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Cataplexy versus pseudoseizure: a case study

Abstract

Cataplexy is frequently misdiagnosed as pseudoseizures. This case study about a young patient shows the necessary steps needed to accurately diagnose and treat cataplexy as well as understand the differences between narcolepsy Type I and Type II.

Keywords

cataplexy, narcolepsy, pseudoseizure

History

The patient is a seventeen-year-old female with a past medical history of depression. She was referred to the sleep center by a neurologist for seizures. She has also been followed by her pediatrician for the past two years where she was first given a diagnosis of seizure versus pseudoseizure. The patient's symptoms started around age fifteen when her family noticed a pattern of her falling to the ground frequently during highly emotional situations.

Clinical Findings

A physical exam of the patient revealed a morbidly obese female with a BMI of 54, blood pressure of 146/90, pulse of 93, height of 5 foot 4 inches, and weight of 314 pounds. Oxygen saturation was 100% on room air. Otherwise, her physical exam was unremarkable except for a Mehta Patti score of 2. The Epworth sleepiness score was 21. Her medications included: Wellbutrin SR 100 mg once a day, Yasmin 28 once a day, and Synthroid 50 mcg once a day. Other laboratory data were unremarkable.

Her family physician suspected a seizure disorder and ordered an electroencephalogram (EEG), which was negative. She was then diagnosed with pseudoseizure due to her significant anxiety and ongoing depression. She continued taking several antidepressants (Venlafaxine XR and Sertraline), which failed to alleviate her symptoms.

She was next evaluated by the neurologist. The patient was losing muscle tone in her legs when laughing, crying, or otherwise showing emotions. She denied jerking movements in the upper or lower extremities, and there was no postictal confusion. It was obvious that the patient was also extremely sleepy; her family attributed her sleepiness and fatigue to depression. She was falling asleep as a passenger in the car and at school during class.

When evaluated at the sleep lab, the patient was noted to have poor sleep hygiene. She went to bed around midnight and awoke around 6 AM, but she took frequent naps during the day. She denied restless legs, though sleep during the night was restless. She did not feel refreshed upon awakening in the morning. Naps of two to three hours at least once a day, however, were very refreshing. She denied sleep paralysis, but she reported vivid dreams. She described her falling episodes as "knee-buckling" with a total loss of muscle tone in the lower extremities only in emotional situations, such as when feeling nervous, crying, or laughing. These falling episodes

were not associated with a loss of consciousness. She denied hallucinations and reported occasional snoring. She was depressed but denied suicidal ideation; she felt embarrassed when these falling episodes happened. The culmination of these symptoms was highly suspicious of cataplexy.

Investigative Findings

Narcolepsy was suspected based on the DSM-V criteria and the presence of cataplexy. To rule out obstructive sleep apnea based on the extreme obesity and presence of snoring, a sleep study was performed after cessation of Wellbutrin for two weeks. The polysomnogram (PSG) revealed a total sleep time of 480 minutes with a somewhat decreased sleep efficiency of 80% with REM latency of 61 minutes. Her AHI was 2.9 and snoring was mild with no significant leg movements. The hypnogram revealed sleep fragmentation and frequent arousals with poor sleep efficiency (Figure 1).

