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Review Article

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Sleep & Stroke - features of occurrence and course

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ABSTRACT

Cerebrovascular diseases, such as ischemic and hemorrhagic strokes, are among the leading causes of disability and mortality worldwide. Evidence shows us that the identification and treatment of sleep disorders should be included in both primary and secondary stroke prevention. Sleep and stroke are often intertwined because sleep disorders, including sleep-disordered breathing, parasomnias, sleep-related movement disorders, insomnia, and hypersomnia, are closely associated with comorbid cardiovascular disease and increase the risk of stroke. Sleep disturbances after stroke can also affect stroke rehabilitation and quality of life, and if left untreated can lead to recurrent strokes.

Keywords: Stroke, sleep, sleep disorders, sleep-disordered breathing, central sleep apnea, obstructive sleep apnea.

INTRODUCTION

Stroke is the second leading cause of death and the most important cause of disability in adult life, often having a major impact on the patient's daily life. More than 60 thousand cases of stroke (acute cerebrovascular accident) are registered annually in Uzbekistan.

At the same time, disability after a stroke is 83.8%, and the percentage of hospital mortality is 17.3% [1].

In European countries, ischemic stroke accounts for up to 87% of the structure of cerebral stroke. Globally, vascular diseases of the brain are the dominant causes of permanent disability and long periods of hospital treatment for patients, which causes obvious economic and social losses.

With a sleep duration of less than 6 hours, the risk of stroke increases by 32%, and by more than 8 hours – by 71% [2].

In addition to the traditional risk factors for stroke, which include arterial hypertension (AH), atrial fibrillation (AF), atherosclerosis, chronic heart failure (CHF), coronary heart disease (CHD), diabetes mellitus (DM) and smoking, Lately, sleep disorders have begun to be identified.

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At present, the relationship between the chronobiological characteristics of stroke patients before the development of acute cerebrovascular accident and the course of the disease, the relationship between the circadian characteristics of the development of cerebral infarction, and sleep quality characteristics with the prognosis of the disease, still remains unstudied. The role of sleep disorders in stroke outcome and recurrence has become a pressing question. Despite estimates of greater than 50% prevalence of sleep disorders after stroke, only about 6% of stroke survivors are offered formal sleep testing and an estimated 2% complete such testing in the 3-month post-stroke period [3]. The reasons for the low rate of screening are at least partly related to the lack of awareness regarding sleep disorders among stroke providers [4].

Sleep disorders, such as sleep-disordered breathing (SDB), insomnia or restless legs syndrome (RLS), are common in the general population and after stroke. In some cases, sleep disturbances are pre-existing, but can also appear de novo as a direct consequence of brain damage or due to stroke-related complications. Furthermore, some sleep conditions may act as a risk factor for stroke. This review explores the available evidence of the two-way relationship between sleep and stroke. Cardiovascular physiological changes during sleep are described, as well as the evidence on the relationship between stroke and sleep duration, SDB, RLS, insomnia, excessive daytime sleepiness (EDS), and circadian rhythm alterations. Potential changes in sleep architecture and the links that may exist between sleep and functional outcomes after stroke are also discussed. Importantly, sleep-related disturbances may be associated with worse stroke recovery outcomes and increased cerebrovascular morbidity. It is therefore relevant that the bidirectional association between stroke and sleep is taken into consideration by clinicians taking care of these patients. Future research may focus on this mutual relationship for a better understanding of the impact of stroke on sleep, the importance of sleep-in stroke incidence and recovery, and further evidence on treatment strategies that may improve functional outcomes after stroke [6].

In recent years, the view of stroke as a chronopathology has become stronger. Moreover, chronobiological features, associated mainly with lifestyle, influence both the formation of cardiovascular risk factors, clinical conditions associated with stroke, and directly the development of cerebral catastrophe [5]. It has been shown that disruption of circadian rhythm is the basis for the formation of circadian sleep disorders, which are often associated with cognitive dysfunction.

The relationship between sleep and stroke is complex and bidirectional [7].

Physiological changes of the cardiovascular system associated with sleep, and due to circadian variations, may play a key role in the aetiology and onset of stroke. Certain sleep disturbances may increase the risk for cerebrovascular events; obstructive sleep apnoea (OSA) and sleep duration have been considered potential, often modifiable, stroke risk factors. Conversely, many sleep disorders, such as restless legs syndrome (RLS), periodic limb movements during sleep (PLMS), insomnia, sleep-disordered breathing (SDB), excessive daytime sleepiness (EDS) and circadian anomalies, are frequently reported and diagnosed in patients having suffered a stroke. These sleep-related complaints, either as de novo presentation or because of their exacerbation after a stroke, may have an impact on the functional recovery of patients [8].

CHANGES IN THE CARDIOVASCULAR SYSTEM, SLEEP AND WAKEFULNESS AND STROKE

Initial studies reported that cerebral infarction occurred more often at night than during the daytime and that symptoms are usually noticed on waking [9].

