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CPAP Therapy Improved Sleep and Reduced Seizures in a Young, Obese Epileptic Woman with OSA

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Abstract

Introduction: Sleep disorders and epilepsy are both common diseases in the general population. Comorbidity between obstructive sleep apnea syndrome (OSAS) and epilepsy varies from 8% to 76%, with a recent meta-analysis reporting a 33.4% prevalence of mild-to-severe OSAS in epileptic patients.

Case report: We describe the case of a 39 year old woman, non-smoker, suffering from temporal lobe epilepsy; she was diagnosed in 2018 upon experiencing her first nocturnal tonic-clonic seizure. She also suffers from hyperinsulinism and obesity (BMI: 36.3). In 2020, she came to our Sleep Clinic because of excessive daytime sleepiness (EDS). At that time, she was on Lacosamide (LAC) 150 mg twice daily with partial control of her seizures. Her EEG showed bi-temporal interictal epileptiform discharges (IEDs) during wake and sleep, more evident over the right hemisphere. Cardiorespiratory monitoring revealed an apneic/hypopneic index (AHI) of 28.9, indicative of moderate to severe OSAS. She was subsequently titrated on CPAP and is currently on CPAP at 9 cmH_2O with complete resolution of EDS and apneic episodes. Disappearance of IEDs during sleep was observed at her check-up in 2022, and LAC was reduced to 50 mg twice a day. Her CPAP compliance is still excellent.

Conclusion: We report a case of a young, obese epileptic woman with persistent seizures despite antiseizure medication. Interestingly, CPAP treatment alone led to EEG normalization with no further seizure recurrence, suggesting a potentially beneficial role of CPAP on both sleep and epilepsy.

Keywords: Epilepsy; Obstructive Sleep Apnea Syndrome; CPAP; Antiseizure Medications.

Introduction

Sleep disorders and epilepsy are both common pathologies in the general population. The comorbidity between obstructive sleep apnea syndrome (OSAS) and epilepsy ranges from 8% to 76% [1]. A recent meta-analysis determined that epileptic patients have a higher likelihood of developing OSAS compared to healthy controls, with a prevalence of 33.4% for mild to severe cases [2]. There is evidence of a bidirectional relationship between OSAS and epilepsy, possibly due to their influence on the neural pathways involved in regulating breathing and sleep, as well as alterations in inflammatory processes that are shared by both conditions [3]. It is also known that epilepsy can exacerbate disorders such as central apnea and OSAS. Indeed, seizures can potentially cause upper airway muscle inflammation. Various anti-seizure medications (ASMs) such as valproic acid and pregabalin can impact OSAS by promoting weight gain, potentially worsening or increasing the likelihood of developing OSAS. Benzodiazepines, instead, are associated with a reduction of upper airway muscle tone and the ventilatory response to hypoxia [3]. On the other hand, negative effects of OSAS in epilepsy include hypoxic damage and intermittent hypoxemia that can cause oxidative stress and trigger

epileptic seizures. The impact of sleep deprivation on patients with OSAS is evident in the altered microarchitecture of their sleep, resulting in increased frequency and duration of the A-phase of the Cyclic Alternating Pattern (CAP). This has been linked to interictal epileptiform discharges (IEDs) and epileptic seizures [3].

We present the case of a 39-year-old non-smoking, obese woman (BMI 36.3), affected by hyperinsulinism and diagnosed with Von Willebrand syndrome in 2013. In 2018, she was diagnosed with temporal lobe epilepsy (TLE) at a local hospital, following the onset of partial epileptic seizures with secondary generalization. An electroencephalogram (EEG) revealed bilateral centro-temporal discharges, predominantly on the right side (Figure 1). Although the patient was treated with 1000 mg bid of Levetiracetam (LEV), she did not experience a reduction in seizure frequency. Moreover, the patient endured important side effects such as headache and diplopia. After consulting with a local neurologist, she discontinued LEV and started treatment with Topiramate (TPM); unfortunately, her epileptic seizures increased, thus, TPM was discontinued and Lacosamide (LAC) was introduced (150 mg bid).