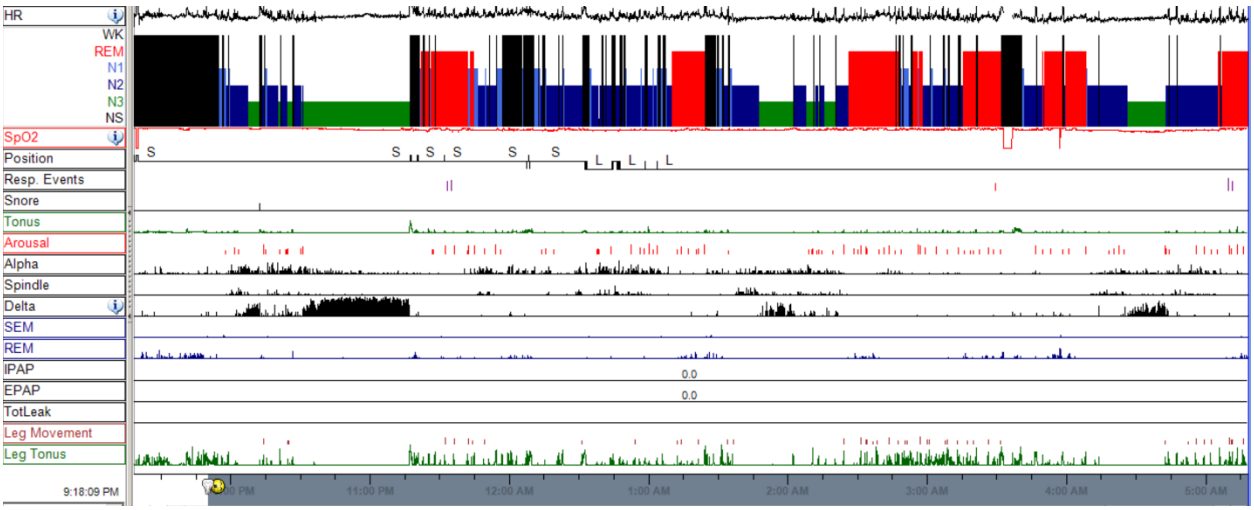
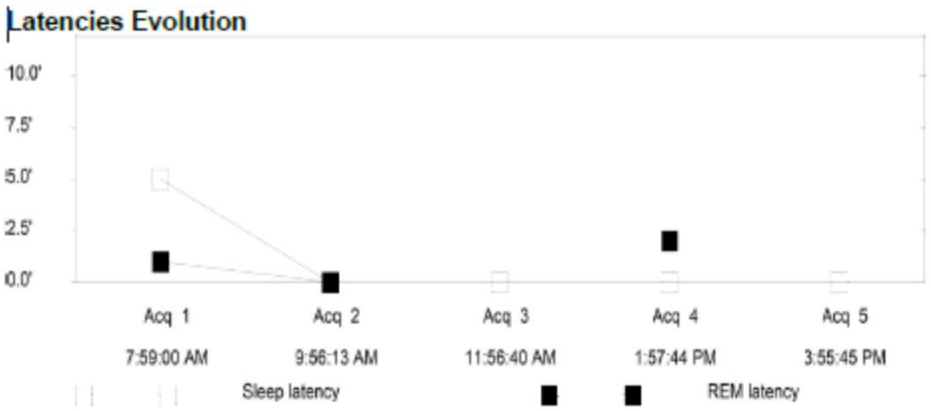


Figure 1. The hypnogram shows the sleep stages, respiratory events, oxygen saturation, sleep waves, snoring, and leg events. The sleep stages in blue (N2), green (N3), and red (REM) are interrupted by frequent arousals in black causing frequent sleep fragmentation.

Additional testing involved a multiple sleep latency test (MSLT). In this diagnostic test for narcolepsy, the patient is given the chance to take five 20-minute naps. This test is used to measure the time to reach the sleep stage for each nap (sleep latency), and if REM sleep is reached within 15 minutes of sleep, then it is considered a sleep-onset REM (SOREM). Mean sleep latency was reduced to 1 minute and the patient went into REM sleep in 3 out of the 5 naps (Figure 2). Furthermore, the patient's drug screen was negative; a diagnosis of narcolepsy was established.



8:02:30 AM	8:08:00 AM	00:05:30	00:01:00
9:59:13 AM	9:59:13 AM	00:00:00	00:00:00
11:59:40 AM	11:59:40 AM	00:00:00	/
2:01:14 PM	2:01:44 PM	00:00:30	00:02:00
3:59:15 PM	3:59:15 PM	00:00:00	/

LATENCY DATA	
Mean Sleep Latency (5 values):	00:01:12
Average REM Latency (3 values):	00:01:00

Figure 2. This MSLT summary shows sleep latencies and REM latencies and confirms the presence of 3 REM sleep onset events (SOREM).

