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Review

Sleep Disturbance: Impact on Psychiatric Disorders

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ABSTRACT

Sleep is one of the essential requirements for maintaining human mental and cognitive function. It is not merely a condition of rest, as was previously believed. Instead, it is composed of several stages and active processes. As new sleep processes are discovered, these factors have increased concern regarding the connection between sleep and diseases. As a consequence of the research concluded, it has become known that sleep, as it comprises standard processes, has a significant role in internal and psychological disorders. It has been demonstrated to be a prognostic, diagnostic, or predictive factor in Alzheimer's disease (AD), significant depression, and anxiety disorders. Major depression also displayed a higher level of specificity than other psychiatric disorders. It has been demonstrated to play a role in other mental conditions such as schizophrenia, attention deficit hyperactivity disorder (ADHD), and autism spectrum disorder (ASD). However, research is still ongoing to elucidate its precise role in these conditions. In this review, relationship between sleep disturbance and schizophrenia, ADHD, AD, ASD, anxiety, and major depressive disorder were discussed.

Keywords: Alzheimer's disease, anxiety, attention deficit hyperactivity disorder, autism spectrum disorder, major depressive disorder, sleep disorders

Sleep is the responsibility of the organism, which is characterized by typical posture; it can be summarized as a decrease in response to stimuli and minimal movement. Contrary to popular opinion, Moruzzi and Magoun[1] found in a significant study conducted in the 1950s that sleep is not the brain's resting state. Despite many studies, there is still a multitude of unresolved questions regarding the mechanism of sleep. This complex process is susceptible to several diseases via different pathways. Other psychological-behavioral disorders may come as a result of or be a causative factor affecting sleep quality.[2]

SLEEP - WAKEFULNESS CYCLE

Circadian rhythm is the mechanism that regulates the sleep-wakefulness cycle, mainly under the influence of sunlight. However, this system is also fed by additional elements like eating, alarm noises, and regular activities. Adrenocorticotropic hormone, norepinephrine (NE), melatonin, and prolactin, which are essential for the body's critical activities and the induction and maintenance of sleep, are some of the hormones that regulate sleep.[3-5]

There are five general phases to the sleep-wakefulness cycle: Wakefulness, N1, N2, N3, and N4. A typical period takes 90 to 100 minutes. It reaches rapid eye movement (REM) sleep stages N1, N2, N3, and N4, respectively. Repeatedly during the rest of the night, the REM period gets progressively shorter. Non-rapid eye movement (NREM) phases are N1, N2, and N3, and REM phases are N4.[4]

The N1 stage is the lightest sleep state, commonly termed the hypnagogic stage. In this stage, hypnic twitches such as audio-visual hallucinations, and a sense of falling from a height are likely to occur.[6] The N2 phase lasts the longest and serves as a bridge to the deeper stages of sleep. It comprises nearly half of the cycle.[4] The N3 phase of sleep is the deepest

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and most challenging to awaken from. Gait disorders and sleep talking are connected to the N3 stage. This phase speeds up the bone and muscle tissue cycle as the body's immune system develops, but as we get older, the period lasts for a shorter duration. However, if muscle control is still in progress, we do the same movements in real life as we did in the dream. When brain waves are viewed with electroencephalography, the waves seen while awake are also seen in this phase, so the brain acts as if it is awake, only there is no body control. Many cognitive and psychological processes, such as the regulation of memory, memories, inputs to the brain during the day, and the establishment of new neuronal connections, which are seen as the physiological purpose of sleep and dreaming, occur at this stage.[4,5,7]

SUBSTANCES AND REGIONS IN THE SLEEP-WAKEFULNESS SYSTEM

The reticular formation consists of neurons that lie between the fibers that pass through the central nucleus of the brain stem. According to the study by French and Magoun[8], even in the presence of intact sensory input, cortical activation and behavioral alertness are not maintained after the destruction of this area. After this study, it was understood that reticular formation has vital mechanisms in maintaining the wakefulness state of the organism and the studies on the sleep-wakefulness system gained momentum.

