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CLINICAL FEATURES OF MIGRAINE IN PATIENTS WITH SLEEP DISTURBANCES


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УЙҚУ БУЗИЛИШИ БЎЛГАН БЕМОРЛАРДА МИГРЕННИНГ КЛИНИК КЎРИНИШЛАРИ

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КЛИНИЧЕСКИЕ ПРОЯВЛЕНИЯ МИГРЕНИ У ПАЦИЕНТОВ С НАРУШЕНИЯМИ СНА

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Резюме. *Мигрень ўртача ёки кучли бош оғриғи хуружлари, кўнгил айниши, қусиш, ёруғлик ва товушга сезгирлик ҳамда кундалик фаолиятнинг пасайиши билан намоён бўладиган кенг тарқалган неврологик касаллик ҳисобланади. Сўнгги йилларда мигрень ва уйқу бузилишлари ўртасидаги боғлиқликка катта эътибор қаратилмоқда, чунки уйқусизлик, обструктив уйқу апноэси, безовта оёқлар синдроми ва циркад ритм бузилишлари эпизодик ҳамда сурункали мигрень билан оғриган беморларда кўп учрайди. Ушбу шарҳнинг мақсади мигрень ва уйқу бузилишлари ўртасидаги клиник ва патофизиологик боғлиқликни, умумий нейробиологик механизмларни, касалликнинг хронизациясини, даволашга жавобни ва комплекс терапия имкониятларини таҳлил қилишдан иборат. Адабиётлар маълумотлари уйқу бузилишлари мигрень хуружларини қўзғатувчи ва давом эттирувчи омил бўлиши мумкинлигини, мигрень эса ўз навбатида уйқу сифатини янада ёмонлаштиришини кўрсатади. Асосий механизмлар қаторига гипоталамус дисфункцияси, циркад ритм бузилиши, мелатонин танқислиги, серотонин ва дофамин тизимлари номутаносиблиги, кортикал гиперқўзғалувчанлик ва марказий сенситизация киради. Шу сабабли мигрень билан оғриган беморларда уйқу сифатини мунтазам баҳолаш ва даволаш режасига уйқуни яхшилашга қаратилган чораларни киритиш муҳим аҳамиятга эга.*

Калит сўзлар: *мигрень, уйқу бузилишлари, уйқусизлик, обструктив уйқу апноэси, безовта оёқлар синдроми, циркад ритм, мелатонин, серотонин, дофамин, когнитив-хулқий терапия.*

Abstract. *Migraine is a highly prevalent neurological disorder characterized by recurrent attacks of moderate to severe headache, frequently accompanied by nausea, vomiting, photophobia, phonophobia and functional disability. In recent years, increasing scientific attention has been paid to the relationship between migraine and sleep disorders, since insomnia, obstructive sleep apnea, restless legs syndrome and circadian rhythm disturbances are commonly reported in patients with episodic and chronic migraine. The aim of this review is to analyze the clinical and pathophysiological relationship between migraine and sleep disorders, with particular attention to shared neurobiological mechanisms, disease chronification, treatment response and possibilities for integrated management. Current literature indicates that sleep disturbances may act both as triggers and maintaining factors of migraine attacks, while recurrent migraine pain may further impair sleep quality, forming a bidirectional pathological cycle. Hypothalamic dysfunction, circadian rhythm disruption, melatonin deficiency, serotonergic and dopaminergic imbalance, cortical hyperexcitability and central sensitization are considered key mechanisms linking both conditions. The available evidence supports the need for routine assessment of sleep quality in migraine patients and the inclusion of sleep-focused interventions, including sleep hygiene, cognitive behavioral therapy for insomnia, treatment of obstructive sleep apnea and rational pharmacological correction. A comprehensive approach involving neurologists, sleep specialists and behavioral therapists may improve headache control, reduce disability and enhance quality of life.*

Keywords: *migraine, sleep disorders, insomnia, obstructive sleep apnea, restless legs syndrome, circadian rhythm, melatonin, serotonin, dopamine, cognitive behavioral therapy.*

Migraine is one of the most common disabling neurological disorders and represents an important medical, social and economic problem worldwide. It is characterized by recurrent headache attacks, usually of moderate or severe intensity, often unilateral and pulsating in nature, and accompanied by nausea, vomiting, photophobia, phonophobia and aggravation by routine physical activity. According to Amiri et al. [1], migraine has a complex epidemiological structure and is associated not only with neurological symptoms, but also with multiple comorbid conditions that influence disease severity, disability and long-term prognosis. Among these comorbidities, sleep disorders occupy a particularly important place because

they affect both the occurrence and the clinical course of migraine.

