Review / *Derleme*

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The Importance of Sleep and Sleep Disorders in Multiple Sclerosis

Multipl Sklerozda Uykunun ve Uyku Bozukluklarının Önemi

İnan Özdemir, Semai Bek, Gülnihal Kutlu

Muğla Sıtkı Koçman University Faculty of Medicine, Department of Neurology, Muğla, Türkiye

Abstract

Multiple sclerosis (MS) and sleep disorders form a vicious cycle when assessing quality of life and disability. Sleep disorders can exacerbate the symptoms and signs of MS, while conversely, symptoms and signs of MS can worsen sleep disorders. Therefore, sleep disorders should be addressed, identified, and treated in the medical practice related to MS. Sleep disorders are summarized under essential headings, and their relationship with MS has been demonstrated based on current literature data.

Keywords: Multiple sclerosis, sleep, sleep disorders

Öz

Multipl skleroz (MS) ve uyku bozuklukları yaşam kalitesi ve engelliliğin değerlendirmesinde bir kısır döngü oluştururlar. Uyku bozuklukları, MS'ye ait belirti ve bulguların artmasına neden olurken diğer yandan MS'nin kendi semptomları ve kullanılan ilaçlar uyku bozukluklarını arttırmaktadır. Dolayısıyla MS pratiğinde uyku bozuklukları sorgulanmalı, tanınmalı ve tedavi edilmelidir. Bu yazıda temel olarak uyku bozuklukları başlıklar halinde özetlenmiş ve MS ile ilişkisi güncel literatür verisi eşliğinde ortaya konmuştur.

Anahtar Kelimeler: Multipl skleroz, uyku, uyku bozuklukları

Introduction

Sleep, once regarded a passive process of life until the mid-20th century, has undergone a conceptual transformation over the years with the advancement of electrophysiological studies. Healthy sleep is the physiological state that arises from a combination of voluntary decisions and involuntary biological activities. If you pay attention, you can actually see what a sleep disorder is in its definition. If biological activity puts you to sleep although you do not want to, the condition is considered a pathology. Similarly, if you cannot sleep although you want to, this is also classified as a pathology. The dynamic relationship between demand and biological activity must be balanced. Based on this interplay, healthy adult individuals are advised to aim for 7-9 hours of sleep per night. The body sleeps in the darkness of the night, integrated with nature, and awakens in the light of the day.

While the circadian rhythm triggers wakefulness, the homeostatic rhythm induces sleep. As long as this balance continues and remains rhythmically stable, we can consider it a healthy sleep. Although the anatomy of sleep physiology is not our main topic, many anatomical regions are active in the sleep-wake cycle. While brainstem histaminergic, serotonergic, noradrenergic, cholinergic, and dopaminergic neurons, along with hypothalamic orexinergic neurons, collaborate to enhance wakefulness, the hypothalamic preoptic area is responsible for inducing sleep. This cycle is controlled by the suprachiasmatic nucleus, which regulates the circadian clock.

Although sleep disorders are prevalent among individuals diagnosed with multiple sclerosis (MS), they are unfortunately much less frequently diagnosed than expected. This is because they are not routinely inquired about in clinical practice and are often not reported by the patients themselves. Of course, systemic disease itself has a deleterious impact on the standard of living. Even if patients are unaware, investigating sleep quality and habits is crucial when obtaining a detailed medical history. Although the prevalence of sleep disorders in healthy individuals is found to be 42-65% in different publications, it is reported that this rate increases approximately four times in patients with MS, despite methodological differences in studies.[1] Sleep disorders cause disability on their own and raise

Address for Correspondence/Yazışma Adresi: İnan Özdemir, MD, Muğla Sıtkı Koçman University Faculty of Medicine, Department of Neurology, Muğla, Türkiye
E-mail: drinanozdemir@gmail.com **ORCID-ID:** orcid.org/0000-0002-7510-4360

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questions about their contribution to or coexistence with MS. As a result, both medical conditions are diseases that require treatment, and their coexistence puts the patient in a more difficult situation.

