Parkinson's Disease Patients having Subjectively Varied Sleep Quality

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Abstract

One typical non-motor symptom of Parkinson's Disease (PD) is sleep disruption. When patients are in their "on medication" condition, Polysomnography (PSG) investigations are typically performed. With poor subjective sleep quality based on Polysomnography (PSG), our study sought to examine changes in sleep structure in drug-naive PD patients and explore potential relationships between sleep structure and clinical characteristics of the disease.

Keywords: Insomnia • dystonia

Introduction

Parkinson's Disease (PD) is a widespread chronic neurodegenerative condition that severely burdens individuals on both a material and mental level. Although motor symptoms are the hallmark of Parkinson's Disease (PD), a wide range of Non-Motor Symptoms (NMS), such as sleep disruption, neuropsychiatric symptoms, and autonomic dysfunction, can also be found in those who have the disease. Insomnia, Rapid Eye Movement (REM) sleep behavior disorder, sleep-disordered breathing, restless legs syndrome, and circadian dysregulation are among the sleep disorders that commonly affect patients with Parkinson's Disease (PD). Among PD patients, the loss in sleep quality is a fairly frequent complaint. The increased frequency of sleep problems may be related to the fact that neurodegenerative disorders impair the circadian rhythm system more quickly than age-related changes.

Additionally, there are significant associations between sleep disruptions and various motor and non-motor symptoms such pain, nocturia, and dystonia. Recently, it has been discovered that worse sleep quality exacerbates both motor and non-motor symptoms in PD. The condition may start out with several sleep issues that get worse as it develops, so it's important to maintain sufficient monitoring.

In the past, a number of sleep scales were mostly used to evaluate the quality of sleep. The Pittsburgh Sleep Quality Index (PSQI) and the Parkinson's Disease Sleep Scale (PDSS) have both been widely utilized in research to evaluate sleep in PD patients. Although these tools are quick to apply and effective, a thorough understanding of the patients' sleep patterns is still missing.

Video-Polysomnography (v-PSG), which is being used frequently, is thought to be the most effective way to diagnose sleep problems. PSG is able to carefully capture the occurrence of certain sleep episodes in addition to statistically analyzing the sleep structure. Previous PSG investigations found that patients with PD have less total sleep duration, less efficient sleep, and more sleep latency and alertness following sleep start than healthy controls. However, anti-PD medications invariably have an impact on sleep architecture. Dopamine agonists can forecast sleep stage 1, whereas MAO-B inhibitors are associated to sleep stages 2 and 3.

In this regard, a cohort of drug-naive patients is crucial for generating fresh perspectives on the non-motor spectrum of PD. With the aid of PSG equipment, this study sought to examine changes in sleep structure in drug-naive PD patients with poor subjective sleep quality compared to those with high sleep quality as well as the relationship between clinical characteristics and sleep structure.

Periodic Limb Movements While Sleeping (PLMS) have reportedly been linked to worsening PD symptoms, higher subjective sleep disruption, and a lower quality of life. Drug-naive PD patients with varying levels of subjective sleep quality did not show any discernible changes in PLMI fluctuation. However, among drug-naive individuals with poor sleep quality, our findings showed a link between PLMI and non-motor symptoms. There is evidence to support the existence of shared pathophysiological pathways between PLMS and PD. While dopaminergic augmentation may be able to regulate PLMS, nigrostriatal degeneration may encourage it. The regression model in our drug-naive PD group revealed that rising PLMI was an independent predictor predicting a higher NMSQ score in insomniacs.

There are a few methodological things to keep in mind. First, despite a few studies examining the characteristics of objective sleep measures in drug-naive PD patients, we carried out a more thorough clinical examination of these patients and further investigated the connection between sleep metrics and clinical aspects. Second, PSG data were the foundation of our work. Few published research on sleep disorders utilized objective ways to assess sleep quality; the bulk of these investigations employed subjective measures. To counteract the first-night effect, patients in our study did not have a second PSG assessment, which is a methodological and racial bias. Furthermore, we excluded healthy individuals who had and did not have sleep disorders such OSA, RBD, and PLMS.

Last but not least, just one center recruited patients for our cohort, which had a tiny number of them. The relationships between sleep measures and clinical features should be confirmed in other investigations using bigger populations and longitudinal follow-ups.

According to our findings, more than 50% of PD patients who are drug-naive have poor subjective sleep quality, which is mostly exhibited by longer nocturnal awakenings and less efficient sleep. A low quality of life and significant non-motor symptoms are both indications of inadequate sleep. Events of nocturnal arousal such as WASO and MAI may indicate the development of motor dysfunction. We offer fresh data on the relationships between sleep measures and clinical traits in PD patients before drug interference starts.

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