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Case Report

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Resolution of central sleep apnea following baclofen discontinuation in a patient with restless legs syndrome

Abstract

Background: Central sleep apnea (CSA) is characterized by a lack of respiratory effort during sleep and may be triggered by medications affecting central respiratory control. Baclofen, a $GABA_B$ agonist used for spasticity and sometimes for restless legs syndrome (RLS), has been associated with respiratory depression, although reports of CSA are limited.

Case Report: A middle-aged patient with RLS was treating it with baclofen. The patient reported increased fatigue and disrupted sleep. Polysomnography revealed an apnea-hypopnea index (AHI) of 19.3 events per hour from central sleep apnea along with an AHI of 11.1 events per hour from obstructive events. After discontinuing baclofen, repeat polysomnography showed resolution of central apneas.

Discussion: This case suggests a causal relationship between baclofen use and CSA, likely due to its central depressant effects on respiratory drive. While baclofen is not commonly recognized as a cause of CSA, clinicians should consider it in the differential diagnosis, especially when symptoms emerge after medication changes.

Conclusion: Baclofen may induce central sleep apnea in susceptible individuals. Recognition of this association is important, as discontinuation of the drug can lead to full resolution of symptoms and normalization of sleep-disordered breathing.

Keywords: Central sleep apnea, obstructive sleep apnea, baclofen, restless legs syndrome, mixed sleep apnea, GABA agonist

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Introduction

Mixed sleep apnea, characterized by coexisting central and obstructive respiratory events, presents a complex therapeutic challenge.[1] Baclofen, a $GABA_B$ receptor agonist, is primarily prescribed for spasticity.[2] However, in one study, it was shown to increase the frequency but decrease the amplitude of periodic leg movement disorder events (seen in 80% of RLS patients) and may be used off-label for restless legs syndrome (RLS).[3,4] While baclofen is known to suppress central respiratory drive and destabilize breathing during sleep, published case reports and series have documented central sleep apnea (CSA) emerging during baclofen therapy.[5-7] We report a case in which CSA was present during baclofen treatment for RLS but resolved following its discontinuation, supporting a causal association.

Case Presentation

A 46-year-old male with a history of moderate restless leg syndrome (RLS) was referred for evaluation of excessive daytime sleepiness and disrupted sleep. His medical history included hypertension and mild obesity (BMI 28.4 kg/m²). He was taking baclofen 20mg nightly for RLS symptoms, which he later reported that it provided limited benefit.

Polysomnography on Baclofen

A diagnostic polysomnogram (PSG) was performed during baclofen therapy, as shown in Figure 1. These studies measure the amount of times a person becomes apneic, as measured by a drop in airflow by 90% for at least 10 seconds, as well as hypopneic, as measured by drop in airflow by 30% for at least 10 seconds along with a drop in oxygen saturation by 3% or an arousal, according to the American Academy of Sleep Medicine.[8] In addition, to determine whether an apneic event is central or obstructive, it measures the respiratory effort through measuring the movement of the belts around the chest and abdomen, which reflects the movement of diaphragm. If the diaphragm is not moving during a sleep apneic, with no change in movement of the chest and abdomen belts, it reflects central sleep apnea is occurring. On the other hand, if the chest and abdomen belts have significant movement during an apneic event, it means that obstructive sleep apnea is occurring.

The polysomnograph demonstrated central sleep apnea (CSA) with an apnea–hypopnea index (AHI) of 31.4 events/hour, including a central AHI of 19.3, and oxygen desaturations to 84%. Normally, a person without central and obstructive sleep apnea would have 0 AHIs. In addition, an individual without lung disease should have oxygen levels above 95%, and their oxygen saturation would not briefly drop ≥ 3%.

An AHI of greater than 30 events/hour is considered a severe sleep apnea. In addition, there are more central sleep apneic events (19.3 AHIs) compared to obstructive sleep apneic events (11.1 AHIs). These sleep apneic events can be tracked on Figure 1 by observing the drops in oxygen saturation in the displayed epochs.

Course and Follow-up

The patient discontinued baclofen several months later due to lack of efficacy for RLS. A repeat polysomnograph was performed. This revealed complete resolution of central events, although an obstructive sleep apnea with an AHI of 22.8 persisted. Figure 2A displays his oxygen saturation curve after not taking baclofen whereas

Figure 2B displays his oxygen saturation curve while taking baclofen during the study's time period of 1:17 to 1:21 (hh:mm). The oxygen saturation does not dip below 90%, as it did in the Figure 2B (which is also present in Figure 1). This implies better oxygenation and may imply less severe desaturation events from sleep apnea. He was subsequently started on continuous positive airway pressure (CPAP) for management of OSA.

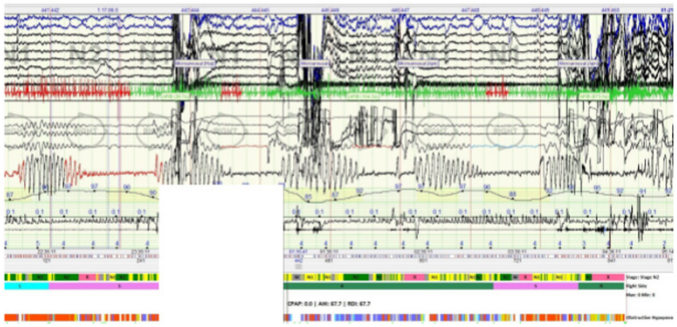

Figure 1 Polysomnograpghy of patient depicting multiple central sleep events after daily baclofen usage (20mg/day). There is a screen capture artifact (an empty white box) present.

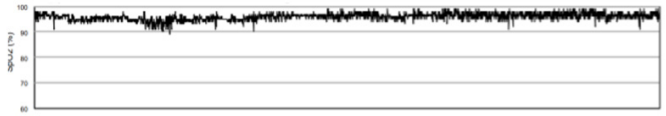

Figure 2A Oxygen Saturation curve of patient after discontinuation of baclofen usage.

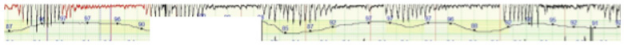

Figure 2B Oxygen saturation curve of patient from Figure 1's polysomnograpgh while taking baclofen from Epoch 441-451.

Discussion

This case illustrates the development of CSA during baclofen therapy and its resolution after withdrawal, strongly suggesting a causal role. Baclofen is a $GABA_B$ agonist that depresses respiratory centers and alters chemosensitivity.[5,9] Prior literature supports baclofen-induced CSA, both at standard and low doses.[4,5] The temporal association in this case—emergence of CSA on baclofen and disappearance after discontinuation—provides real-world confirmation of this adverse effect.

A potential mechanism underlying this phenomenon is depressed ventilatory drive. Baclofen acts on $GABA_B$ receptors within brainstem respiratory centers, including the parafacial respiratory group, leading to reduced tidal volume, inspiratory flow, and overall ventilatory output. Notably, administration of $GABA_B$ antagonists in animal models restored ventilatory drive after baclofen-induced suppression, supporting a causal role for GABAergic modulation of central respiratory rhythm.[8]

The persistence of OSA in this patient reflects an anatomically predisposed upper airway collapse, unrelated to baclofen.

Conclusion

This case underscores baclofen as a reversible cause of central sleep apnea. Clinicians prescribing baclofen, even at modest doses, should monitor for central respiratory events, especially in patients with underlying sleep-disordered breathing. Discontinuation of baclofen may lead to complete resolution of CSA.

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Disclosures

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