The pathophysiology of MS-related sleep disorders is multifactorial. MS, which is based on autoimmune pathology, and sleep, whose immunology is not fully understood, create a dilemma. Interleukin-1 (IL1), IL2, IL6, IL8, IL15, IL18, epidermal growth factor, tumor necrosis factor-alpha, acidic fibroblast growth factor, colony-stimulating factor, and interferons trigger sleep. IL1 receptor antagonist IL1-RA, IL4, IL10, IL13, insulin-like growth factor, and soluble IL1 inhibit sleep.[2] In MS, overproduction of proinflammatory cytokines (IL-1, IL-6, tumor necrosis factor-alpha (TNF-α) contributes to both neuronal damage and sleep architecture disruptions. These cytokines particularly affect non-rapid eye movement (NREM) sleep, leading to decreased sleep duration and quality.[2] Although IL-1 and TNF-α have been shown to increase NREM sleep, these effects may disrupt homeostatic sleep regulation in chronic inflammation.[2] Another factor involved in the pathophysiology is disruptions in melatonin secretion and circadian rhythms. Disturbances in melatonin secretion are common in individuals with MS. The melatonin rhythm is disrupted by involvement of the suprachiasmatic nucleus and pineal gland.[1,2] One study identified genetic polymorphisms in melatonin metabolism, particularly in the progressive MS subtype, and the resulting decrease in melatonin levels disrupts the sleep-wake cycle.[3] MS lesions occurring in areas involved in sleep and respiratory regulation, such as the brainstem, hypothalamus, and cervical cord, play a role in the pathophysiology of disorders, such as obstructive sleep apnea (OSAS) and restless legs syndrome (RLS).[2-4] Medications used to treat MS, such as beta-interferons, antidepressants, and muscle relaxants, can impair sleep quality. These medications are associated with insomnia, daytime sleepiness, or parasomnias.[4] The onset of sleep disorders approximately five years before the clinical diagnosis of MS, and their significant exacerbation by the time of diagnosis, suggest that MS and sleep disorders may be linked in a cause-and-effect relationship rather than simply co-occurring.[5]

The International Classification of Sleep Disorders published by the American Academy of Sleep Medicine is main source for setting standards in sleep medicine. Within this classification framework, sleep disorders will be categorized into main headings, such as insomnia, sleep-related breathing disorders, centrally caused hypersomnolence, circadian rhythm sleep-wakefulness disorders, parasomnia, and sleep-related movement disorders. The evaluation will focus on their relationship with MS.[6]

Insomnia: Under the title of insomnia, there are chronic insomnia, short-term insomnia, isolated symptoms, other insomnia disorders, and normal variants (long-time in bed and short sleepers). Researchers have conducted extensive research on the coexistence of MS and chronic insomnia in patients with MS. Insomnia is defined as persistent difficulty in initiating, maintaining, intensifying, and maintaining the quality of sleep, resulting in impairment in daily activities, despite having adequate opportunities and conditions for sleep. In general terms, insomnia refers to the inability to sleep despite having the opportunity to obtain sufficient and high-quality sleep, leading to daytime dysfunction. Although its prevalence is 10% in the healthy population, it is observed six times more frequently in patients with MS.[7]

Depression, nocturia, pain, and spasticity are the most significant causes of insomnia in MS.[8] While depression is a more prevalent cause in women, pain, resulting from the effects on sensory pathways, is the most common cause in men. Nocturia caused by brainstem and particularly pontine lesions more prominently leads to insomnia.[9]

The first-line treatment for insomnia is cognitive behavioral therapy. It provides more lasting results than medication, has no side effects, and does not increase the medication burden in patients with MS. It includes sleep hygiene, stimulus control, and relaxation techniques. It also improves fatigue symptoms in MS.[1-4] The basic principle in treating it is to ensure excellent sleep hygiene. Under the concept of sleep hygiene, the patient should adhere to a consistent bedtime and wake-up time. If the patient takes a nap during the day, it should not exceed 1 hour and should be completed by 1:00 PM at the latest. The patient should avoid consuming food and drinks after dinner and refrain from napping in bed, including activities, such as watching television or using the phone. It is advisable to avoid using sleep-inducing devices with light, to only go to bed for sleeping purposes, and not to engage in activities, such as reading books. To obtain restorative slumber, it is essential to reside in an environment characterized by minimal illumination and sound, accompanied by the judicious selection of bedding and pillows. 5-Hydroxytryptamine 2 ligands, such as risperidone, olanzapine, mirtazapine, and mianserin can be used in medical treatment. It is important to recognize that these pharmacological agents may trigger or exacerbate symptoms related to RLS. Melatonin can be administered in doses up to 12 mg per day. There are study results reporting that a series of treatments, in the form of daily moderate-weight resistance exercises and transcranial direct electrical stimulation, facilitates the process of achieving sleep.[10-12]

Sleep-related Breathing Disorders: Obstructive sleep apnea syndrome, central sleep apnea syndrome (CSAS), sleep-related hypoventilation disorder [$PaCO_2$>55 mmHg and >10 min. (minimum)], sleep-related hypoxemia disorder (SpO_2<88% and <5 min.), as well as isolated symptoms and normal variants, such as snoring and catathrenia. Studies on sleep disorders and MS have primarily focused on apnea.

