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THE MAIN CAUSES OF COGNITIVE IMPAIRMENT

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Cognitive impairment is a common condition among elderly patients in medical practice. Currently, there is a tendency in the world to increasing of rate cognitive impairment of various etiologies, which allows us to regard this pathology as an urgent social and medical problem. Different types of cognitive impairment in the elderly are associated with poor quality of life, increased morbidity and early mortality. This article presents an overview based on publication data of the etiology and risk factors of cognitive impairments.

Keywords: cognitive impairment; dementia; hypertension; thyroid disease; diabetes; metabolic syndrome; depression; anemia.

ОСНОВНЫЕ ПРИЧИНЫ РАЗВИТИЯ КОГНИТИВНЫХ НАРУШЕНИЙ

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Когнитивные нарушения — распространенное состояние среди пациентов пожилого возраста во врачебной практике. В настоящее время в мире прослеживается тенденция к повышению частоты встречаемости когнитивных нарушений различной этиологии, что позволяет расценивать данную патологию как актуальную социально-медицинскую проблему. Различные виды когнитивных нарушений у пожилых людей связаны с низким качеством жизни, повышенной заболеваемостью и ранней смертностью. В статье на основе литературных данных проанализированы причины и факторы риска развития данной патологии.

Ключевые слова: когнитивные нарушения; деменция; артериальная гипертензия; заболевания щитовидной железы; сахарный диабет; метаболический синдром; депрессия; анемия.

Background

Mental health in the elderly, due to several factors, is a problem not only for the individual but also for society as a whole. First, most modern people spend nearly 50% of their lives in old age. Changes in the age composition of the population in most countries appear to not only increase the average life expectancy, but also increase the population of elderly people in society. In Russia, people aged >60 years make up 25% of the population. Second, elderly and senium patients can be classified as a group at high risk for mental disorders. This is due to both the physiological processes occurring in the body from a certain age along with psychological and social factors.

Arterial hypertension

The vascular system and the substance of the brain, along with the heart, kidneys, and retina, are target organs of arterial hypertension (AH). During the development and progression of hypertension, a complex set of functional and structural changes is formed in the vascular system of the brain. Pathology of the central nervous system is a serious consequence of hypertension and is one of the main causes of cognitive disorders of the elderly. A common outcome of hypertension is hypertonic encephalopathy, which leads to the development of vascular dementia. The development of cognitive disorders is influenced not only by blood pressure (BP) level but also by other factors such as the BP variability, which has been shown in several prospective studies [2–4]. In the study conducted by N. Cho et al. [2], the results of a survey of 232 outpatients with an average age of 77.7 ± 8.3 years were analyzed. All patients underwent outpatient BP monitoring and were tested using the Japanese version of the Montreal cognitive scale (MoCA-J). According to the results, patients with an average daily variability of BP of ≥19.6 mm Hg had the lowest mean grade on total MoCA-J ($p = 0.001$), and on controlled

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tests that evaluate attention ($p = 0.001$) and operation functions ($p = 0.012$).

Queen et al. [3] concluded that a higher variation in systolic BP was associated with a faster decline in cognitive function from visit to visit, and a higher variation in diastolic BP was associated with a faster decline in cognitive function, independent of an average diastolic BP, among adults aged from 55 to 64 years, but not among patients aged ≥65 years.

McDonald et al. [4] demonstrated that increased daily variability in systolic and diastolic BP is associated with impaired cognitive function in older adults and may represent a new modifiable risk factor for cognitive decline.

In addition to BP variability, the duration of AH plays a special role in the development of cognitive disorders [5, 6].

According to the results of a study by E.V. Osipov et al. [5] in 2015, the severity of cognitive dysfunction significantly depends on the duration and stage of AH.

The English Maastricht Aging Study (MAAS) [6], a prospective cohort study, evaluated the effect of existing and newly emerging hypertension on the cognitive functions of patients. At the base visit in 1993–1995, the study included 1,805 people aged 24–81 years, 35.3% of whom were diagnosed with AH. After 6 and 12 years of monitoring, patients diagnosed with AH at the initial visit showed a faster decline in memory, deterioration of control functions, and speed of information processing compared to patients without AH. After adjustment for age, it was found that for patients with AH at the age of <65 years, indicators in all three cognitive domains decreased rapidly, while for patients aged ≥65 years, the presence of AH at the initial visit was a predictor of a faster decrease only in information processing speed. At the same time, for patients who were diagnosed with AH during the monitoring (at the time of the initial visit, their BP figures were within normal values), cognitive impairment at the end of the monitoring period was expressed to a statistically significantly lower degree compared to patients who had hypertension at the initial visit. This indicates that the duration of the disease affects the state of cognitive functions.

Disbalance of a carbohydrate metabolism

Diabetes mellitus is a complex metabolic disease that leads to disorders of many systems in the body. The most common complications of this disease are blindness, gangrene of the lower extremities, nephropathy, and various diseases of the cardiovascular system. A less common complication is diabetic encephalopathy, which causes the development of cognitive disorders.

