

A Rare Type of Epilepsy: A Case Report

Ali Hassan Salman Nasser^{1*}

Article Information

Received: December 05, 2022

Accepted: December 16, 2022

Published: December 23, 2022

Keywords

*Epilepsy, Obstructive
Sleep Apnea, Depression,
Hypersomnolence, Insomnia,
Pregabalin, Case Study*

ABSTRACT

Epilepsy is one of the most frequent severe brain disorders, impacting about 70 million individuals globally. Its prevalence is bimodal, with neonates and the elderly suffering the highest risk. Whether using medications or not, nearly two out of every three individuals suffering from new-onset epilepsy will achieve long-term seizures remission, although around half will experience mild to moderately severe side effects. Patients with epilepsy, mainly the 20-30percent of the respondents whose seizures are not fully controlled by present treatments (drug-resistant epilepsy), have a significantly increased risk of death, mental and physical comorbidities, and severe pharmaceutical effects. Here we present a case of 56-year-old man who was admitted to a private hospital and diagnosed with a rare type of epilepsy. He was unconscious, hence reported as comatose state. Mental diseases, such as depression, have also been predominantly studied in our patients, although there was a rising acknowledgment of a bidirectional association between the epilepsy and depression. The patient has been introduced to pregabalin 75mg once to rouse from a comatose state. Following this regime, he was declared conscious and alive. He was discharged and continued to pregabalin 75mg daily until the next follow-up. Pregabalin was found to be a significant addition to the treatment of epilepsy because of its effective antiseizure action, favorable pharmacokinetic properties, and efficacy in frequent co-morbidities. Antiepileptic drug development must be resuscitated rapidly in order to identify new effective anti-seizure medications for the treatment of drug-resistant epilepsy International health practitioners may continue the further investigation related to the onset of this condition to reach significant evidence and treatment.

INTRODUCTION

The choice of whether or not to do a formal laboratory assessment of the sleeping patient is crucial to the evaluation of patients with sleep problems. Many of the presenting symptoms may be related to different diseases that entail sleep disruption; however, much of the information in this section focuses on the patient with respiratory irregularities during sleep. Though a more comprehensive system for categorizing sleep disorders has been devised and published elsewhere (Coleman *et al.*, 1982; Thorpy, 2012), the method used here is more streamlined and focuses on symptoms. Since there are more sleep labs available than ever before, this article will concentrate

on how to recommend individuals who may be suffering from a sleep issue to one. Table 1 provides an exhaustive list of symptoms experienced by individuals with sleep-related breathing issues. In contrast, Table 2 provides the most prevalent signs that indicate sleep disturbance as the underlying cause. Patients with sleep disorders often have insomnia (trouble starting or keeping asleep) or excessive daytime drowsiness (hypersomnolence) (Bixler *et al.*, 1979; Thorpy, 2012). Because of the intertwined nature of sleep and wakefulness, it is not uncommon for both of these conditions to coexist. However, to treat either disease effectively, it is necessary first to identify whether insomnia or hypersomnolence is more prevalent.

Table 1: Symptoms in Patients with breathing disorders during sleep

| |
|---|
| Major or Common symptoms |
| Awakening from slumber (occasionally with shortness of breath) |
| Abrupt cessations of breathing (as reported by bed partner) |
| An excessive inability to stay awake throughout the day (hypersomnolence) |
| Screeching snores |
| Minor or less common symptoms |
| Sleep-related abnormal motor behavior |
| Insomnia |

Table 2: Symptoms Suggesting Underlying sleeping disorders

| |
|-----------------|
| Insomnia |
| Hypersomnolence |

Hypersomnolence

Simply asking patients may diagnose pathologic daytime drowsiness if they have experienced tiredness during

typically awake activities like chatting or eating (Smith, 1988). Sleepiness during sedentary activities like reading or watching television is a common symptom of a