In 2020, the patient began experiencing excessive daytime sleepiness (EDS) and sought assistance at our Sleep Clinic in 2021. As part of her evaluation, she completed the following self-administered questionnaires: Epworth Sleepiness Scale (ESS; 13, normal value <10); Pittsburgh Sleep Quality Index (PSQI; 14, normal value <5), Hamilton Anxiety Rating Scale (HAM-A; 23, normal value <17), and Beck Depression Inventory (BDI: 22, normal value <10). As part of the general physical examination, the patient's height was determined to be 170 cm and her body weight was recorded as 105 kg, resulting in a BMI of 36.3. Additionally, her neck circumference was measured at 37 cm, waist circumference at 113 cm, and she presented a class II Mallampati score. Ambulatory cardiorespiratory monitoring disclosed an Apnea-Hypopnea Index (AHI) of 28.9 (normal value <5), with an average oxygen saturation of 93%, T90 of 1.2% (Figure 2). Therefore, she initiated continuous positive airway pressure (CPAP) titration, ultimately reaching a steady pressure of 9 cm H_20. During her follow-up visit in February 2022, she presented hematologic and cardiologic evaluations, a pneumological assessment with spirometry, an ENT examination with flexible laryngoscopy and a brain MRI, all within normal limits. In March 2022, she underwent an EEG which displayed fewer IEDs compared to her previous one (Figure 3). The patient reported an improved quality of life, reduced EDS and fewer seizures, leading to a reduction of LAC dosage to 100 mg bid. The HARNESS (Harmonized Neuroimaging of Epilepsy Structural Sequences) brain MRI protocol was used (Figure 4) in February 2023, showing a hyperintense region over the right hippocampal tail (best seen in coronal FLAIR sequences). Another follow-up EEG was conducted in October 2023, revealing a further reduction in paroxysmal alterations. Additionally, she repeated the self-administered assessments: ESS: 1, PSQI: 0, BDI: 0, and cardio-respiratory monitoring displayed an AHI of 0.5. Thus, LAC dosage was reduced to 50 mg bid and the continuance of CPAP therapy was prescribed.

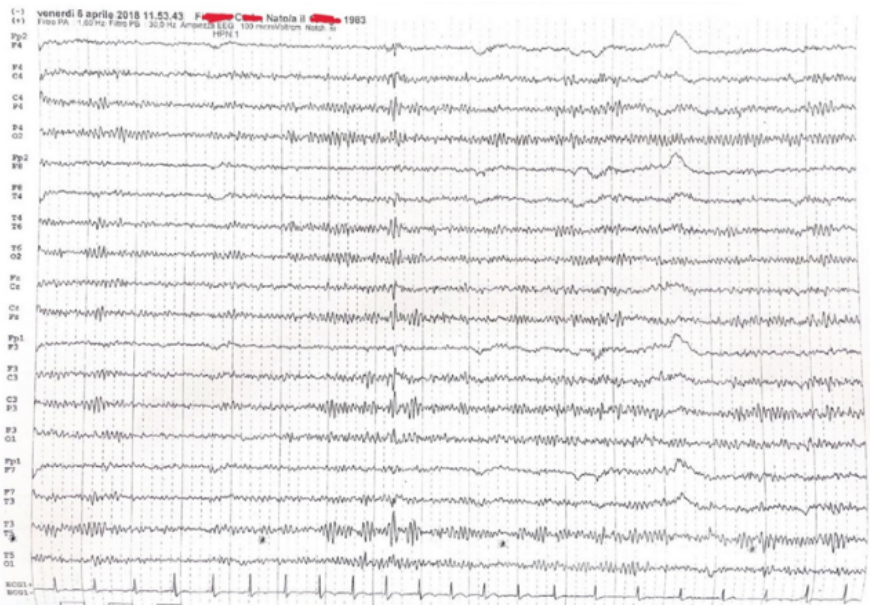

Figure 1: An electroencephalogram (EEG) revealed bilateral centro-temporal discharges, predominantly on the right side.

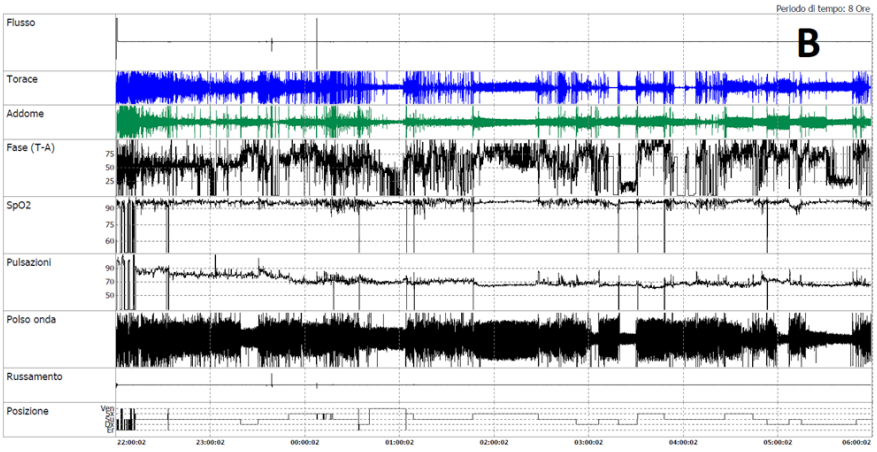

Figure 2: Ambulatory cardiorespiratory monitoring disclosed an Apnea-Hypopnea Index.