The relationship between migraine and sleep disturbances is clinically significant because sleep is one of the major regulators of brain excitability, pain modulation, autonomic tone and neuroendocrine balance. Tiseo et al. [9], in a systematic review, emphasized that migraine is frequently associated with insomnia, poor sleep quality, obstructive sleep apnea, restless legs syndrome and circadian rhythm disturbances. These associations are not accidental, since migraine and sleep disorders share several neurophysiological pathways. Vgontzas and Pavlovic [10] noted that sleep disorders and migraine may influence one another through hypothalamic dysfunction, neurotransmitter imbalance, altered pain processing and dysregulation of arousal systems.

One of the most important aspects of this relationship is its bidirectional character. Poor sleep quality, sleep deprivation, irregular sleep schedules and fragmented sleep may increase the probability of migraine attacks. At the same time, migraine attacks themselves may disturb sleep by causing nocturnal awakening, anticipatory anxiety, pain-related hyperarousal and postdromal fatigue. Buse et al. [4], using data from the Chronic Migraine Epidemiology and Outcomes study, demonstrated that sleep disorders are common among individuals with migraine and are particularly relevant in patients with higher headache frequency and greater disease burden. This finding supports the view that sleep disturbances are not simply accompanying symptoms, but clinically meaningful factors that may contribute to migraine chronification.

The hypothalamus is considered one of the central structures linking migraine and sleep regulation. It participates in circadian rhythm control, autonomic regulation, endocrine homeostasis, hunger, thirst, thermoregulation and arousal. Neurobiological studies suggest that hypothalamic activation may occur before the headache phase of migraine, which explains prodromal symptoms such as yawning, fatigue, mood changes, food craving and sleepiness. Since the hypothalamus also regulates sleep-wake cycles, its dysfunction may create a shared substrate for both migraine attacks and sleep instability. Vgontzas and Pavlovic [10] highlighted the importance of hypothalamic and brainstem mechanisms in the interaction between migraine and sleep disorders.

Circadian rhythm disruption is another important mechanism. Patients with irregular sleep-wake cycles, shift work, delayed sleep phase tendencies or inconsistent bedtime patterns may experience more frequent migraine attacks. Sleep irregularity can alter melatonin secretion, cortisol rhythm, autonomic balance and cortical excitability. These changes may lower the threshold for migraine initiation. The clinical relevance of circadian mechanisms is supported by observations that migraine attacks often occur at particular times of day, especially in the early morning, and may be associated with changes in sleep architecture and REM sleep regulation [9,10].

Melatonin imbalance has received considerable attention in migraine research. Melatonin is a hormone secreted by the pineal gland and is involved in circadian rhythm synchronization, sleep initiation, antioxidant defense, anti-inflammatory activity and pain modulation. Reduced melatonin levels have been reported in some migraine patients, particularly in those with poor sleep quality or chronic migraine. A decrease in melatonin may contribute to sleep fragmentation, increased neuronal excitability and impaired endogenous pain inhibition. Therefore, melatonin is considered not only a marker of circadian dysregulation, but also a potential therapeutic agent in selected migraine patients with sleep disturbances.

Neurotransmitter dysfunction also plays a major role in the overlap between migraine and sleep pathology. Serotonin is involved in pain modulation, vascular regulation, mood, sleep initiation and REM sleep mechanisms. Serotonergic imbalance has long been implicated in migraine pathogenesis and may also contribute to insomnia and altered sleep architecture. Dopamine is another relevant neurotransmitter, especially because dopaminergic dysfunction is associated with migraine prodromal symptoms, nausea, yawning, mood changes and restless legs syndrome. The frequent coexistence of migraine and restless legs syndrome may therefore reflect a shared dopaminergic vulnerability [9,10].