¹ AVP, Oman Insurance Co Dubai, United Arab Emirates

* Corresponding author's e-mail: AliHassan35S@outlook.com

sleep disturbance in its early stages, but it may be easy to overlook. It is thus important to listen to the input of loved ones who may have noticed a shift in the subject's degree of attention. Hypersomnolence differs from exhaustion, fatigue, or lethargy since those states do not automatically lead to drowsiness. The social shame and fear of job loss sometimes lead individuals to conceal clinically obvious hypersomnolence (Smith, 1988). Moreover, sleep apnea and other sleep disorders may cause cognitive impairment, reducing the individual's sensitivity to tiredness. Therefore, it may be more important to assess how well a person sleeps than how well they can remain up when trying to provoke this symptom. Inquire as to whether or not you tend to nod off when doing boring or repetitive tasks.

Many factors may lead to excessive sleepiness. Night-time sleep problems are the first cause of daytime drowsiness because they alter the typical sleep pattern (Smith, 1988). Sleep disorders like sleep apnea or restless leg syndrome may drastically reduce restorative slow-wave sleep while boosting stage 1 sleep. The frequency of simultaneous partial arousals, which may follow respiratory or movement abnormalities, is more significant than any changes in the distribution of phases. A night of sleep may be severely fragmented and shortened by as many as 300 to 500 arousals, each lasting 15 to 20 seconds. Despite the high frequency, patients are unaware of a sleep disorder since the arousals do not lead to complete restlessness (Zarcone, 1973).

Insomnia

Insomnia, which is not a diagnosis in and of itself but rather a symptom complex defined by difficulty in beginning sleep, intermittent arousal from sleep, or early morning awakenings with an inability to return to rest, is another important presenting symptom of individuals with sleep disorders. It is common to practice dividing insomnia into two categories: temporary and chronic (Thorpy, 2012). Insomnia that lasts just a few nights and is brought on by external factors that often pass on their own is considered transient or episodic. Psychophysiological stress is the leading cause of temporary sleeplessness (Carskadon *et al.*, 1976). Though sleep lab examinations often show normal sleep architecture, chronic or persistent insomnia is typically attributable to a chronic worry state (Carskadon *et al.*, 1976).

Suppose a patient has real insomnia, as shown by decreased total sleep duration, fragmented sleep, or early morning awakenings. In that case, they will likely also have chronic weariness or lassitude but not hypersomnolence. Insomnia is common in patients who suffer from sleep apnea or movement problems because of the numerous full awakenings that might occur as a result. The elderly may have a lower arousal threshold than younger patients, making it more difficult to fall asleep and stay asleep even when mild central or obstructive sleep apnea is present. Similarly, to patients with hypersomnolence, endogenous and external stimuli may have tertiary effects on sleep.

Insomnia and drowsiness throughout the day are common side effects of serious medical conditions such as chronic pain, dyspnea, and others. However, normal sleep start with early morning awakenings and is more common among depressed people. Significant problems with falling asleep may also be caused by exogenous causes, such as stimulant drugs or substance usage.

Epilepsy

Around 1% of the population has epilepsy, making it the second most frequent major neurological illness after stroke (Stafstrom, 2006). About 50 million individuals worldwide have epilepsy, with 90 percent of those affected living in poorer nations (Kandar *et al.*, 2012). Our knowledge of epilepsy has advanced in various ways during the last several years. It is a common chronic neurological condition characterized by repeated unprovoked seizures and an imbalance between brain excitability and inhibition (Blume *et al.*, 2001; Holmes & Ben-Ari, 2001).