The development of diabetic encephalopathy is a multi-factorial process similar to that of diabetic polyneuropathy. Vascular dysfunction develops and causes a decrease in blood supply to the nerves and brain tissue, trophic disorders, and the direct toxic effect of hyperglycemia on the nerves. The effect of diabetes on the brain is more pronounced for older adults and leads to accelerated cognitive decline due to aging and the development of encephalopathy.

In a healthy brain, insulin contributes to the potentiation of the hippocampus, which is associated with learning and memory. Insulin also regulates the concentration of neurotransmitters (acetylcholine, norepinephrine, and epinephrine) that play an important role in memory formation. In contrast to the beneficial effect of insulin on a healthy brain, increasing the level of insulin through infusion does not improve the central nervous system. Rather, it leads to disorganization of many systems, which in turn leads to a decrease in cognitive abilities.

According to the Rotterdam study, which lasted 2.1 years, type 2 diabetes mellitus was associated with double the risk of Alzheimer's disease and dementia. Patients who received insulin had the highest risk of developing dementia. A higher risk of developing cognitive impairment was also observed for patients with obesity, which is usually associated with type 2 diabetes mellitus; thus, the risk of developing dementia is much higher with type 2 diabetes mellitus.

It is believed that insulin also regulates the function of neurons in the central nervous system and affects the metabolism of β-amyloid. It is interesting that in the development of type 2 diabetes mellitus, there is a loss of beta cells of the pancreas, in which amyloid is later deposited in their place. This process is very similar to the loss of neurons and deposition of β-amyloid of the Alzheimer's disease. The structure of insular and neural β-amyloids is similar; both are toxic to islets and neurons, accordingly [7-9]. Based on histological studies of agents of the brain and pancreas, it was noted that insular amyloid was more common for patients with Alzheimer's disease than for patients without it.

Thus, in type 2 diabetes mellitus, there is a slight or moderate deficit in cognitive function, which may be more pronounced with a concomitant metabolic syndrome. The pathophysiology of diabetic cognitive disorders is complex, but may include impaired insulin signaling, increased inflammation, and oxidative stress. Identification of key pathophysiological components is important for the development of new approaches to therapy.

Disbalance of the functions of the thyroid gland

The relationship between endocrine pathology and cognitive disorders has been actively studied. The role of the thyroid gland in the development of dementia is the least studied. The first attempt to prove the link between the increased prevalence of senium dementia and thyroid pathology was made in 1989 [12]. Thyroid hormones are crucial in developing the brain and maintaining its normal functioning. For people with thyroid diseases, changes in mental function and mood are observed. For example, impaired memory, anxiety, depression, and reduced hippocampus size have been reported in adults with hypothyroidism. However, these changes are reversible, which highlights the important role of thyroid hormones in maintaining normal adult brain function. Detailed studies of age-related changes in the hypothalamus and the pituitary–thyroid system, and their role in cognitive impairment, are important, since such changes in the thyroid are often found in older adults and may be an early risk factor for dementia in the old age [10]. A higher overall triiodothyronine (T_3) level was found in cerebrospinal fluid in a group of participants with hippocampal sclerosis at autopsy, while a reduced concentration of T_3 was registered in the prefrontal cortex of postmortem brain tissue of patients with Alzheimer's disease using radioimmune analysis [11]. According to current literature, there is a proven link between Alzheimer's disease and Hashimoto's thyroiditis. It was found that the phagocytic activity of microglia in the central nervous system is controlled by thyroid hormones. In hypothyroidism, insufficiently active phagocytosis and accumulation of Alzheimer's plaque material are observed, and in hyperthyroidism, excessive glial cell activity is observed, which contributes to autophagocytosis and autoimmune inflammation. Later, it was proved that T_3 binds to microglia receptors and activates its phagocytic activity [12].

Despite the well-known pathogenesis of cognitive disorders in thyroid gland dysfunction, there are conflicting data in the literature. For example, in a meta-analysis of 11 prospective cohort studies (16,805 participants observed for 44.4 months), of which five were devoted to subclinical hyperthyroidism and six to subclinical hypothyroidism, it was found that subclinical hyperthyroidism is associated with an increased risk of developing dementia in contrast to subclinical hypothyroidism, in which an increased risk was not detected. There was also no rapid decrease in indicators according to the English Mini Mental State Examination scale over time in either disease, compared to individuals with euthyroidism [13].

Bykova et al. reported that for patients who had an ischemic stroke and suffered from hypothyroidism, the cognitive deficit was more pronounced than in the absence of an endocrine pathology. At the same time, all patients had a disbalance of several cortical functions. The obtained data confirm the pathogenetic role of thyroid hormones in the development of cognitive disorders [14].

In a prospective randomized study of 90 patients with nodular toxic goiter and diffuse toxic goiter confirmed by thyrotoxicosis, it was found that patients who underwent a surgical procedure for the thyroid gland, in the early postoperative period experienced changes in mental functions, including impaired concentration, short-term and visual memory [15].