Over the decades, it has been understood that the wake-awareness system does not consist of only cycles. Many neurotransmitter substances secreted from some brain regions also play a primary role in sleep.[9,10] These substances can be divided into wake-promoting neurotransmitters and sleep-promoting neurotransmitters. Essential substances that provide wakefulness; can be summarized as acetylcholine (ACh), NE, dopamine (DA), histamine (His), serotonin (5-HT), and hypocretin/orexin (HCRT). The main sleep-stimulating substances are; gamma-aminobutyric acid (GABA), ACh, melanocyte-stimulating hormone (MCH), and various non-neurotransmitters.[7,11]

Acetylcholine is secreted from the basal forebrain region and the lateral tegmental region.[12] It is the neurotransmitter that contributes the highest to cortical stimulation while awake and during REM sleep; this is its role in the sleep-stimulating system. Norepinephrine is secreted from the locus coeruleus and almost never stays during REM sleep after reaching its highest level at wakefulness and gradually decreasing. In moments of stress and obvious attention, the firing of neurons in this area increases markedly. Dopamine is an important neurotransmitter in the wakefulness system as well as systems such as motor control and learning reward. The sedative effects seen as a result of the use of D2 receptor agonists in treating Parkinson's disease and restless legs syndrome also support this situation. While histamine is secreted chiefly while awake, its effect on alertness is most evident in the use of first-generation antihistamine allergic drugs. Patients using these drugs tend to sleep without a change in sleep quality or structure. Serotonin, dorsal raphe nucleus (DRN), which is in the midline of the brain stem, is the main neuronal population responsible for sleep-wakefulness control. Most regions involved in the sleep-wakefulness cycle, including the basal forebrain, preoptic area, and hypothalamus, receive input from the DRN. Hypocretin plays a role in the transition between the two states in the sleep-wakefulness cycle. However, it is also secreted in high amounts in many behavioral events such as personal care, nutrition, and discovery.[7]

Each of the substances that provide alertness can provide this situation independently. The fact that there are different and many systems from each other are explained in two ways. The first proposes from an evolutionary point of view that many systems have evolved so that damage to any given system does not affect the entire sleep-wakefulness system but remains minimal. The second suggests that each arousal system maintains different states of alertness. For example, in stressful or new environments, NE and His, contribute to the wakefulness cycle by increasing attention, while DA is associated with limbic system-linked reward-driven behaviors. HCRT, on the other hand, plays a critical role in waking up from sleep. Despite these different tasks, as we mentioned before, the individual activation of these systems ensures wakefulness through the stimulation of the thalamus and cortex.[6,7]

The sleep-stimulating system is controlled by inhibitory secretions, GABA and alanine, of the ventrolateral preoptic nucleus (VLPO) and median preoptic areas. It simply stimulates sleep by inhibiting neurotransmitters involved in the wakefulness system. Normally, VLPO neurons are inhibited by system secretions that provide wakefulness. During sleep, VLPO stops cortical activation by inhibiting the wakefulness system with GABA and alanine secretions. This inhibition thoroughly reduces the

inhibition of the VLPO and increases the inhibition of the wakefulness system even more. Thus, sleep sustainability is ensured. At the same time, the circadian rhythm is predominantly influenced by GABA stimuli. GABA also plays a role in ensuring REM sleep. Given all this, many sedative/hypnotic drugs exert efficacy through the GABA-A receptor. In addition to ACh, and MCH, dozens of substances that play a role in sleep, which are not neurotransmitters classified as somnogenes, also play an important role.[5,7]

As seen, the sleep-wakefulness system is spread over a large area of the brain and is controlled by dozens of different endogenous-exogenous stimuli. This situation results in many diseases affecting the sleep-wakefulness cycle relatively easily. In the same way, a disorder in the sleep-wakefulness system can lead to many diseases.[13,14]

Having studies being conducted in the context of psychiatric disorders and sleep, this two-way connection is becoming more and more apparent. It has been recognized that the type and quality of sleep have a significant role in the development of the brain, mental health, and propensity for psychiatric disorders, particularly in studies involving the childhood-adolescent age group.[15-17]

MAJOR DEPRESSIVE DISORDER

Major depressive disorder (MDD) is a condition in which sleep issues are frequent. Numerous polysomnographic investigations on changes in sleep architecture or quality among mental disorders have been undertaken, and they provide some significant, albeit general, findings.[16-19] There is a dual relationship between depression and sleep. Extant research has demonstrated that it should be assessed from multiple angles.[20-22]

Patients with insomnia are two to four times more likely to be depressed compared to patients with normal sleep patterns. Likewise, patients diagnosed with MDD have a higher risk of suffering from a sleep disorder either at the beginning or in the later stages of the disease.[20,23,24] Some studies supported by these findings suggest that insomnia may be beneficial in the prediction of MDD. In contrast, relatively large numbers of studies suggest that emphasize that it gives clues about the severity, course, and recurrence of the disease rather than prediction.[25,26] A reason for this is that insomnia is a comorbidity that can be seen in many diseases (especially substance use disorder, anxiety, behavioral disorders, and psychiatric disorders such as MDD, but it also shows comorbidity in many other diseases).[21]