Keeping the thalamus and its cortical connections, the temporolimbic system, and the ascending reticular system in good working order is essential for maintaining consciousness. If the ascending reticular system were damaged structurally or functionally, the patient would have severe abnormalities in their ability to quantify their surroundings or perhaps go into a coma. EEG patterns in a coma have been used to predict coma outcomes (Bauer *et al.*, 2013) for decades and correlate to the degree of consciousness impairment and the depth of coma. However, the normal EEG patterns do not indicate a specific coma etiology (Synek, 1989, 1990a, 1990b). These developments have led to a new taxonomy of epileptic seizures and epilepsies. The diagnosis is founded on a thorough clinical history and a credible eyewitness description of a seizure (Thijs *et al.*, 2019). Over the last decade, novel medicines for the treatment of epilepsy have proliferated. With an ever-expanding range of possibilities, physicians must select which therapy, or combined effect of interventions, is optimal for a specific individual. Despite the existence of controlled scientific trials for each therapy option, the answers to these concerns may remain elusive (Leeman-Markowski & Schachter, 2017; Thijs *et al.*, 2019). Ancillary investigations can aid in the determination of the etiology and prognosis (Stafstrom & Carmant, 2015). Therefore, we report a case of a male who have suffered from a rare cognitive behavior that included epilepsy and depression with variables like insomnia, Obstructive sleep apnea, and Hypersomnolence. The combination of such conditions have turned the case as rare.

Case Presentation

A 56-year-old male, who is receiving treatment in a private hospital in the United Arab Emirates, suffers from a very strange condition for which the doctors did not find an explanation and considered it a very rare case in medical science. The patient's chronic symptoms had a history of depression during sleeping and he used to

take “Clonazepam gabapentin” to treat depression. The patient was also diabetic for which he takes “Metformin” 750mg per day. History of cardiovascular disease was also reported and “atrova STATIN” was used to treat congenital heart disease. Along with these medications, “Esomeprazole” was used to treat stomach acid 20mg once a day, “Ergocalciferol” one capsule per week was given to the patient that enhances VIT D level, “Neurobion” 100mg to enhance vitamin B complex and magnesium oxide 400mg once a day.

The acute illness of patient reported the onset symptoms 5 years ago, which was worst on the right leg and worsened over time in the form of mild irritability in the leg, causing difficulty in moving. He had difficulty and utility of the feet in both legs, which was worsening during sleep and at times of prolonged sitting. The abnormal leg movements started worsening, and he used to have wild flinging movements in sleep that resulted in fatigue and numbness of the outer aspect of the left thigh. The patient was started with SIFROL, Lyrica, and lidocaine to treat this condition, but no effect was measured. Due to this, the doctor advised shifting towards another treatment, i.e., “Pramipexole with gabapentin,” to treat the numbness and burning sensation and increased the dose of Lyrica and lidocaine. Insomnia was also reported from the patient’s history, and “Zolpidem” was used to treat it, 5mg once a day. No history of difficulty of gait or any dystonic posturing, no history session of involvement of higher mental functions; cranial nerves, cerebellar system, autonomic nervous system.

At the time of admittance, patient was unconscious for straight seven hours. No seizures were reported. According to the neurologist, this problem is not limited to his area of expertise; as the patient experiences this condition four or five times a year. There have been no warning symptoms before the onset of this condition; it occurred suddenly. Doctors in the emergency department take immediate measures to bring a comatose patient back to life, but no improvement was observed initially. The physician also implanted a needle into the patient’s body, used cold water and bits of iron to contact the patient’s feet to rouse him. After ensuring that the patient’s vital organs are functioning correctly, he was taken to the medical imaging room for MRI & CT scan. The patient was neither dead nor alive; and merely “Attentive” to the movements and sounds in his immediate environment. After 7 hours, the patient spontaneously rouses from his coma, without the help of any medical professionals or mechanical aids, and expresses a desire to be discharged; hence the patient was considered “Alive.” After regaining consciousness, the patient answered the basic questions correctly about his identity and present state. However, the patient remained silent when the doctor inquires about the events that transpired half an hour before his loss of consciousness. The patient was sent to a team of specialists, including a cardiologist, psychiatrist, oncologist, ENT, and pulmonologist. Moreover, the internal brain and nerves, and the result came from the doctors after the

testing, from x-rays to medical tests, showed everything was normal. Since he was in unconscious state for 7 hours, the neurologist prescribed him Antiepileptic drugs; Pregabalin and Lyrica. According to the neurologist, the condition of unconsciousness could have been a possible reason of a rare type of epilepsy.

