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THE CONDITION OF THE BRAIN'S VENOUS CIRCULATION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME

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Abstract: Cerebrovascular disorders are a widespread issue worldwide. The study of the cerebral arteries receives the majority of research attention. However, little is known about the venous component of the cerebral circulation, which is crucial to the development of the disease. The suction force of the chest during inspiration is one of the elements of the mechanism of venous outflow from the head. As a result, respiratory problems might make brain venous congestion worse. The data from a study on sleep-related breathing problems in people with chronic cerebral ischemia are presented in the paper. It was found that there was a correlation between the severity of OSAS and cognitive impairment.

Keywords: obstructive sleep apnea syndrome, polysomnography, chronic cerebral ischemia, apnea-hypopnea index, memory disorders

Numerous studies completed worldwide over the past two to three decades indicate that 20 to 26 percent of adults snore when they sleep on a regular basis [6]. One of the primary signs of obstructive sleep apnea is frequently snoring.

Due to increased airway resistance to airflow, obstructive sleep apnea (OSA) is characterized by periods of breathing pauses during sleep. The primary pathogenetic processes for the onset of the disease are caused by such recurrent episodes of apnea that last throughout the course of the night and result in a decrease in blood oxygen saturation.

According to gender, age, and the presence of co-occurring disorders, the prevalence of OSA varies by gender and is 3-9% in women and 10-17% in males [1,5].

Obesity, arterial hypertension, metabolic syndrome, chronic obstructive pulmonary disease, pulmonary hypertension, diabetes mellitus and other endocrine disorders, ENT pathology, characteristics of the face skeleton, etc. are known to be the main causes of OSA. These disorders cause respiratory tract obstruction, which makes it harder for air to pass through during sleep, resulting in tissue hypoxia.

Desaturation brought on by repeated apnea-hypopnea episodes during sleep causes endothelial damage, sympathetic nervous system activation, and hormonal system imbalance.

OSA increases the chance of vascular accidents, such as myocardial infarction and stroke, and it also makes chronic cerebrovascular diseases that already present worse. The majority of studies investigating obstructive sleep breathing disorders place a high priority on examining the arterial connection, specifically changes in the arteries of the extra- and intracranial portions of the brain's vascular system. The study of the brain's venous bed and the changes that can occur in this system, which can lead to some disease symptoms and also act as a key pathogenetic mechanism for the development of pathological changes in the brain, is a subject that is still understudied.

It should be mentioned that the poorly developed valve apparatus of the venous system in the area of the head and the upper half of the human body as a whole increases the risk of cranial venous stasis.

In addition to gravitational forces, the right half of the heart's active pumping function and the chest's suction force during inspiration are also linked to the principal mechanisms

British Medical Journal Volume-3, No 1

of venous outflow from the cranial cavity.

According to the findings of various research, OSA patients with negative intrapulmonary pressure have increased systemic venous return, which includes the brain's venous system [1,4]. As a result of the increased pressure in the cerebral venous system, the intracranial pressure also rises, which ultimately worsens cerebral perfusion. The venous system of the brain and extracranial veins were thus shown to have undergone modifications, as per the findings of investigations by Hsin Yi Chi et al. (2015).

When comparing patients with OSAS to the control group, they discovered a substantial reduction in blood flow in the internal jugular vein and a reduction in blood flow in the transverse sinus on both sides (p 0.05) [4].

All of these shows that patients with OSA have disrupted venous outflow pathways. Nevertheless, despite the evidence, not enough research has been done on this problem. There are particularly limited studies on how chronic cerebral ischemia (CCI) and venous congestion in OSA patients develop and progress. The decline of memory, attention, and other cognitive skills in OSA and their aggravation in this condition have been the subject of a few studies [3, 5].

The following techniques are employed for OSA screening, diagnosis, and monitoring of the pathology's dynamics:

1.Interrogation. There are numerous different scales and questionnaires that can be used to determine whether patients are suffering from sleep disturbances. The sleep apnea screening questionnaire, the STOP-BANG questionnaire, the Berlin sleep apnea questionnaire, the Epworth daytime sleepiness scale, the Lausanne scale, etc. are the most used questionnaires due to convenience, simplicity, and informativeness.

2.Monitoring computer pulse oximetry (MCP) is a research technique that involves recording a patient's saturation over an extended period of time as they sleep. With this technique, you may track the occurrence and length of desaturations (a drop in saturation of more than 3% from baseline saturation) while you're sleeping.

3.A diagnostic technique called cardiorespiratory monitoring (CRM) is used to find sleep apnea in patients who have a high pre-test likelihood of having the condition. The inclusion of a channel for recording respiratory flow and snoring in addition to a pulse oximetry sensor distinguishes this method from monitoring pulse oximetry.