It is necessary to continue research to confirm the causal relationship and to timely identify elderly adults with subclinical thyroid gland disorders who are at risk.

Anemia

One of the most common etiological factors of cognitive disorders is anemia. According to data from 161 countries, approximately 1.9 billion people, or 27% of the world's population, suffered from anemia in 2013 [16]. Epidemiological data indicate that the prevalence of anemia increases with age, ranging from 4.3% to 26% in different age and gender groups [17]. A comparative analysis of research data (11 years of monitoring) of 445 participants showed that the subjects who suffered from anemia at the

initial stage of the project developed dementia 41% more often than their peers without anemia [18]. The mechanisms underlying this link are not well understood, but it is believed that a decrease in the level of oxygen-containing capacity of the blood can lead to hypoperfusion of the brain, further oxidative stress, and inflammatory cellular reactions that cause neurodegenerative processes [19]. In addition, anemia can be an indicator of such a pathological process as kidney dysfunction and can lead to a decrease in the level of erythropoietin, as well as an increased risk of neural degeneration, since erythropoietin has a neuroprotective effect [20].

The study, conducted in Italy, included 3,099 patients. A significantly higher risk of cognitive impairment was observed for individuals with lower concentrations of hemoglobin. However, participants with lower hemoglobin levels were less exposed to several potential risk factors for cognitive impairment, such as smoking, alcohol, hypertension, diabetes, and cardiovascular disease; still, the incidence of cognitive impairment during the monitoring in this group was higher [21]. It was also found that patients with cognitive impairment are less committed to therapy [22].

The degree of anemia at folic acid deficiency is poorly correlated with the presence of disorders of the nervous system. One of the nonspecific clinical appearances of a folic acid deficiency is cerebral atrophy, assessed *in vivo* using neuroimaging methods. However, a deficiency of vitamin B_{12} and folic acid, as well as an increase in homocysteine levels, can lead to the development of cognitive disorders. Timely diagnosis and adequate therapy, initiated before the development of severe cognitive disorders, can prevent the development of dementia [23].

Patients with a low concentration of vitamin B_{12} in the blood showed significantly lower memory indicators, in particular the ability to learn and the ability to recognize, and there was a tendency of decreased long-term memory indicators compared to patients with a high concentration of vitamin B_{12} [24].

According to some data, low levels of vitamins B_9 and B_{12} were associated with a higher risk of death from Alzheimer's disease with a decrease in hemoglobin levels. Thus, adequate folate and vitamin B_{12} intake is a preventive strategy for reducing Alzheimer's disease mortality, especially for individuals at high risk of anemia [25].

Depression

Depression is a mental illness characterized by the Kraepelin triad: temper decline, and motor and ideational inhibition [26]. Three types of cognitive functions are impaired in depression: concentration, memory, and executive functions [27].

In multiple studies, it has been found that individuals with depression have deeper cognitive impairments than individuals with only cognitive dysfunction. In addition, they are characterized by a higher degree of progress from mild forms of cognitive impairment to dementia [28].

R. McIntyre et al. identified the following main factors that can affect the speed and depth of development of cognitive disorders:

– age at the time of examination;
– age of the beginning of the disease;
– severity of the current depressive episode;
– level of education;
– number of depressive episodes per year; and
– use of psychoactive substances, etc.

Cognitive disorders in depression are preceded by the following psychological and biological prerequisites [26, 29]:

– negative influence of emotional state on the ability to correctly distribute attention;
– reduced motivation (pessimism, a lack of confidence in their abilities lead to the cessation of all intellectual activity, and this exacerbates cognitive disorders);
– decrease in the synthesis and activity of cerebral neurotransmitters (a key neurochemical mechanism for the formation of emotional disorders);
– decrease in the synthesis and activity of serotonin, norepinephrine and dopamine in the brain (which serves as a neurochemical substrate for the formation of a cognitive syndrome);
– activation of the hypothalamic–pituitary–adrenal system (which leads to an increased production of steroid hormones, and this in its turn leads to the activation of cerebral atrophic changes); and
– sleep disorders (during sleep, the process of processing and consolidating information ends; a lack of sleep leads to a decrease in the activation of the cerebral cortex by the stem subcortical structures, which is clinically appeared by a deterioration in concentration, activity and the speed of a cognitive activity).

Thus, disorders of cognitive functions are important components of the clinical picture of a depression; they negatively affect the quality of life of patients and their daily activities, including work.

Conclusion

Older adults suffer from a variety of diseases. It is not always possible to accurately determine the cause of cognitive disorders, so it is especially important to correct the somatic status timely and carry out measures to prevent the development and aggravation of cognitive deficits. It is proposed to use a comprehensive approach to prevent the development of cognitive disorders that includes effects that cover numerous areas: physical activity, cognitive exercise, rational nutrition, social activity, and control of vascular and metabolic risk factors.

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