Cortical hyperexcitability is a recognized feature of migraine, particularly in relation to sensory hypersensitivity and susceptibility to cortical spreading depolarization. Sleep deprivation may further increase cortical excitability and reduce inhibitory control within thalamocortical networks. This mechanism may explain why inadequate sleep often precedes migraine attacks and why patients with chronic insomnia may present with increased headache frequency. In addition, persistent sleep loss may enhance central sensitization, resulting in greater pain intensity, longer attack duration and reduced responsiveness to analgesic and preventive treatment.

Psychiatric comorbidity is another important clinical dimension. Migraine is frequently associated with depression, anxiety and other psychiatric conditions, which may independently impair sleep quality and increase headache-related disability. Bergman-Bock [2] described strong associations between migraine and common psychiatric comorbidities, while Buse et al. [3] emphasized that psychiatric comorbidities are relevant in both episodic and chronic migraine. Jette et al. [6] also confirmed, in a population-based study, that migraine is associated with psychiatric disorders at the national level. These findings are clinically important because insomnia, anxiety

and depression may form a mutually reinforcing triad with migraine, leading to greater disability and more difficult treatment.

McDermott et al. [7] demonstrated that migraine may coexist with psychiatric disorders in vulnerable clinical populations, showing that comorbidity patterns can substantially complicate disease management. In such patients, sleep disturbances may not be isolated symptoms, but part of a broader neuropsychological and behavioral syndrome. Therefore, evaluation of migraine patients should include not only headache characteristics, but also sleep quality, mood symptoms, anxiety level, medication use, substance use, daily routine and psychosocial stressors.

Insomnia is one of the most frequently reported sleep disorders in migraine patients. It may include difficulty falling asleep, frequent awakenings, early morning awakening or non-restorative sleep. Chronic insomnia increases stress system activation, sympathetic tone and inflammatory signaling, all of which may contribute to migraine susceptibility. Patients with insomnia often report more frequent headaches, longer attack duration and increased use of acute medications. This is clinically relevant because excessive use of analgesics or triptans may increase the risk of medication-overuse headache and further promote migraine chronification.

Obstructive sleep apnea is another clinically important condition associated with headache and migraine-like symptoms. OSA causes recurrent episodes of upper airway obstruction during sleep, resulting in intermittent hypoxia, sleep fragmentation, autonomic activation and morning headaches. Although not every patient with OSA has migraine, the presence of snoring, witnessed apneas, obesity, excessive daytime sleepiness and morning headache should prompt clinicians to consider sleep apnea assessment. Treatment with continuous positive airway pressure may improve sleep continuity, reduce hypoxic stress and, in some patients, decrease headache burden. This supports the need for targeted screening in migraine patients with suggestive symptoms.

Restless legs syndrome is also relevant to migraine because both conditions involve dopaminergic mechanisms and sleep disruption. RLS causes unpleasant sensations in the legs and an urge to move, usually worsening at rest and in the evening. This leads to difficulty falling asleep and fragmented sleep. Patients with both migraine and RLS may therefore experience worsening headache patterns due to chronic sleep deprivation. Recognition of RLS is important because specific treatment of this disorder may improve sleep continuity and indirectly reduce migraine susceptibility.

The clinical features of migraine in patients with sleep disorders often differ from those seen in patients without sleep disturbances. Such patients may have higher attack frequency, greater pain intensity, prolonged recovery, more prominent fatigue, increased daytime sleepiness and reduced treatment response. Buse et al. [5] demonstrated that comorbid and co-occurring conditions are associated with increased headache pain intensity and frequency in migraine patients. This supports the concept that comorbid sleep and psychiatric conditions amplify migraine burden and should be considered in clinical decision-making.

From a diagnostic point of view, migraine patients should be routinely questioned about sleep duration, sleep quality, snoring, witnessed apnea, restless legs symptoms, daytime sleepiness, shift work, bedtime variability, caffeine consumption, screen exposure and use of sedative or stimulating medications. Standardized tools such as the Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale and headache disability questionnaires may help quantify the burden of sleep disturbances and migraine-related impairment. This approach is consistent with modern patient-centered care, where comorbidity assessment is essential for individualized treatment planning.