DISCUSSION

From the investigation it is evident that patient has a rare type of epilepsy. Nowadays, several safe antiepileptic drugs are introduced limiting the indications for depression and anxiety prescription. Despite the fact that there are safer ways to treat status epilepticus, the medicine has its own market. Increased chloride inflow and neural inhibition are hypothesized to be responsible for the antiepileptic effects of the drugs through its impact on gamma-aminobutyric acid (GABA) receptors. (Olsen *et al.*, 1986). Depression is an under recognized yet prevalent comorbidity of epilepsy. The identification of mood disorders is the first and most crucial step in providing effective treatment for this group. Improved detection and treatment quality may be achieved by increased clinical attention and the regular use of a validated screening instrument. People who have epilepsy, like the general community, are increasingly curious in complementary and alternative medicine treatments for persistent conditions like depression (Fenton & Udwin, 1965).

Epileptic disorders include Sleep disorders, such as narcolepsy (NRCLP), restless legs syndrome (RLS), and obstructive sleep apnea syndrome (OSAS), are increasingly thought to be influenced by a set of inherited factors, and evidence for this is mounting (Caylak, 2009). Excessive daytime drowsiness, cataplexy, sleep paralysis, and hallucinations are characteristic symptoms of NRCLP. A common symptom of RLS is an incessant itch to move one’s legs, which may make it difficult to fall asleep. Recurrent bouts of upper airway collapse and blockage during sleep define Obstructive Sleep Apnea Syndrome (OSAS), a serious sleep disorder (Almbaidheen & Bodur, 2021).

However, the results of our research imply that antiepileptic medicine pregabalin might be useful in the treatment of individuals suffering from epilepsy, depression, and OSA. Pregabalin, the S-enantiomer of 3-aminomethyl-5-methylhexanoic acid, is an antiepileptic drug (AED) of the second generation, developed after gabapentin, with enhanced pharmacokinetic and pharmacological properties. Pregabalin is a favorable therapy choice due to its rapid absorption, high bioavailability, and absence of transporter saturation at therapeutic levels. More than ninety percent of the drug is removed in the urine, and nearly none is metabolized by the liver. Pregabalin is believed to exert its antiepileptic action through modifying calcium channel traffic and physiology by binding to the 2 subunit of P/Q-type voltage-gated calcium channels, hence reducing presynaptic calcium influx and neurotransmitter release (Hamandi & Sander,

2006; Schulze-Bonhage, 2013).

Currently, our group is conducting follow-up experiments to validate this rare case and to investigate the potential benefits of antiepileptic medications. Moreover, the current scenario is also under study by international hospitals and medical professionals.

CONCLUSION

It is generally agreed that epilepsy, with a lack of clarity in its definition, is best understood as a transitional condition. The health professionals diagnosed our patient with a very unusual kind of epilepsy. Our findings indicate a strong link between epilepsy, OSA, depression, and RLS. Moreover, medical researchers worldwide are also examining the present situation. In our case, Pregabalin has proven to be effective against epilepsy as it prevents seizures in epilepsy by lowering aberrant electrical activity in the brain. It suppresses the nerves pain by influencing pain messages traveling through the brain and down the spine. It prevents brain from producing the chemicals that cause anxiety. More antiepileptogenic drugs are also required to prevent epilepsy before the first seizure in at-risk individuals, as well as disease-modifying medications to treat persistent severe seizures associated with progressing underlying illness.

Acknowledgement

The author is thankful to the University of Manchester, Saudi Arabia, for the continuous support of this research study.

Funding

No funding sources are reported.

Conflict of Interest

The author does not have any conflict of interest.