The presence of apneas characterized by upper airway narrowing or closure despite continuing respiratory effort is defined as OSAS, and the presence of apneas that occur after a decrease or cessation of airflow secondary to lost or decreased respiratory effort is defined as CSAS (Figure 1 and 2). The majority of sleep laboratory patients are diagnosed with OSAS. However, when sleep studies are conducted in patients with MS, the rate of OSAS diagnosis can be as high as 62%. This rate increases particularly in the phase of (REM) sleep, which is distinguished by rapid movements and a state of muscular

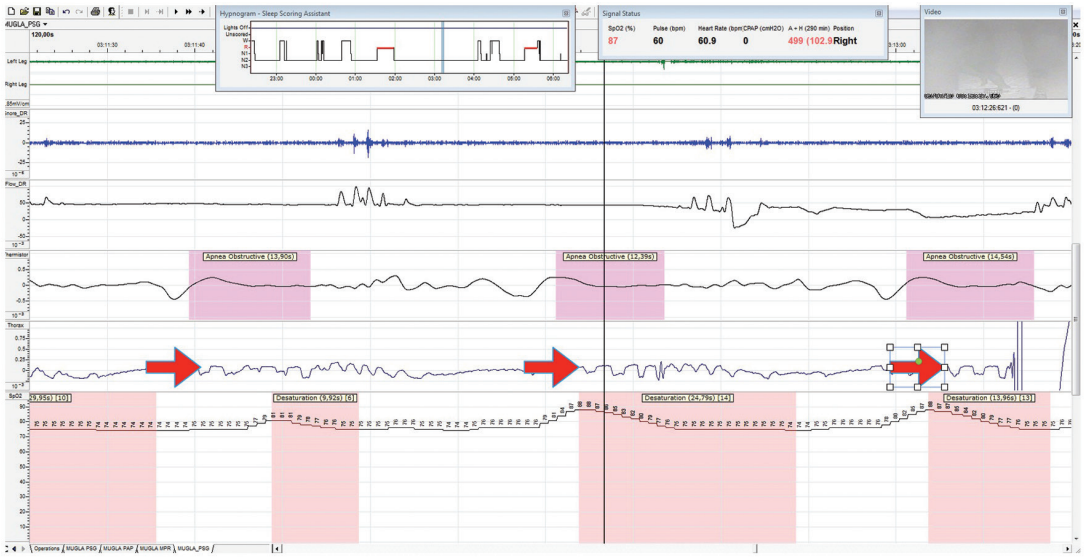

Figure 1. During the polysomnography recording, obstructive sleep apnea is observed in the epoch where respiratory effort (indicated by the red arrow) persists during apneas (highlighted in pink), leading to desaturations (shown in salmon color) detected in pulse oximetry