The management of migraine in patients with sleep disorders requires an integrated strategy. Pharmacological treatment alone may be insufficient if sleep disturbances remain untreated. Lifestyle modification should include regular sleep timing, adequate sleep duration, limitation of caffeine and alcohol, reduction of evening screen exposure, relaxation techniques and avoidance of irregular sleep patterns. These interventions may appear simple, but they target core behavioral triggers that can influence circadian rhythm stability and migraine threshold.

Pharmacological management should be individualized. Preventive migraine medications may have different effects on sleep. Some agents may improve sleep continuity, while others may worsen insomnia or daytime fatigue. In patients with restless legs syndrome or obstructive sleep apnea, specific therapy for the sleep disorder is necessary rather than simply increasing migraine medication.

The role of patient education is also fundamental. Many patients are unaware that irregular sleep, insufficient sleep or excessive weekend sleep may trigger migraine attacks. Education should emphasize that both sleep deprivation and oversleeping may destabilize migraine control. Educational resources such as those provided by national health institutions also highlight the importance of recognizing headache triggers and seeking appropriate management [8].

A multidisciplinary approach is often required in patients with chronic migraine and severe sleep disturbances. Neurologists, sleep medicine specialists, psychologists and primary care physicians may all contribute to comprehensive care. Such collaboration is especially important when migraine coexists with psychiatric disorders, obstructive sleep apnea, chronic insomnia, medication overuse or significant

disability. Integrated care may reduce fragmentation of treatment and improve adherence to both migraine and sleep interventions.

Overall, the available literature strongly supports the clinical importance of the migraine-sleep relationship. Migraine and sleep disorders are connected through shared neurobiological mechanisms, including hypothalamic dysfunction, circadian rhythm disruption, neurotransmitter imbalance, melatonin changes, cortical hyperexcitability and central sensitization. Clinically, sleep disorders may increase migraine frequency, pain intensity, disability and risk of chronification. At the same time, migraine may worsen sleep quality and contribute to persistent fatigue and reduced quality of life.

Thus, the assessment of sleep should become a routine component of migraine care. Treatment strategies should be directed not only at suppressing headache attacks, but also at stabilizing sleep, correcting comorbid sleep disorders and improving behavioral regulation. Future studies should further investigate the long-term effects of targeted sleep interventions on migraine chronification, treatment response and quality of life. A better understanding of shared molecular and neurophysiological pathways may also support the development of therapies that simultaneously target migraine mechanisms and sleep regulation.

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КЛИНИЧЕСКИЕ ПРОЯВЛЕНИЯ МИГРЕНИ У ПАЦИЕНТОВ С НАРУШЕНИЯМИ СНА

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Резюме. *Мигрень представляет собой распространённое неврологическое заболевание, характеризующееся повторяющимися приступами умеренной или выраженной головной боли, часто сопровождающейся тошнотой, рвотой, фотофобией, фонофобией и снижением повседневной активности. В последние годы всё большее внимание уделяется взаимосвязи мигрени с нарушениями сна, поскольку бессонница, обструктивное апноэ сна, синдром беспокойных ног и расстройства циркадного ритма часто встречаются у пациентов с эпизодической и хронической мигренью. Целью данного обзора является анализ клинической и патофизиологической связи между мигренью и нарушениями сна с акцентом на общие нейробиологические механизмы, хронизацию заболевания, ответ на лечение и возможности комплексной терапии. Данные литературы свидетельствуют о том, что нарушения сна могут выступать как триггерами, так и факторами поддержания мигренозных приступов, тогда как повторяющиеся боли при мигрени дополнительно ухудшают качество сна. К ключевым механизмам относятся дисфункция гипоталамуса, нарушение циркадных ритмов, дефицит мелатонина, серотонинергический и дофаминергический дисбаланс, корковая гипервозбудимость и центральная сенситизация. Полученные данные подчёркивают необходимость регулярной оценки качества сна у пациентов с мигренью и включения в лечение мероприятий, направленных на коррекцию сна.*

Ключевые слова: *мигрень, нарушения сна, бессонница, обструктивное апноэ сна, синдром беспокойных ног, циркадный ритм, мелатонин, серотонин, дофамин, когнитивно-поведенческая терапия.*