

CLINICAL VIGNETTE

Recurrent Isolated Sleep Paralysis

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Introduction

Recurrent Isolated Sleep Paralysis is a transient condition where involuntary body paralysis (with the exception of ocular and respiratory muscles) is experienced with full consciousness at either sleep onset or upon awakening.¹ Sleep paralysis can occur as a symptom of sleep disorders such as narcolepsy, idiopathic hypersomnia, or obstructive sleep apnea. However, in recurrent isolated sleep paralysis, there is no primary sleep disorder, and episodes occur repeatedly. The phenomenon is characterized by persistent muscle atonia in the wake state which is normally only seen in rapid eye movement (REM) sleep. The prevalence of at least one episode of sleep paralysis is thought to be 15% to 40%, with higher frequencies observed in students and psychiatric patients.^{1,2} In 25% to 75% of sleep paralysis episodes, hallucinatory phenomenon can be present.³ These hallucinations can include dark shadows, demons, a sense of suffocation or chest heaviness.

Case Presentation

An 18-year-old female with no significant past medical history presents with several years of excessive daytime sleepiness and frequent episodes of sleep paralysis. In college, it occurred intermittently. However, in recent months she reports several episodes each night. During these episodes she intentionally wakes herself up, knowing that if she lets herself drift back to sleep the episode will recur.

The patient works night shifts four days each week. She typically sleeps 8-9 hours on non-work nights. After working a night shift, she sleeps 6-7 hours and requires a nap 1-3 times a week to compensate. She otherwise intentionally does not nap, to avoid episodes of sleep paralysis. She takes no chronic medications and does not drink alcohol or caffeinated beverages. Exam included normal vital signs, BMI 19. Epworth Sleepiness Scale was 9/24, and STOP BANG scored 1/8.

After her initial clinic visit, she changed her work schedule to a regular day shift. Her sleep paralysis persisted, but with reduced frequency. She underwent an overnight polysomnogram and multiple sleep latency test after her sleep and work schedule became more regular.

Sleep study results include:

Polysomnogram

Apnea Hypopnea Index 0.2/hr, Respiratory Disturbance Index 2.1/hr
O2 nadir 92% from baseline 96%
Time in Bed: 395 mins
Total Sleep Time 308 mins
Sleep Onset Latency 35.5 mins
REM onset latency 110.5 mins

Multiple Sleep Latency Test

Sleep in 2/5 naps
Sleep Onset REM 0/5 naps
Mean Sleep Onset Latency 16 mins 12 seconds

These results showed no evidence of sleep related breathing disorder or a central disorder of hypersomnolence.

She was started on Venlafaxine XR 35 mg qday. After starting the medication, her sleep paralysis episodes and daytime sleepiness resolved and she no longer needed to nap.

Discussion

Muscle atonia is a normal phenomenon in REM sleep thought to help prevent dream enactment and injury if it occurred while asleep. When a disruption in sleep architecture leads to boundary breakdown between REM and wake states, sleep paralysis can persist while the patient is transitioning between those states. This allows the person to experience features such as paralysis and consciousness, at the same time.

The condition is benign but can cause psychological distress and fear related to sleep. For instance, sleep paralysis has been associated with superstition and the paranormal. In Hong Kong, there is a condition known as “ghost oppression” and a similar phenomenon in Japan, *kanashibari*, descriptions of which resemble sleep paralysis.^{4,5} Both conditions in their respective countries are thought to be caused by evil spirits. There has also been an association found between sleep paralysis and lower sleep quality.⁶ In our patient, the frequency of these events caused her to be fearful of sleep, leading to subsequent insufficient sleep and daytime sleepiness.

The usual triggers for sleep paralysis include sleep deprivation, irregular sleep-wake schedules, supine sleep, alcohol, and anxiolytic medications.^{1,7,8} The condition is typically well controlled with reassurance, avoidance of known triggers, and sleep hygiene. If the sleep paralysis is recurrent or there is significant clinical distress, a pharmacological agent can be started. After our patient adjusted her work schedule to daytime, her sleep paralysis episodes persisted.

The pharmacological treatment for recurrent isolated sleep paralysis includes low dose tricyclic antidepressants or selective serotonin reuptake inhibitors. Both classes are thought to work by inhibiting REM sleep.⁸ Examples of tricyclic antidepressants include clomipramine 25-50 mg qday, imipramine 25-150 mg qday, protriptyline 10-40 mg qday, and desmethyl-imipramine 25-150 mg qday.⁸ Selective serotonin reuptake inhibitors include fluoxetine 40-80 mg qday, femoxetine 600 mg qday, and venlafaxine XR 37.5 mg qday (3,8). Of note, the hallucinations seen in sleep paralysis has been proposed by Jalal to occur by activation of the serotonin 2A receptor.⁹ In our patient, venlafaxine XR 37.5mg controlled her sleep paralysis allowing for resumption of adequate sleep. She no longer required daytime naps.

While muscle atonia is a normal phenomenon in REM sleep, it can sometimes persist upon awakening from REM sleep due to a disruptive trigger. While benign, these episodes can induce fear surrounding sleep and can lead to poor daytime functioning and sleepiness. Typically, adequate sleep hygiene and avoidance of triggers can prevent episodes. However, if recurrent, treatment with tricyclic antidepressants or selective serotonin inhibitors can be used to reduce frequency.

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