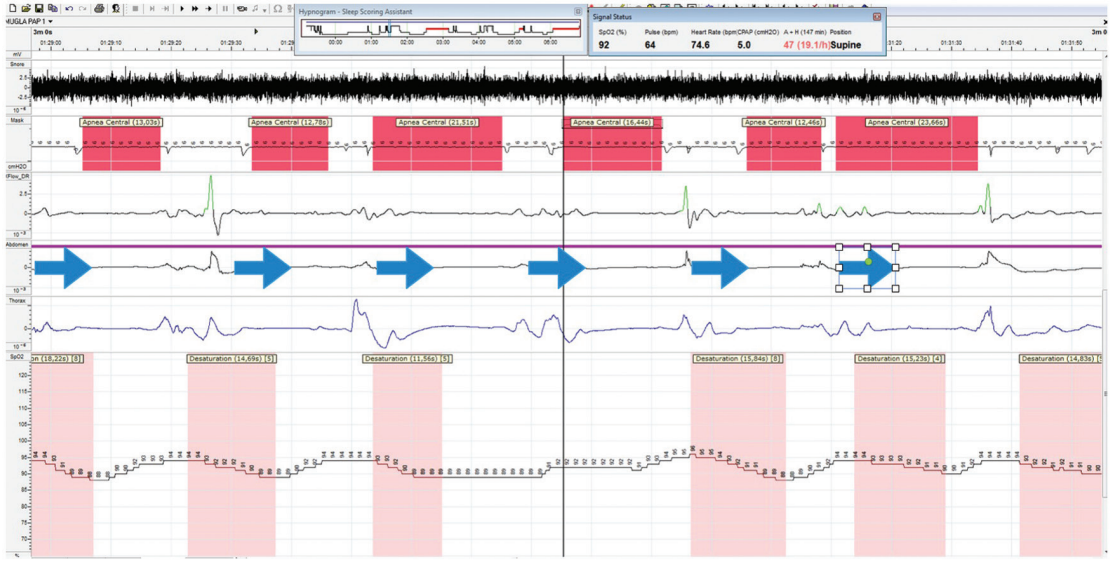

Figure 2. During the epoch captured in the polysomnography record, central sleep apnea is observed. This is characterized by a lack of respiratory effort (indicated by the blue arrow) during apneas (highlighted by the red arrow), leading to desaturations (shown in salmon color) detected in pulse oximetry

atonia. As expected, the frequency of OSAS tends to increase in individuals with brainstem lesions. However, studies indicate that the rise in OSAS frequency is associated with low Expanded Disability Status Scale (EDSS) scores rather than with brainstem lesions.[13-15] CSAS, which occurs in 1% of the healthy population, is 8 times more prevalent in patients with MS. Although CSAS is more common in pontine and mesencephalic lesions, as expected, this increase in frequency was not found in medullary lesions.[16-18]

The coexistence of apnea and MS has been determined to be directly related to fatigue, daytime sleepiness, neurocognitive dysfunction, and falls.[19] Therefore, sleep disturbance should be considered in patients experiencing falls that are unrelated to the EDSS score.

Even when subjective scales are used to diagnose apnea, the literature indicates that this approach is not very objective for patients with MS. The treatment of apnea primarily involves the application of positive airway pressure (PAP). In the study conducted in the polysomnography (PSG) unit, the appropriate

pressure level (continuous or bi-level) of PAP pressure that will decrease apneas and hypopneas and prevent the patient's oxygen desaturation is identified. Subsequently, the patient is instructed to use the PAP device at this optimal pressure. It has been observed that fatigue levels decrease after PAP application in patients with MS with moderate apnea.[20] In another case series, a decrease in neurocognitive dysfunction was observed after PAP treatment in patients with MS with apnea.[21] Apart from PAP, treatment options include reducing body weight, cessation of smoking and alcohol consumption, using orthoses to relax the airway, and surgical intervention for cases unresponsive to PAP.[19]

Central Disorders of Hypersomnolence: It is characterized by the existence of daytime sleepiness without nighttime sleep disorders or circadian rhythm sleep disorders. In general, the examination encompasses narcolepsy types I and II, idiopathic hypersomnia, Klein-Levin syndrome (characterized by disinhibition and eating disorder between long sleep attacks), other causes of hypersomnolence, insufficient sleep syndrome, isolated symptoms, and normal variants (such as long sleepers who sleep for more than 10 hours). Subjective scales that evaluate daytime sleepiness and the multiple sleep latency test performed in the PSG laboratory are helpful in diagnosis.

It has been reported that central hypersomnolence is observed in 53% of patients with MS experiencing daytime sleepiness and fatigue.[22] Narcolepsy is six times more prevalent in patients with MS.[23] Owing to the restricted quantity of cases, literature information is primarily based on case reports.[24,25] The treatment approach does not vary from that of patients without MS.

Circadian Rhythm Sleep-wake Disorders: It is basically examined under the headings of delayed sleep-wake phase disorder, advanced phase sleep syndrome, irregular sleep-wake rhythm disorder, non-24-hour sleep-wake disorder, other causes of hypersomnolence, shift work disorder, and jet lag. Generally, delayed sleep-wake phase disorder is more common in adolescence, while it is more widespread in the geriatric age group. Shift work disorder is most commonly observed in health and safety workers. Demyelination of the afferents and efferents of the suprachiasmatic nucleus may be attributed to the effects on the anatomical structures responsible for regulating the circadian rhythm and can be observed in 30% of patients with MS.[19] Compliance with sleep hygiene is a fundamental element of the therapeutic approach. Bright light therapy is a widely used treatment option, particularly in Scandinavian countries. Melatonin remains a viable therapeutic option; however, its bioavailability is significantly reduced to approximately 20% due to the effects of hepatic first-pass metabolism.[26]

Parasomnia: Parasomnia refers to undesirable physical events or behaviors occurring during sleep onset, sleep, or awakening. Parasomnias are classified into two main categories based on sleep phase: REM sleep or NREM sleep. Relatively common NREM parasomnias include confusional arousal, somnambulism, pavor nocturnus, and sleep-related eating disorder. REM Parasomnias include REM sleep behavior disorder, recurrent isolated sleep paralysis, and nightmare disorder. Among others, these include exploding head syndrome, sleep-related hallucinations, enuresis, and sleep talking.

