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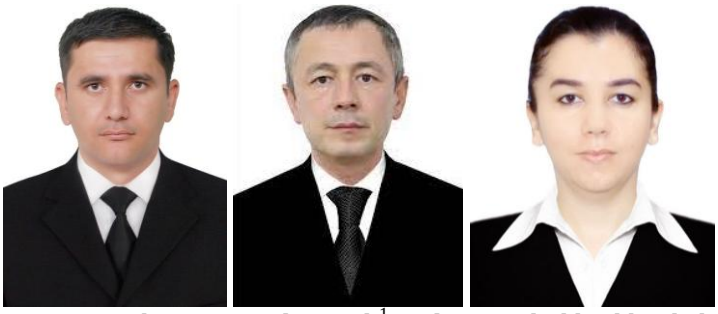
FEATURES OF THE MANIFESTATION OF DEPRESSIVE DISORDER IN PATIENTS WITH DEMENTIA



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ДЕМЕНЦИЯ БИЛАН КАСАЛЛАНГАН БЕМОРЛАРДА ДЕПРЕССИВ БУЗИЛИШНИНГ НАМОЁН БЎЛИШ ХУСУСИЯТЛАРИ

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ОСОБЕННОСТИ ПРОЯВЛЕНИЯ ДЕПРЕССИВНОГО РАССТРОЙСТВА У БОЛЬНЫХ ДЕМЕНЦИЕЙ

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Резюме. *Деменция ва депрессия кекса ёшдаги кишилар ўртасида энг кенг тарқалган психопатологик синдромлардан бири бўлиб, 60 ёшдан ошганларнинг 5-10 фоизида деменция, 15-25 фоизида когнитив бузилишлар, 20-30 фоизида депрессив бузилишлар кузатилади. Депрессия ва деменция ўртасидаги муносабат мураккаб бўлиб, сўнги вақтлардаги илмий мақолаларда ушбу патологиялар ўртасидаги муносабат ва уларнинг намоён бўлиш хусусиятлари тўлиқ ёритиб берилмаган. Ушбу мақолада деманция билан касалланган беморларда депрессив бузилишнинг намоён бўлиш хусусиятлари тўлиқ тушунтирилган.*

Калит сўзлар: *деманция, депрессия, когнитив бузилиш, Альцгеймер касаллиги.*

Abstract. *Dementia and depression are among the most common psychopathological syndromes among the elderly: dementia is observed in 5-10% of people over 60 years old, cognitive impairment in 15-25%, and depressive disorders in 20-30%. The relationship between depression and dementia is complex, and recently scientific articles have not fully covered the relationship of these pathologies and the features of their manifestation. This article fully explains the features of the manifestation of depressive disorder in patients with dementia.*

Key words: *dementia, depression, cognitive impairment, Alzheimer's disease.*

Depressive disorder can cause cognitive impairment. This opinion is based on a number of studies showing that patients with depression perform worse on neuropsychological tests for attention, memory, psychomotor and visual-spatial functions, speech functioning, etc., than those who do not suffer from depression [1]. In addition, impaired concentration and mental activity is one of the main criteria for diagnosing depression. However, these concepts are not entirely correct and do not take into account the heterogeneity of patients with depression. With an adequate objective comprehensive assessment of cognitive functions in a significant proportion of patients with depression, significant deviations from the age norm cannot be detected, despite frequent complaints of distractibility or forgetfulness [3].

On the other hand, some patients with depression fail tests that require energy and motivation [4]. It is easier for them to answer "I don't know" or "I don't remember" than to concentrate when trying to complete a task, sometimes very simple, but requiring mechanical force, for example, repeating a number string they heard. At the same time, if they seem more interesting and do not require effort, they can

easily perform more complex memory or thinking tasks. If the tests are repeated, their results may unexpectedly improve or worsen. In addition, if the patient did not pass the previous test for certain reasons, he may categorically refuse further testing. Thus, a very mosaic picture is formed, which does not allow identifying certain affected cognitive functions, but rather reflects an emotional-volitional deficit [2].