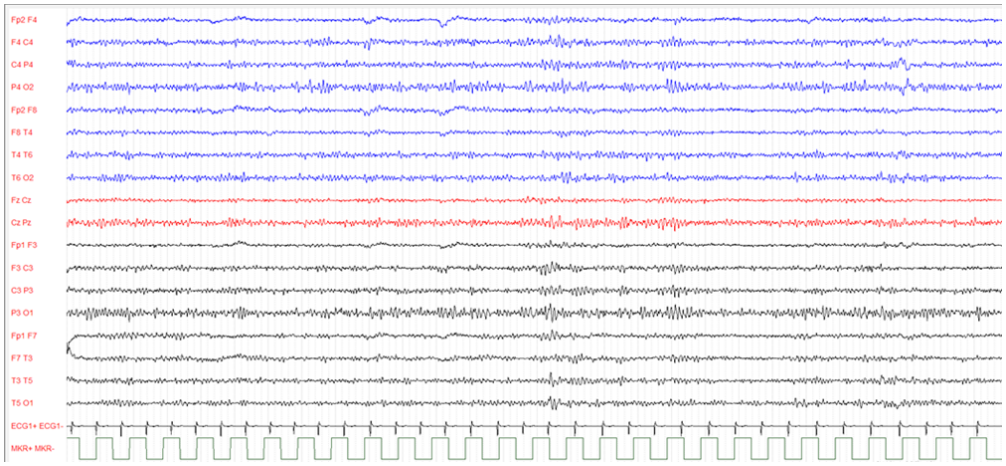

Figure 3: Fewer interictal epileptiform discharges compared to her previous EEG.

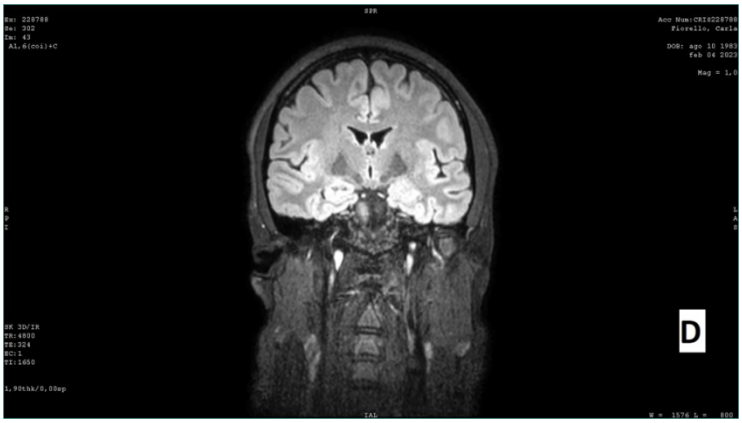

Figure 4: Harmonized Neuroimaging of Epilepsy Structural Sequences.

Discussion

According to recent evidence and various studies conducted over the past two decades, there is a strong association between epilepsy and sleep disorders, specifically OSAS. Numerous studies have demonstrated that IEDs are more frequent in OSAS patients compared to the general population. Certain ASMs used to treat epileptic seizures may result in drowsiness as a side effect, although the extent of this effect varies greatly among individuals, and some ASMs have a low propensity to cause drowsiness. CPAP is considered the gold standard for OSAS treatment. In both a retrospective review and a prospective study, CPAP was found to be effective in improving seizure control. Furthermore, a study conducted in 2022 included participants with comorbid epilepsy and OSA, as well as those with only OSA. The results showed that nearly half (41%) of the comorbid patients who used CPAP were seizure-free at baseline [2,4,5]. The limitations of previous studies arise from the predominantly retrospective nature of these investigations. Moreover, several studies exhibit various confounding factors: some patients displayed poor adherence to CPAP treatment, others underwent adjustments to their ASM dosage or prescription, and others sustained epilepsy surgery while using CPAP, resulting in a subsequent reduction in seizures [5].

Our case study concerns a young woman who received a diagnosis of epilepsy in 2018 and began experiencing EDS in 2021. Subsequently, she was diagnosed with OSAS at our Sleep Clinic and initiated CPAP therapy. The patient had a significant number of epileptic seizures despite treatment with LAC 150 mg bid. From 2021 onwards, a gradual reduction in epileptic seizures, a decrease in EDS, and a reduction of paroxysmal alterations in follow-up EEGs were observed. This allowed us to reduce the ASM, coinciding with a progressive improvement in both symptomatic (epileptic seizures and EDS) and electrophysiological aspects with CPAP therapy. Currently, there is a paucity of well-designed retrospective or prospective studies to validate the notion that CPAP therapy improves the quality of life or life expectancy of epileptic patients. In light of this, our case study seeks to document the potential beneficial impact of CPAP treatment on both OSAS symptoms and the occurrence of epileptic seizures in a comorbid patient. Larger prospective studies are warranted to provide insight into whether CPAP treatment can improve seizure control and potentially serve as a first-line treatment for OSA-Epilepsy patients.

Conflict of Interest: None.

References

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