Almost all of the sleep disorders categorized under this heading are not more prevalent in patients with MS compared to those without MS, and the treatment approach remains unchanged. However, if new complaints occur even though there is no sleep disorder in the premorbid condition, a new lesion should be searched for through cerebral imaging. Only REM sleep behavior disorder is observed six times more frequently in patients with MS. The fact that the lesion is anatomically close to the pedunculopontine nucleus suggests that glutamatergic neurons located in the pontine sublateral dorsal nucleus are responsible for its pathology.[17]

Sleep-related Movement Disorders: This is the primary sleep disorder most commonly reported by patients. RLS, periodic limb movement disorder (PLMD), isolated symptoms, and normal variants (excessive fragmentary myoclonus, hypnagogic foot tremor, hypnic jerks) fall under this category.

RLS is clearly distinguished from PLMD in that it occurs while awake rather than asleep (Figure 3). It is twice as common in patients with MS compared to the healthy population. In some studies, frequencies of up to 65% have been reported.[17] Although iron deficiency constitutes the predominant etiology of RLS, in secondary RLS patients do not support this association. Hypothetically, the condition is common in patients whose descending dopaminergic pathways are affected.[27-29] Advanced age, high EDSS scores, and cervical cord lesions are more common in patients, but their presence is not associated with the severity of RLS. Patients with MS who have RLS exhibit higher fatigue scores. It occurs concurrently with the diagnosis of MS in half of the patients. However, in the other half, it begins before the disease. It is considered that this may be related to spinal involvement, which does not cause clinical findings before diagnosis. Pathological changes in the spinal cord that current imaging methods cannot detect may trigger RLS.[30]

Low-dose evening treatment with dopamine agonists (e.g., pramipexole, ropinirole) is recommended for patients with MS diagnosed with RLS. If iron deficiency is present, replacement is recommended. Antiemetics, antipsychotics, antidepressants, and antihistamines, among other symptomatic agents used by the patient, can exacerbate RLS symptoms. Discontinuing these medications may be enough to alleviate the symptoms.

Periodic leg movements during sleep are more common in patients with MS (Figure 4). It is posited to originate from decreased supraspinal inhibitory control. However, when accompanied by RLS, it creates an indication for treatment.[17] Excessive fragmentary myoclonus may occur due to increased excitability of spinal segmental motor generators.[17]

The coexistence of sleep disorders and MS is now widely accepted based on numerous studies. Sleep disorders are observed in 24% to 62% of individuals diagnosed with MS. Sleep disruption exerts a negative influence on the comprehensive quality of life, both individually and cumulatively, by increasing

fatigue, depression, and disability.[31] A vicious cycle occurs as fatigue, depression, and disability also affect sleep, leading to insomnia and daytime sleepiness.[32] It is inevitable that cognitive deterioration will also become part of this vicious circle.

Do attacks, disease-modifying treatments, or symptomatic treatments in MS have an effect on sleep, or do sleep disorders trigger attacks in MS? Interferon beta, methylprednisolone, selective serotonin reuptake inhibitors, modafinil, and methylphenidate have sleep-decreasing effects, while baclofen, tizanidine, gabapentin, oxybutynin, and carbamazepine have sleep-promoting effects.[33,34] It has been reported that sleep disturbance can increase attacks by 1.75 times.[35]

Conclusion

Sleep disorders and MS, whether experienced individually or concurrently, negatively impact the patients' quality of life. In the long journey of MS, which develops on an autoimmune basis and managed through disease-modifying and symptomatic treatments rather than curative ones, it is crucial in clinical practice to inquire about our patients' sleep disorders, identify any issues, and implement appropriate interventions to minimize the patient's disability. Of course, correct questioning is only possible by recognizing sleep disorders. In this study, sleep disorders are evaluated under different headings, and their relationship with MS is discussed based on current literature data.