However, in some patients with depression, cognitive impairments are objectively detected, which are not explained only by insufficient efforts during tests. As a rule, the deficiency affects neurodynamic and regulatory cognitive functions and manifests itself in tests for reaction speed, stability of concentration and attention, as well as in planning and performing competitive or sequential actions. There may be a dysfunction of the frontal cortex associated with the functional separation of subcortical and frontal structures [5]. In these cases, the profile of memory impairment is particularly indicative: slow-motion reproduction decreases, while recognition and indirect reproduction persist, which is explained only by a violation of the regulation (frontal) of cognitive functions [7]. In addition, a moderate decrease in phonetic speech activity (only words starting with a certain letter are called per minute) is associated with the relative safety of semantic speech activity (the number of words belonging to a certain category, for example, the so-called plants per minute) [6]. At the same time, the general level of intelligence, praxis, gnosis, orientation in time and space do not significantly affect. As a rule, there are no paraphyletic errors and impulsive incorrect answers. Patients usually do not forget the task conditions typical for patients with dementia. Cognitive functions can be completely normalized with the use of antidepressants, which can prove the connection of cognitive deficits with depression, but the relationship between the severity of cognitive and affective disorders is usually not found [10].

The absence of a direct effect of depression on cognitive functions is also confirmed by data from surveys of stroke patients. On the one hand, patients with post-stroke depression have more cognitive impairments than patients without depression. However, R. As Robinson showed [9], this pattern is observed only in patients with left-sided (but not right-sided) cerebral infarction, and this relationship was noted only in the first months after stroke. Thus, it would be more correct to consider depression and cognitive impairment as two relatively independent manifestations, and more serious cognitive impairment in patients with depression is explained not by the consequences of affective disorders, but by a certain localization or breadth of brain damage. In left-sided heart attacks, the function of the frontal lobes may be more damaged, which explains the development of cognitive impairment (depression, according to the author, is mainly associated with dysfunction of the temporal structures) [8].

Cognitive deficits are especially often diagnosed in elderly patients with depression if it first appeared later in life. In at least half of these cases, cognitive impairment persists despite normalization of the affective state associated with the use of antidepressants or the spontaneous cessation of a depressive episode [6].

At least in a significant proportion of patients with depression, the neuropsychological defect does not reflect the effect of depression on the cognitive sphere, but a parallel developing cognitive disorder that may be associated with the same organic brain damage as depression.

This condition can be described as "depression plus". One of the most common causes of "depression" is vascular damage to the white or subcortical gray matter of the cerebral hemispheres (the so-called "vascular depression") [7]. In some studies, more than 90% of patients with the onset of depression in old age had vascular changes in neuroimaging.

In the late onset of depression, especially if depression is accompanied by cognitive impairment, the risk of developing dementia increases by 1,5-3 times [8]. In these cases, it can be assumed that depression develops at an earlier stage of organic damage, and dementia at a later stage. At least, similar events are shown in the model of cerebrovascular disease [2]. In this context, depression can be considered as a Prodrome of dementia. It has been shown that symptoms of depression can precede symptoms of dementia by several years as part of a steadily developing disease [4].

There is more conflicting evidence about the likelihood of developing dementia in patients with onset of depression at a young or middle age. According to a number of studies, an increased risk of developing dementia is observed later in life, and high-frequency dementia develops after one or several decades [6]. In addition, the risk of developing dementia may increase with repeated episodes of depression. Thus, the risk of dementia roughly doubled after four episodes of depression and tripled after five or more episodes [8]. Other researchers contributed to this, in particular Heun R. and the data of others are contradictory. (2002) showed that the risk of developing dementia does not increase if depression is not accompanied by cognitive impairment [1].

The increased risk of dementia in patients with depression can be explained by a decrease in the volume of the hippocampus, which, in turn, may be associated with constant excessive cortisol production. Evidence of a link between depression and high cortisol levels in a patient with cognitive impairment has been confirmed. However, Gerlings M. I. et al., an increased risk of dementia in early onset depression is not associated with hippocampal atrophy [10]. Ap-

parently, it cannot be said that depression in such cases "stimulates" the development of the main pathological process leading to dementia, but it accelerates the onset of dementia, "undermines" the cognitive reserve, allowing you to compensate for the growing cognitive defect for a certain time. At the same time, the presence of depression reflects the significant prevalence and activity of the pathological process, which, along with the hippocampus, includes, for example, frontal-limbic structures.

From a similar perspective, it is also possible to evaluate research data showing that in the presence of depression, the transition from moderate cognitive impairment (mild cognitive impairment) to dementia occurs faster and more often than in its absence. It has been shown that symptoms of depression are detected in about a quarter of patients with moderate cognitive impairment. Alheimer's type dementia developed in 85% of patients with moderate cognitive impairment of the amnesic type with symptoms of depression and in 32% of patients without depression during the last follow-up period [4]. At the same time, Wilson R. and according to others. (2008) found that in cases of moderate cognitive impairment or progression of Alzheimer's disease in patients with depression, there is no parallel increase in depressive symptoms, indicating relative independence in the development of these disorders [9].

In recent years, it has been shown that depression can contribute to the development of the pathological process leading to dementia by activating the neuroinflammatory process or blocking the production of neurotrophic factors in the brain.