It has been observed that this relationship continues significantly even in patients diagnosed without hypersomnia or insomnia in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria used in the diagnosis of MDD to prevent definitional contamination, and sleep disorders may develop in some patients over time. While this two-way interaction was more pronounced in insomnia alone, hypersomnia did not show any substantial or robust links. Significant and higher specificity relations were observed in the coexistence of these two diseases.[22,24,27]

In 2016, Baglioni et al.[18] according to a comprehensive meta-analysis study on major depression, sleep problems are associated with sleep continuity, depth, and REM intensity. The rapid eye movement latency, REM intensity, and REM duration change together, making these findings more specific for MDD. It is even thought that changes in REM intensity may herald an episode of major depression through changes in various neuronal connections in the central nervous system. Moreover, in studies conducted with subgroups to reduce bias, it was observed that REM delay and REM duration did not change in depressed patients without comorbidity. In contrast, increased REM intensity persisted even in this situation. According to a comprehensive meta-analysis study conducted by Baglioni et al.[18] in 2016, sleep problems in MDD were associated with sleep continuity, depth, and REM intensity. The co-variation of REM latency, intensity, and duration makes these findings more specific for MDD. It is even thought that changes in REM intensity may herald an episode of major depression through changes in various neuronal connections in the central nervous system.

Moreover, in studies conducted with subgroups to reduce bias, it was observed that REM delay and REM duration did not change in depressed patients without comorbidity, whereas increased REM intensity persisted even in this situation. This suggests that it may be a relatively specific finding. Despite the results of the extensive study above, in another study conducted in 2008 with pediatric and pubertal patients[25], the results of polysomnography (PSG) give contradictory findings. In fact, patients with depression and severe sleep problems showed the highest sleep quality in PSG results. In addition, significant differences were also seen in gender-based comparisons that may be related to circadian rhythm

and hormones.[29]

One crucial aspect that should not be overlooked needs to be emphasized: Investigations, studies, or experiments that look at the connections between sleep and the disorders covered by this study have significant limitations. It is well established that these disorders are not borderline conditions, that different studies employ different definitions and diagnostic standards, and that numerous biopsychosocial factors influence them (social relations, income level, occupation, age, gender, light exposure, sleep quality, genetic-epigenetic factors, etc.). For example, a review examining the effect of food allergy on psychiatric disorders reported that sleep is deeply affected due to the overactivation of the cholinergic system by food allergy in patients with MDD.[28] Due to the small number of studies that exclude controllable factors, which are only a part of these and other factors, disparate and conflicting results are also remarkably present in the literature.[18,26]

AUTISM SPECTRUM DISORDER

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by repetitive behavior disorder and social communication problems.[29] Many comorbid conditions such as sleep problems, speech disorders, anxiety, depression, and motor problems are also seen in their course.[30] Its prevalence has increased steadily over the past 40 years. Although there is no epidemiological study in Turkey, it was thought to be one in 10,000 in the United States in 1980, but it increased to one in 59 in 2018. This is attributed to several variables, including increased societal awareness, easier diagnosis, and the growth of preventive diagnosis facilities like child psychiatry.[31,32]

Since there are fewer longitudinal studies on sleep disorders in ASD than cross-sectional studies, it is not easy to evaluate sleep disturbance with complete accuracy in ASD. However, sleep problems have been reported in 50-80% of children with ASD. In many studies, more than 50% of the problems of starting to sleep and diving were seen. Sleepwalking, night terrors, and nightmares are seen at a much lower rate. In several studies on sleep architecture, REM-related disorders have been generally detected in ASD.[33]

Most ASD patients experience sleep issues permanently, and it has been shown that over half of ASD patients who have reached adolescence still experience sleep issues. In contrast to pharmacotherapeutic therapies, behavioral techniques are known to be effective if used consistently. However, there is no single medicine that the Food and Drug Administration has licensed for use in patients with ASD.[32,34,35] There is a need for high-level studies in many areas and perspectives on this subject in the literature, as can be seen in the study published in 2020 by Cortese et al.[36] However, knowing that sleep issues and their influence can be controlled in people with ASD will improve patients' quality of life.