REFERENCES

- Almbaidheen, M., & Bodur, M. (2021). Case report: early onset narcolepsy initially misdiagnosed as obstructive sleep apnea syndrome. *The Turkish Journal of Pediatrics*, 63(2), 334-338.
- Bauer, G., Trinka, E., & Kaplan, P. W. (2013). EEG Patterns in hypoxic encephalopathies (post-cardiac arrest syndrome): fluctuations, transitions, and reactions. *Journal of Clinical Neurophysiology*, 30(5), 477-489.
- Bixler, E. O., Kales, A., Soldatos, C. R., Kales, J. D., & Healey, S. (1979). Prevalence of sleep disorders in the Los Angeles metropolitan area. *The American journal of psychiatry*.
- Blume, W. T., Lüders, H. O., Mizrahi, E., Tassinari, C., van Emde Boas, W., & Engel Jr, E. o., Jerome. (2001). Glossary of descriptive terminology for ictal semiology: report of the ILAE task force on classification and terminology. *Epilepsia*, 42(9), 1212-1218.
- Caraskadon, M. A., Dement, W. C., Mitlet, M., Guilleminault, C., Zarcone, V. P., & Spiegel, R. (1976). Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *Am J Psychiatry*, 133(12), 1382-1388.
- Caylak, E. (2009). The genetics of sleep disorders in humans: narcolepsy, restless legs syndrome, and obstructive sleep apnea syndrome. *American Journal of Medical Genetics Part A*, 149(11), 2612-2626.
- Coleman, R. M., Roffwarg, H. P., Kennedy, S. J., Guilleminault, C., Cinque, J., Cohn, M. A., Karacan, I., Kupfer, D. J., Lemmi, H., & Miles, L. E. (1982). Sleep-wake disorders based on a polysomnographic diagnosis: a national cooperative study. *Jama*, 247(7), 997-1003.
- Fenton, G. W., & Udwin, E. (1965). Homicide, temporal lobe epilepsy and depression: a case report. *The British Journal of Psychiatry*, 111(473), 304-306.
- Hamandi, K., & Sander, J. W. (2006). Pregabalin: a new antiepileptic drug for refractory epilepsy. *Seizure*, 15(2), 73-78.
- Holmes, G. L., & Ben-Ari, Y. (2001). The neurobiology and consequences of epilepsy in the developing brain. *Pediatric research*, 49(3), 320-325.
- Kandar, H., Das, S. K., Ghosh, L., & Gupta, B. K. (2012). Epilepsy and its management: A review. *Journal of PharmaSciTech*, 1(2), 20-26.
- Leeman-Markowski, B. A., & Schachter, S. C. (2017). Cognitive and behavioral interventions in epilepsy. *Current neurology and neuroscience reports*, 17(5), 1-11.
- Olsen, R. W., Yang, J., King, R. G., Dilber, A., Stauber, G. B., & Ransom, R. W. (1986). V. Barbiturate and benzodiazepine modulation of GABA receptor binding and function. *Life sciences*, 39(21), 1969-1976.
- Schulze-Bonhage, A. (2013). Pharmacokinetic and pharmacodynamic profile of pregabalin and its role in the treatment of epilepsy. *Expert Opinion on Drug Metabolism & Toxicology*, 9(1), 105-115.
- Smith, P. L. (1988). Evaluation of patients with sleep disorders. *Seminars in respiratory medicine*,
- Stafstrom, C. E. (2006). Epilepsy: a review of selected clinical syndromes and advances in basic science. *Journal of Cerebral Blood Flow & Metabolism*, 26(8), 983-1004.
- Stafstrom, C. E., & Carmant, L. (2015). Seizures and epilepsy: an overview for neuroscientists. *Cold Spring Harbor perspectives in medicine*, 5(6), a022426.
- Synek, V. (1989). Validity of a revised EEG coma scale for predicting survival in anoxic encephalopathy. *Clinical and experimental neurology*, 26, 119-127.
- Synek, V. (1990a). Revised EEG coma scale in diffuse acute head injuries in adults. *Clinical and experimental neurology*, 27, 99-111.
- Synek, V. (1990b). Value of a revised EEG coma scale for prognosis after cerebral anoxia and diffuse head injury. *Clinical Electroencephalography*, 21(1), 25-30.
- Thijs, R. D., Surges, R., O'Brien, T. J., & Sander, J. W. (2019). Epilepsy in adults. *The lancet*, 393(10172), 689-701.
- Thorpy, M. J. (2012). Classification of sleep disorders. *Neurotherapeutics*, 9(4), 687-701.
- Zarcone, V. (1973). Narcolepsy. *New England journal of medicine*, 288(22), 1156-1166.