On average, symptoms of depression are more common in one third of patients with dementia than in people of the same age who do not suffer from dementia [4]. However, this fact should be considered as a reflection of the comorbidity of depression and dementia in old age. It is important to note that the level of depression in patients with dementia is very high. This partly depends on the nosological form and severity of dementia, diagnostic criteria for depression, and consideration of all forms of depression (major, minor, subsyndromic, dysthymia, etc.). In Alzheimer's disease, depression is somewhat less common (about 20-30%), in dementia associated with the involvement of subcortical (subcortical-frontal) structures (dementia with Levi's corpuscles, Parkinson's disease with dementia, vascular dementia) – somewhat more often (30-60%), but clinical manifestations of depression. these forms of dementia are similar [6].

According to a common view, as dementia progresses, the tendency to develop depression decreases, as patients' criticality to their condition decreases. In patients with Alzheimer's disease, the level of depression increases as dementia develops. in the mild stage of dementia, depression is observed in 10% of patients, in the middle stage of dementia – in 40% of patients, and in the severe stage of dementia – in 60% of patients [6]. These figures show an increase in the prevalence of depression, but the incidence of depression, which reflects the emergence of new cases, remains approximately the same throughout the disease. The increased risk of depression in patients with progressive dementia is not associated with a psychological reaction to cognitive impairment and increased helplessness, but reflects the "expansion" of organic brain damage, which is an excitatory structure important for the development of depression (for example, bluish dots, suture nuclei, amygdala, cingulate gyrus and frontal cortex, etc. It is no coincidence that when a patient is depressed, dementia develops faster and is accompanied by a more pronounced functional deficit, while when comorbid depression is present in the cerebral cortex, patients with Alzheimer's disease have more neurofibrillary glomeruli [9]. It is also important to remember that the symptoms of depression in a patient with dementia can be caused by medications (for example, some corticosteroids, sleeping pills, antihypertensives, estrogens, etc. D.), insufficient activity of the thyroid gland, metabolic disorders. A comprehensive laboratory examination is required, which is not always performed in patients with dementia.

Special difficulties arise when differentiating depression from apathy – this makes it difficult to determine the true frequency and exacerbates depression. Apathy is characterized by a decrease in motivation, interest, emotional dullness, apathy, apathy, and a person's inability to be satisfied. This may be a manifestation of depression, which in this case is accompanied by longing and anxiety, or it may be observed as an independent affective syndrome. With apathy without depression, the patient is not concerned about his condition and increasing helplessness. Apathy without other manifestations of depression is more than twice as common in patients with dementia and Parkinson's disease than apathy in depressive syndrome [6]. In Alzheimer's disease, apathy is found in 42% of patients with mild dementia, in 80% of patients with moderate severity, and in 92% of patients with severe dementia [3].

Insensitivity is more often associated with structural changes in the brain than depression, including cognitive impairment, especially the type of regulation and frontal-frontal systems. Like depression, apathy can be a harbinger of dementia, but more accurately predicts the further development of dementia. The severity of apathy correlates more accurately with the severity of dementia. Apathy is one of the main factors that lead a patient with dementia to social isolation, limit the level of his daily activities, and increase the burden of care. Unlike depression, apathy does not respond to antidepressants, it is based on cholinergic and dopaminergic mechanisms rather

than serotonergic ones, although existing cholinomimetics and dopaminergic drugs are not highly effective in apathy [5].

However, the line between depression and apathy may not be so clear. It has been shown that not only apathy, but also the severity of depression in patients with Parkinson's disease are associated with cholinergic denervation of the cortex [6]. On the other hand, symptoms of depression can also respond to dopaminergic effects [3, 6], therefore it is more correct to consider depression and apathy as different poles in a single spectrum of affective disorders. Affective lability and tears may also be a sign of depression in a patient with dementia, but they should be distinguished from strong crying in pseudobulbar syndrome. With depression, there are prolonged episodes of crying that occur against the background of a depressed mood. Crying in pseudobulbar syndrome is unpredictable, usually fleeting and is not associated with constant mood depression. In a patient with dementia, one should always remember about depression with a rapid deterioration in the patient's functional state.

The usual questionnaires used to identify and assess the severity of depression in patients with dementia are not suitable because patients cannot adequately assess their condition. The geriatric depression Scale, designed specifically for the elderly, can be used, but it is difficult to apply in patients with severe cognitive impairment. To date, the best indicator of depression in patients with dementia is the Cornell Depression Scale, which allows surveys among caregivers [7].