ALZHEIMER'S DISEASE

Dementia is a group of neurodegenerative disorders in which memory loss is at the forefront, communication disorders, lack of information processing, sleep problems, mood disorders, and even motor functions are affected in the later stages.[37,38] There are approximately 24 million dementia patients. The most common cause of dementia is Alzheimer's disease (AD), which accounts for an average of 60-80%.[39,40]

The role of sleep in memory has been known for many years. Only the mechanisms of action have not yet been fully elucidated. The dominant theory is that new memories are perpetuated and adapted to long-term memory. Another theory claims that the transition between slow-wave sleep (SWS) and REM sleep provides this condition. Studies on rats have shown that sleep interruption from the SWS stage to the REM phase negatively affects memory. However, no such effect has been observed in cases where the REM phase follows SWS.[14]

Sleep problems are often seen in AD. The fact that there is a two-way interaction between sleep problems and AD is coming to the fore with an increasing number of shreds of evidence day by day.[41-43] The clustering of the amyloid-beta (Aβ) protein, which has an important role in the etiopathogenesis of AD, begins 15-20 years before the onset of AD on average. Likewise, sleep problems can be seen as a predictive factor before the onset of the disease. A strong correlation has been shown between delayed napping time, poor sleep hygiene, short sleep duration, and Aβ accumulation.[44] This correlation has even been observed in one-night sleep deprivation.[14,45] Although it is obvious that the level of Aβ has a relationship with waking time, there is a possibility that many mechanisms cause it. For example, the accumulation of Aβ in the regulatory regions of the sleep-wake system at various brain points can cause sleep problems in AD. However, due to many animal models and studies on humans, the

fact that this relationship is two-way seems to be the most plausible explanation.[45-49]

A meta-analysis study of 69216 patient data published in 2017 showed that individuals with sleep problems had a 1.7-3.8 times higher risk of developing AD. In the same study, it is reported that sleep problems are seen in 15% of AD patients, and it is thought that sleep may be a factor that has at least a 15% effect on the etiology of AD.[48]

ANXIETY

Sleep problems, for example, can occur in combination with insomnia and anxiety (38%) as a cause (18%) or a consequence of anxiety disorders (44%).[50] Likewise, anxiety was seen in 30% of patients with insomnia. Furthermore, there are many subheadings of these sleep problems and anxiety disorders, and the results vary greatly according to these subheadings. For instance, anxiety disorders are classified under about 10 headings according to the DSM-V criteria, and in some diseases, such as posttraumatic stress disorder, sleep problems are a diagnostic criterion and in others, they are not included in the diagnostic criteria.[51] However, according to patient statements with anxiety disorders, close to 75% of patients with anxiety disorders suffer from sleep problems.[23,50] Even if there were not enough polysomnography or actigraphy studies focused on objective data in this area, there was no specific finding of anxiety disorders in those who did. Delay in falling into REM and sleep, shortening of their duration, and shortening of SWS were observed.[22,25,52]

Most patients with subjective or objective signs of sleep problems are diagnosed with generalized anxiety disorder and posttraumatic stress disorder. Furthermore, sleep deprivation is already a symptom of these diseases.

In studies conducted with children, it has been observed that 85% of children with anxiety disorders experience sleep problems at least once, and 50% have chronic sleep problems. In a large-scale controlled study, a group diagnosed with anxiety disorder and a second group diagnosed with major depression were compared with a control group. The PSG findings mentioned above were higher in the anxiety group than in the major depressive disorder group.[25] On the other hand, it can be said that the available data are insufficient due to the limited number of studies focused on objective findings, the limited and non-specific findings, and the fact that anxiety disorders are a topic that includes many different diseases. The limitations here affect the results of the studies more than in many diseases because many psychiatric disorders contain anxiety-based problems in themselves. Thus, it becomes very difficult to associate sleep with anxiety as the sole cause directly.[53] For example, during childhood, sleep can be caused by environmental factors, parental upbringing, traumas, etc. It is known that it is easily affected, and if not treated, this condition becomes chronic. Likewise, it has been stated that sleep problems and anxiety in children with anxiety disorder occur due to environmental factors.[27] Therefore, studies examining the relationship between anxiety disorders and sleep problems should pay more attention to limitations.