However, nowadays, it is known that several types of cardiovascular events, including stroke, acute myocardial infarction, and sudden cardiac death, display significant circadian variation in symptom onset [10,11,12].

A meta-analysis identified a 49% excess risk for ischaemic stroke between 6 a.m. and noon compared with the number expected if there was no circadian predominance [13].

Furthermore, in studies that analysed specifically whether strokes occurred during sleep, they described a decrease in stroke frequency in the night sleep hours [14], therefore, proposing the idea of a protective effect associated with night sleep or an activator effect in awakening. Overall, about 25% of strokes occur during sleep [15,16], and a large proportion of them take place in the last part [17] when rapid-eye-movement (REM) sleep is more prominent. Changes during the REM sleep stage may trigger mechanisms for thrombotic events and may present clinically only after arousal [18].

Although the exact mechanisms are still not clear, there are several physiologic variations occurring during sleep and arousal that may predispose to a cerebrovascular event [19].

PHYSIOLOGY AND ANATOMY OF BREATHING DURING SLEEP

During sleep, ventilation is reduced compared to wake, in parallel with the restorative and toning-down changes that occur to heart rate, temperature and blood pressure [4].

Volitional or behavioural input on breathing is absent during sleep; only brainstem neurons, peripheral chemoreceptors and respiratory muscle afferents regulate breathing [20].

Groups of chemoreceptive neurons in the brainstem, including those of the dorsolateral pons, nucleus solitarius and ventral medullary respiratory column, respond to changes in the partial pressure of carbon dioxide and oxygen and thereby serve as a pacemaker regulating the breathing rhythm [21].

Along with effects on the breathing pattern, these brainstem neurons cause a reduction in the upper airway tone at sleep onset through reduced activity of airway dilator muscles, especially the genioglossus, which forms the bulk of the tongue [20].

The stability of sleep can be affected by brief (3 to 15 seconds) arousals that occur in response to changes in airflow. Respiratory events include 10 seconds or longer breathing cessation (apneas) and 30% or greater airflow reduction with associated oxygen desaturation or arousals (hypopneas). These are summed in a sleep study to generate an apnea-hypopnea index (AHI)—the number of respiratory events per hour of sleep. The AHI in OSA is typically classified as mild (5 to 14 per hour), moderate (15 to 29 per hour) or severe (≥ 30 per hour) [4]. Respiratory-provoked arousals during sleep, when breathing is otherwise reliant on respiratory and chemical control feedback mechanisms, serve to terminate the apnea or hypopnea by opening the airway in response to collapse and increasing the rate of ventilation in response to hypercapnia [20]. Although the arousals during sleep may play an important compensatory role in people with SDB, they may also have deleterious effects on sleep stability and other physiologic parameters before and after stroke [4].

CENTRAL AND OBSTRUCTIVE SLEEP APNEA

Two types of SDB, central sleep apnea (CSA) and obstructive sleep apnea (OSA), vary considerably in their aetiology, prevalence, relative improvement after stroke and effects on stroke outcome [4]. Central apneas occur most commonly in heart failure and opioid use but can also be observed after stroke due to distinct brain lesions involving autonomic and volitional respiratory centres [21,22,23].

Obstructive apneas or hypopneas occur despite the activity of the thoracic muscles (diaphragm and intercostal muscles) [21].

In a meta-analysis of SDB after ischemic or hemorrhagic stroke or transient ischemic attack (TIA), 72% of patients had an AHI of at least 5 per hour but only 7% of patients had primarily central apneas [24].

Further, CSA tends to improve after acute stroke [4]. In a study of 161 patients with first-ever stroke or TIA who underwent portable sleep studies within 48 to 72 hours from admission and again after 3 months, the rate of SDB decreased from 71% to 62% with a significant reduction in central apneas but not obstructive apneas [25].

The prevalence of OSA after stroke or TIA may be associated with several factors: brain damage per se can impair breathing control, similar risk factors are associated with stroke and SDB, there are stroke-related upper airway tone changes, and some of the patients with OSA detected after stroke may have had pre-existent undiagnosed OSA. To date, there is no conclusive link between the prevalence or severity of OSA and stroke subtype, topography, size or severity [26].