There is no doubt that the incorrect recognition of dementia in a depressed patient is a gross mistake that deprives the patient of effective therapy. But experience shows that in clinical practice, ignoring the symptoms of dementia disguised as real depression or pseudodepression is more common in a patient with depression than an incorrect diagnosis of dementia. However, it is difficult to overestimate the importance of early diagnosis of dementia, since it opens the way to effective basic dementia therapy, which allows you to more or less stabilize your mental state, maintain internal independence and delay the moment of helplessness [2].

In any case, it is important to understand that a diagnosis of depression does not exclude a diagnosis of dementia, and a diagnosis of dementia does not exclude a diagnosis of depression. On the contrary, the above data show that depression and dementia often combine with each other, mutually reinforcing each other's influence on the patient's daily life and at the same time "masking" each other. Difficulties with diagnosis lead to the fact that less than a quarter of patients with dementia and depression receive adequate antidepressant therapy [8].

To avoid mistakes, patients with dementia should be regularly screened for depression, and patients with depression, especially in the elderly, are shown a neuropsychological examination. Assessment of the neuropsychological profile can be the key to the diagnosis of dementia, for which a short scale of mental state can be used, as well as a set of tests, including the "drawing clock" test, speech activity, visual-spatial or auditory examination of speech memory [5]. When treating with antidepressants, it is necessary to take into account the dynamics of the cognitive state. Resistance to antidepressants in cognitive disorders or the presence of signs of dysfunction of the temporomandibular structures (for example, low semantic speech activity, impaired recognition in memory tests) indicates the development of cognitive disorders associated with organic brain damage.

When studying the effectiveness of antidepressants in patients with depression with cognitive impairment, the following results were obtained. In patients with dementia, depression contributes to behavioral change, reduces the level of daily independence, increases the burden on caregivers, increases mortality and, of course, requires treatment. However, the results of antidepressant treatment in patients with dementia vary. Several studies have shown that antidepressants can improve the affective state of a patient with dementia, reduce behavioral disorders such as agitation, aggression or irritability, and improve the quality of life of patients and relatives [9].

However, in patients with dementia, the effect of antidepressants is reduced compared to patients without cognitive impairment. Since depression may not be the only cause of poor functioning in patients with cognitive impairment, antidepressants tend to improve functional status less than primary depression. Patients with vascular dementia with impaired regulation of cognitive functions are more resistant to antidepressants [3]. In addition, in patients with dementia, the response to antidepressants may be slower, and it takes 6-8 weeks for it to develop. The duration of this period is often explained by the need for slow titration of the dose in order to avoid side effects in the elderly.

Relapses of depression are more common in patients with cognitive impairment, which requires longer-term therapy, as well as a combination of pharmacotherapy with psychotherapeutic help [8].

Anticholinergic drugs such as tricyclic antidepressants or paroxetine should be avoided in the treatment of depression in patients with dementia. Tricyclic antidepressants have been shown to not only impair cognitive function, causing cardiovascular dysfunction, but also contribute to an increase in leukoencephalopathy in patients with vascular dementia. In particular, the most studied selective serotonin reuptake inhibitors citalopram and sertraline are

recommended for the treatment of depression in dementia in this category of patients. If symptoms of depression are persistent, a patient with dementia should add a cholinesterase inhibitor or memantine to an antidepressant, and in the presence of psychotic disorders, an atypical antipsychotic should be added [2].

Long-term prospective studies show that in patients with advanced dementia (for example, Alzheimer's disease), the use of antidepressants can improve cognitive function in the short term, but ultimately does not affect the trajectory of cognitive decline. In this regard, it is important to combine antidepressants with antitumor drugs, which, in turn, helps to improve the affective state. It has been shown that cholinesterase inhibitors can reduce the manifestations of dysphoria, irritability, and affective lability in patients with Alzheimer's disease and thus reduce the grief of caregivers.

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ОСОБЕННОСТИ ПРОЯВЛЕНИЯ ДЕПРЕССИВНОГО РАССТРОЙСТВА У БОЛЬНЫХ ДЕМЕНЦИЕЙ

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Резюме. *Деменция и депрессия являются одними из наиболее распространенных психопатологических синдромов среди пожилых людей: деменция наблюдается у 5-10% людей старше 60 лет, когнитивные нарушения-у 15-25%, а депрессивные расстройства-у 20-30%. Взаимосвязь депрессии и слабоумия сложна, и в последнее время научные статьи не до конца освещают взаимосвязь этих патологий и особенности их проявления. В этой статье полностью объясняются особенности проявления депрессивного расстройства у пациентов с деменцией.*

Ключевые слова: *деменция, депрессия, когнитивные нарушения, болезнь Альцгеймера.*