ATTENTION DEFICIT HYPERACTIVITY DISORDER

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder that can be permanent and manifests primarily with distractibility, hyperactivity, and/or impulsivity. It is one of the most common psychiatric diseases, especially in childhood, and is seen in 5% of children.[54,55]

It is reported that children and adolescents with attention deficit hyperactivity disorder experience sleep problems in 25-50% of cases, according to subjective complaints and parental notifications.[56,57] Cortese et al.[58] examined sleep research published in the literature between 1987 and 2008. A meta-analysis study was published on samples with attention deficit hyperactivity disorder but not receiving drug treatment and without comorbidities. According to this study, ADHD patients were found to have difficulty in falling asleep, waking up, resistance to lying down, and daytime sleepiness significantly different from the controls. In terms of objective findings, sleep initiation delay and sleep stage changes were significantly higher than the controls, and the time to fall asleep and the actual sleep time were significantly lower than those of the controls. Similar results have been seen in other studies in the literature.[59,60] It has also been associated with various comorbid conditions such as restless legs syndrome, obstructive sleep apnea (OSA), and circadian rhythm disturbance.[61,62]

The results of the studies in which objective findings are examined are more diverse than those in which subjective findings are examined. One of the important reasons for this may be that the populations in the studies are different from the disease subgroup,

that is, the patient characteristics and many features of the studies are different, especially in the foreground that there is no common methodology of the studies.[63] For example, as mentioned above, OSA causes sleep problems as ADHD comorbidity in children. However, no comprehensible conclusion has emerged when this relationship is examined in adults.[64-66] Another study compared ADHD and anxiety disorders from a sleep perspective, and it was found that anxiety-directed treatment reduced sleep problems.[67-68] According to a 2018 review, 50% of children diagnosed with a breathing-related sleep disorder (BRSD) and ADHD did not meet the DSM-IV criteria for ADHD a year after BRSD treatment.[69]

SCHIZOPHRENIA

Schizophrenia is a complex disorder that negatively affects the lives of individuals and their function in society.[70] There are not yet enough studies to say that sleep disorders in schizophrenia patients are significant. In the last decade, there have been several small specific studies and review articles with increasing interest in this topic. One shows that schizophrenia patients with impaired circadian rhythm give more negative results on cognitive tests than those who do not.[71] The results reached in most of the studies, in general, are similar: unchanged REM duration, difficulty falling asleep, maintenance problems, REM latency, and SWS. On the other hand, there are studies suggesting that sleep disorders in schizophrenia patients may be useful in endophenotype.[72-74] However, it is seen that these data are obtained from a limited number of studies in which dozens of limiting factors cannot be excluded. It should not be forgotten that many factors such as the stage of the disease, its characteristics, the effects of the drugs used, neurotransmitter abnormalities such as DA, and GABA, and positive, negative, and cognitive symptoms in patients with schizophrenia may affect sleep architecture. In fact, according to a comprehensive meta-analysis study published in 2016, it is stated that a significant relationship between sleep disorders and schizophrenia cannot be established in schizophrenia patients, especially in the absence of comorbidities such as depression.[17]

In light of the available data, it does not yet seem possible to establish a relationship between sleep architecture/disorders and schizophrenia. Long-term and comprehensive studies in which the above limiting factors are excluded are needed.

In conclusion, it has been known for many years that sleep and psychiatric disorders overlap pathophysiologically in many points such as hormones, mechanisms, brain regions in charge, etc. In medical practice, complaints about sleep problems are underestimated, and their predictive, prognostic, or diagnostic side is often overlooked. However, numerous studies over the last 50 years have clarified the relationship between sleep disorders and diseases in a variety of ways. Its predictive and/or prognostic significance has been shown, especially in MDD and AD. Even highly specific objective findings were observed in polysomnographic test results in MDD patients. In AD, sleep problems are considered a risk factor in 15% of cases. The risk of AD in individuals with sleep problems is 1.7-3.8 times higher than in the normal population. In both of these diseases, the relationship with sleep problems is bidirectional. In other words, sleep disorders offer a prediction in the diagnosis of the disease and can be seen in the course of the disease. In autism spectrum disorder, sleep problems come to the fore in treatment and the course of the disease. It is now known that if the sleep problems of these individuals are taken into account in the treatment process, positive results are obtained. In anxiety disorders and ADHD patients, sleep problems manifest themselves as an important comorbidity that affects the quality of life and activities of daily living. Similarly, it is recognized that it is an important factor that improves the quality of life of patients when considered in the treatment and progression of the disease. There was no psychiatric disorder with a PSG result specific enough to make a diagnosis, nor were there two diseases with identical PSG results. This situation demonstrates the significance of sleep problems in various issues, including mental disorder diagnosis and treatment, as well as patient quality of life. Sleep problems should be considered an important factor in medical practice as a result of future large-scale studies on the subject.

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