THE INFLUENCE OF OBSTRUCTIVE SLEEP APNEA ON THE OCCURRENCE, RECURRENCE AND RECOVERY OF STROKE

Population-based epidemiologic studies show that OSA independently predisposes to stroke [4]. In a prospective analysis of 1,189 healthy participants in the Wisconsin Sleep Cohort study, an AHI ≥ 20 per hour was associated with an increased risk of stroke over the next 4 years (unadjusted OR 4.31; 95% CI 1.31–14.15; p=0.02), though this relationship was not significant after adjustment for potential confounders [26]. In the community-based Sleep Heart Health Study, 5,422 healthy participants without stroke were evaluated with polysomnography and followed for a median of 8.7 years [27]. An AHI > 15 per hour was 30% more common among participants who had an ischemic stroke compared to those who remained stroke-free. Men with moderate or severe OSA had an almost threefold increased risk of ischemic stroke compared to those without OSA, with an estimated 6% increased risk of stroke per unit increase in the AHI from 5 to 25 per hour, whereas women had an increased risk of stroke only with an AHI > 25 per hour. In an observational cohort study of 1,022 clinic patients referred for sleep evaluation, patients with OSA had a nearly two-fold increase in stroke

or death from any cause after a median of 3.4 years independent of known vascular risk factors and with increased risk associated with OSA severity [29].

CSA AND STROKE

CSA episodes may be associated with an increased risk of adverse cardiovascular outcomes since evidence from animal models has shown an increment of adrenergic hyperexcitation independent of arousals [30], as well as desaturation and hypoxia [6]. Furthermore, a relationship between CSA and decreased ventricular function has been described in various studies [31], and it may serve as a marker of underlying cardiac vulnerability, predisposing to increased cardiac arrhythmias [32]. CSA may also emerge as a consequence of stroke, and it has been observed in patients with vascular injury to the respiratory centres in the medulla, infratentorial lesions, and bilateral hemispheric lesions [33].

CSA tends to improve after acute stroke [24]. In a study of 161 patients with first-ever stroke or TIA who underwent sleep studies in the acute phase and again after 3 months, the rate of SDB decreased with a significant reduction in central but not obstructive apnoeas, suggesting that CSA following acute stroke is often self-limited [25].

Taken together, all these changes with a predominance of circadian rhythms occurring during sleep or wakefulness may be involved in the development of cerebrovascular disorders.

CONCLUSION

Thus, after reviewing the literature, we can say that sleep disorders, including sleep-disordered breathing, parasomnias, sleep-related movement disorders, insomnia and hypersomnia, and restless legs syndrome, are closely associated with concomitant cardiovascular diseases and play an important role in the quality of life of patients. However, it should be noted that some points have not yet been studied, such as the relationship of chronobiological characteristics in stroke patients before the development of acute cerebrovascular accident with the course of the disease, the relationship of circadian characteristics of the development of cerebral infarction, characteristics of sleep quality with the prognosis of the disease.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open the publication.

Availability of data and material – Available.

Competing interests – No.

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UYQU VA INSULT. YUZAGA KELISHI VA KECHISHI XUSUSIYATLARI.

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Toshkent tibbiyot akademiyasi

ABSTRAKT

Ishemik va gemorragik insult kabi serebrovaskulyar kasalliklar butun dunyo bo'ylab nogironlik va o'limning asosiy sabablaridan biridir. Dalillar shuni ko'rsatadiki, uyqu buzilishini aniqlash va davolash birlamchi va ikkilamchi insultning oldini olishga kiritilishi kerak. Uyqu va insult ko'pincha bir-biri bilan chambarchas bog'liq, chunki uyqu buzilishi, shu jumladan uyqu bilan nafas olishning buzilishi, parasomnialar, uyqu bilan bog'liq harakatlarning buzilishi, uyqusizlik va gipersomniya yurak-qon tomir kasalliklari bilan chambarchas bog'liq va insult xavfini oshiradi. Qon tomiridan keyin uyqu buzilishi ham insult reabilitatsiyasiga va hayot sifatiga ta'sir qilishi mumkin va agar davolanmasa, takroriy insultga olib kelishi mumkin.

Kalit so'zlar: insult, uyqu, uyqu buzilishi, uyquda nafas olishning buzilishi, markaziy uyqu apneasi, obstruktiv uyqu apneasi.

СОН И ИНСУЛЬТ. ОСОБЕННОСТИ ВОЗНИКНОВЕНИЯ И ТЕЧЕНИЯ.

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АБСТРАКТ

Цереброваскулярные заболевания, такие как ишемические и геморрагические инсульты, являются одними из ведущих причин инвалидности и смертности во всем мире. Данные показывают нам, что выявление и лечение нарушений сна должно производиться как в первичной, так и во вторичной профилактике инсульта. Сон и инсульт часто переплетаются, поскольку нарушения сна, включая нарушения дыхания во сне, парасомнии, двигательные расстройства, связанные со сном, бессонницу и гиперсомнию, тесно связаны с сопутствующими сердечно-сосудистыми заболеваниями и повышают риск инсульта. Нарушения сна после инсульта также могут повлиять на реабилитацию после инсульта и качество жизни, а при отсутствии лечения могут привести к повторным инсультам.

Ключевые слова: инсульт, сон, нарушения сна, нарушение дыхания во сне, центральное апноэ во сне, обструктивное апноэ во сне.