electrodes lie over the inferior frontal cortex, but most often record anterior temporal activity and are often referred to as the *fronto-temporal* electrodes. One way to determine the source of their activity is to evaluate involvement of adjacent electrodes. For example, if T4 is involved, F8 is likely recording anterior temporal activity; if F4 is involved, F8 is more likely to be recording inferior frontal activity. The field on our patient's contralateral ear average reference montage includes both F4 and T4, indicating a right hemispheric focus extending from the inferior frontal cortex to the middle temporal cortex.

While it is well known that frontal lobe epilepsy may manifest with complex partial seizures during sleep, the occurrence of nocturnal complex partial seizures in temporal lobe epilepsy has received less attention. Simple partial seizures are common in nocturnal temporal lobe epilepsy (NTLE); they consist of psychic (e.g., fear, panic, déjà vu), sensory (e.g., gustatory/olfactory hallucinations, vertigo), or autonomic (e.g., epigastric sensations, nausea, tachycardia) phenomena and often wake the patient from sleep.¹ Compared to frontal lobe epilepsy, seizures in NTLE are less frequent, not clustered, and tend to lack prominent motor activity unless the seizures become generalized. Similar to frontal lobe epilepsy, post-ictal confusion or amnesia is either absent or brief (**Table 1**).

The inter-ictal right fronto-temporal epileptiform discharges support a diagnosis of localization related epilepsy. The history of left upper extremity tonic posturing during sleep is also suggestive of a right hemispheric focus. History of orolimentary automatisms consisting of tongue movements with swallowing suggests mesial temporal lobe involvement. Our patient's infrequent non-clustered seizures with absence of family history of epilepsy are consistent with prior descriptions of NTLE.

The dramatic increase in epileptiform discharges during SWS suggests electrical status epilepticus of sleep (ESES). ESES is characterized by marked potentiation of epileptiform activity during sleep with near continuous slow spikes and waves during SWS. Initial definitions of ESES proposed that slow spikes and waves should occupy no less than 85% of total SWS; however, the definition of ESES by the International League Against Epilepsy (ILEA) does not include a specific cutoff percentage or an exact method to measure the spike wave index.² As a result, it is currently undefined how many epileptiform charges per epoch are required to meet criteria for ESES.

Table 1—Temporal and frontal lobe seizures: semiological distinctions

Features	Temporal Lobe	Frontal Lobe
Seizure frequency	Less frequent	Frequent, often daily
Seizure duration	Longer	Brief
Complex posture	Less frequent	Frequent and prominent
Prominent motor activity	Rare	Common
Vocalization	Speech (non-dominant)	Loud, non-speech (grunt, scream, moan)
Post-ictal confusion/amnesia	Long duration	Short duration

The EEG pattern of ESES appears in, but is not exclusive to, the following three childhood epilepsy syndromes: continuous spike and wave in sleep (CSWS), an age related epileptic encephalopathy with global regression; Landau-Kleffner Syndrome, an age related epileptic encephalopathy with mainly language regression; and benign epilepsy of childhood with centro-temporal spikes (BECTS).

In children with CSWS, no significant electroclinical differences were noted between children with > 85% of SWS containing epileptiform discharges and children with < 85% of SWS containing epileptiform discharges.³ In adults, ESES appears to be less common, with only three adult cases reported in the literature.⁴⁻⁶ In each case > 85% of SWS was occupied by epileptiform discharges, but the exact method of measurement is not described. Unilateral hemiparesis has been described in patients with ESES in the setting of abnormal MRI findings.⁷

When measuring epileptiform discharges per second of SWS in children with CSWS, the frequency of epileptiform discharges evolves over time from an acute stage with 1-2 spikes per second of SWS to a residual stage with 0-2.4 spikes per second of SWS.⁸ Levetiracetam has been reported effective in reducing the spike and wave frequency during SWS in patients with ESES.⁹

Though epileptiform discharges were noted in each epoch of SWS during our patient's two PSGs, the frequency of epileptiform discharges per second of SWS was smaller than frequencies described in children with CSWS. This may be in part due both to our patient's age, and partial resolution of the epileptiform discharges with levetiracetam. In addition, our patient's history and neurological examination were not consistent with the neurological syndromes typically associated with ESES.

In patients with varying clinical presentations of ESES, nocturnal oral diazepam has demonstrated efficacy in reducing mean epileptiform activity.¹⁰ Diazepam 2 mg at bedtime was initiated, and a repeat PSG one month later showed a decrease in the average number of epileptiform discharges per second of SWS to 0.05 (2.48 epileptiform discharges per epoch). The patient reported an overall improvement in nocturnal sleep quality while on diazepam with no nocturnal events reported.

SLEEP MEDICINE PEARLS

1. The source of activity on F7/F8 electrodes is determined by evaluating involvement in adjacent electrodes

2. Nocturnal complex partial seizures may be seen in both frontal lobe epilepsy and temporal lobe epilepsy.
3. Electrical status epilepticus of sleep (ESES) is characterized by marked sleep potentiation of epileptiform activity during SWS. Specific cutoff thresholds or measurement methods for epileptiform activity frequency have yet to be defined.

CITATION

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