The patient was treated with Adderall XR 10 mg daily, which was increased to 20 mg after 1 week due to ineffectiveness. Her hypersomnia significantly improved after the increased dosage of Adderall XR and her Epworth Score decreased to 9. She continued to experience cataplexy symptoms, and thus, Effexor X 37.5mg was added at bedtime. With the addition of this medication, the cataplexy symptoms completely resolved, and school performance significantly improved. Her three-month follow-up appointment was remarkable for complete resolution of her cataplexy and daytime sleepiness; her self-reported depression symptoms also significantly improved.

Discussion: the significance of proper diagnosis of cataplexy

Narcolepsy is a common sleep disorder associated with extreme daytime sleepiness that affects 1 in 2,000 people.[1,2] Narcolepsy commonly starts in childhood, although diagnosis is commonly missed and delayed by at least ten years. Excessive daytime sleepiness is always present, but the other symptoms, including sleep paralysis, hallucinations, cataplexy, and sleep paralysis, are not always present. Cataplexy is found in 60-70% of patients with narcolepsy. [2,3] Cataplexy causes muscle weakness, which occurs during REM sleep, during the waking hours, and in association with a form of emotional expression. This sudden loss of muscle tone may also initially develop

as experiencing slight jaw drop to slight or significant weakness in the arms and legs where the person can no longer stand.

There are two types of narcolepsy. Narcolepsy type I is defined as narcolepsy with cataplexy, and Narcolepsy type II is defined as narcolepsy without cataplexy.[2,4] The etiology of narcolepsy is not well known, but nearly all patients with Narcolepsy type I have low or absent hypocretin, which is a hormone responsible for wakefulness and regulating REM sleep. An autoimmune etiology has been suggested, even though the disease is not genetic, but it does cluster in families.

The diagnosis of narcolepsy is based on the results of a PSG and a multiple sleep latency test (MSLT). A negative PSG rules out sleep apnea. To confirm a diagnosis of narcolepsy, the MSLT, which assesses the daytime sleepiness by allowing patients to take five naps the following day, shows short (<8 min) sleep latency and occurrence of REM within 15 min of sleep onset in at least two of the five naps during the MSLT. If the patient experiences REM sleep onset within 15 minutes from sleep onset during the PSG, this episode may be counted as one of the MSLT SOREM episodes. Once two episodes of SOREM are confirmed, the patient is diagnosed with narcolepsy. Cataplexy is confirmed by witnessed episodes of the patient and family. Measuring hypocretin levels in cerebral spinal fluid is confirmatory diagnostic, but not commonly performed.

Genetic tests are still in the early stages. We know that patients who have the genetic marker HLA –DQB1*06:02 are at increased risk for narcolepsy and especially Narcolepsy type I.[4] However, testing for this genetic marker is not proven yet to help establish a narcolepsy diagnosis.

Treatment options include stimulants such as modafinil and amphetamines.[4,5] Newly approved stimulants in 2019, including a histamine-3 receptor agonist, Pitolisant, and a dopamine/norepinephrine reuptake inhibitor Solriamfetol, are also effective treatment options. Sodium oxybate, which is usually administered twice a night, is approved for the treatment of narcolepsy and cataplexy. In addition, antidepressants such as selective serotonin reuptake inhibitors (SSRIs) are commonly administered to treat the cataplexy symptoms. Due to the delay in the narcolepsy diagnosis, patients with narcolepsy frequently experience depression. Therefore, addressing that issue is essential to improve the quality of life. It is not uncommon for cataplexy to be confused with conversion disorder, due to the association of cataplexy with emotional situations. Finally, patients must learn that lifestyle modifications are important such as taking daily naps.

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