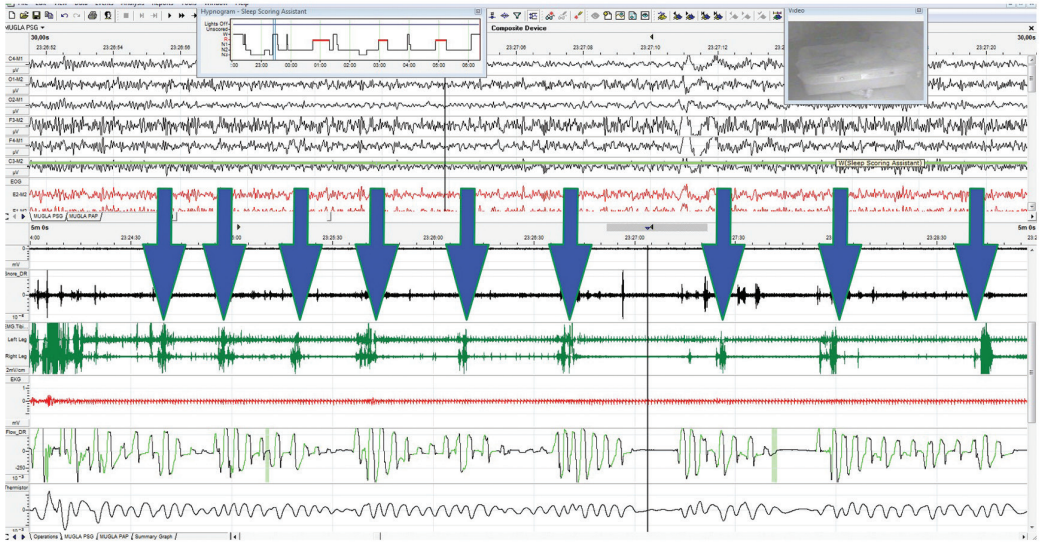

Figure 3. In the polysomnography recording, the epoch showing leg movements characteristic of restless legs syndrome (indicated by the dark blue arrow) recorded from the anterior tibial muscle electrodes (shown in green) while the patient is awake is observed

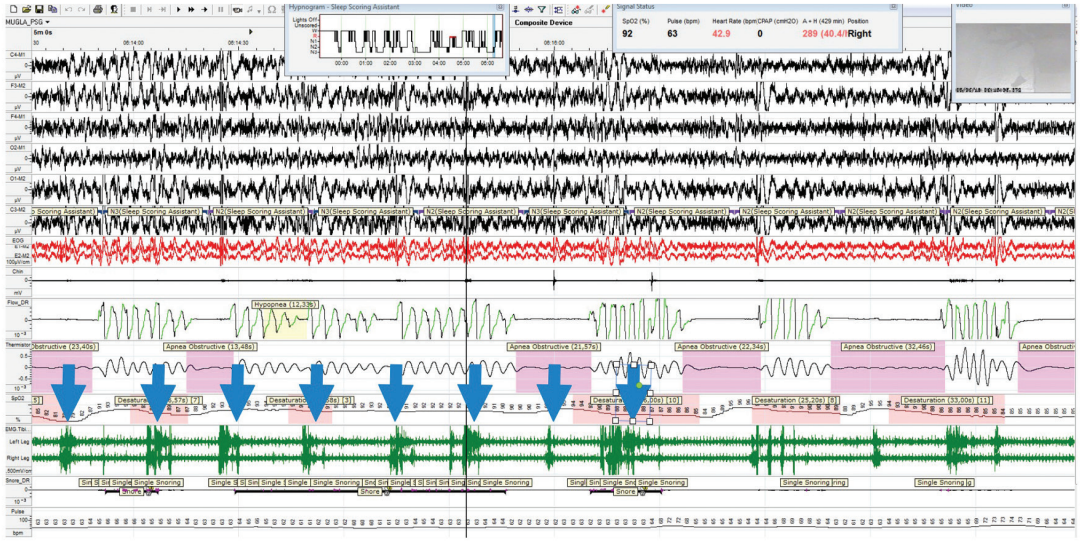

Figure 4. In the polysomnography recording, the epoch shows the leg movements observed during the periodic leg movements of sleep (blue arrow) recorded from the anterior tibial muscle electrodes (green color) while the patient was asleep

Footnotes

Authorship Contributions

Concept: İ.Ö., S.B., G.K., Design: İ.Ö., S.B., G.K., Data Collection or Processing: İ.Ö., S.B., G.K., Analysis or Interpretation: İ.Ö., S.B., G.K., Literature Search: İ.Ö., S.B., G.K., Writing: İ.Ö., S.B., G.K.

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