4.The "gold standard" for diagnosing sleep disturbances is polysomnography (PSG). The research method entails continuous monitoring of a number of physiological parameters throughout the course of the night, including electroencephalography, electrooculography, electromyography from the muscles of the chin and lower extremities, electrocardiography, registration of respiratory flow, snoring, respiratory efforts of the chest and abdominal walls, oxygen saturation, and body position.

The apnea-hypopnea index (AHI), which measures the frequency of respiratory episodes (apnea and hypopnea) during a single hour of sleep, is used to diagnose sleep apnea syndrome based on PSG and CRM and to establish its type and severity. According to the generally accepted classification (AASM), there are three different degrees of OSA, depending on the apnea-hypopnea index (AHI): mild, with an AHI of 5-15 per hour; average, with an AHI of 15-30 per hour; and severe, with an AHI of more than 30 per hour [2].

Research objectives.

The goal of our study was to examine the characteristics of changes in the veins of the extracranial brachiocephalic trunk (in particular, the internal jugular vein) and correlate them with clinical data and the course of CCI with concurrent OSA in patients due to the lack of knowledge regarding the role of the venous system of the brain as one of the factors that can influence the course of CCI as a comorbid or background disease

British Medical Journal Volume-3, No 1

in OSA.

Materials and methods

We studied 146 patients with OSA and CCI to investigate the characteristics of cerebral venous discirculation.

The primary group included 106 patients with a diagnosis of OSA based on clinical, individual, and polysomnography (PSG) results. The patients ranged in age from 50 to 80. The main group was split into two smaller subgroups: subgroup 1 included 63 patients with mild OSA, while subgroup 2 included 43 patients with severe OSA. 40 patients without OSA who were of a similar age to the main group made up the control group.

All patients were questioned on standard scales for the detection of sleep apnea syndrome - a sleep apnea screening questionnaire, determination of the level of daytime sleepiness on the Epworth scale, Lausanne scale, as well as monitoring computer pulse oximetry during night sleep. 57 patients had cardiorespiratory sleep monitoring, and 34 patients underwent a polysomnographic investigation. Additionally, the severity of cognitive impairment in CCI was assessed for all patients using the MMSE and MiniCog, two internationally recognized assessments. Duplex scanning of extracranial and intracranial arteries and veins was performed on all patients as part of an ultrasound examination of the vessels of the brachiocephalic trunk to look for signs of impaired venous outflow from the cranium, including the presence and severity of changes in arteries and veins (change in velocity parameters along the internal jugular vein).

Using the Mini Mental State Examination scale, cognitive function was evaluated in all patients. The sum of the points on this scale was used to determine the degree of cognitive impairment, and the results were interpreted as follows: no cognitive impairment was defined as a score of 28 to 30, pre-dementia cognitive impairment was defined as a score of 24-27, mild dementia was defined as a score of 20 to 23, moderate dementia was defined as a score of 11 to 19, and severe dementia was defined as a score of 0-10.

Results and conclusions

The average MMSE score in the main group was 20 points in the subgroup of patients with mild OSA, and 12 points in the subgroup of patients with severe OSA. This indicator was 24 points in the CCI-negative, OSA-free control group of patients.

In 50 patients (79%) in the first subgroup of the main group, the analysis of data from brachiocephalic arteries' duplex scanning showed a substantial shift in velocity parameters along the internal jugular vein (patients with CCI and moderate OSA). 38 patients (88%) of the second subgroup of the main group had the same alterations (patients with CCI and severe OSA). At the same time, 52 patients (or 55%) in the control group had symptoms of venous congestion. According to the collected data, patients who have OSA and CCI are significantly more likely to have cognitive impairment, and the degree of the latter corresponds with the severity of OSA (p0.05).

The data collected show a direct correlation and reliance between the degree of cognitive impairment and the degree of obstructive sleep apnea syndrome, which is the root cause of reduced cerebral venous outflow in the patients with chronic cerebral ischemia under study.

Thus, the condition known as chronic cerebral ischemia depends on both venous outflow and atherosclerotic lesions of the arteries, with decreased venous outflow having the greatest impact. In light of this, it is crucial to carefully assess individuals with chronic cerebral ischemia who run the risk of experiencing breathing problems while they sleep, paying special attention to any issues with the brain's venous circulation. It is vital to modify the therapeutic strategies in the care of patients who fit this profile as the symptoms of venous congestion of the brain become more apparent. Patients with

British Medical Journal Volume-3, No 1

CCI with OSA will benefit from the prevention or slowing of the advancement of cognitive impairment thanks to early detection and treatment of sleep breathing difficulties and their effects on the course of CCI. This will ultimately improve the patients' quality of life.

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