DOI: 10.54538/2707-5265-2024-5-2-14-22

Depression and cardiovascular disease

Sh.F. Odinaev, N.A. Khalikova, D. Sh. Faizieva
Department of Internal Diseases N 1, State Educational Institution "Avicenna Tajik State Medical University"

The review of literature presents current data on the relationship of stress, anxiety and depression with cardiovascular diseases. It is noted that anxiety-depressive disorders are detected in more than half of patients with arterial hypertension and ischaemic heart disease. Affective disorders in patients with cardiovascular pathology increase the risk of complications of the primary disease and increase the mortality rate. An increase in the prevalence of depression with age has been observed.

The possible association between depression and increased cardiovascular morbidity and mortality is discussed. It is indicated that new generation antidepressants such as selective serotonin reuptake inhibitors (SSRIs) are devoid of side effects typical for tricyclic antidepressants and can be used for treatment of cardiovascular patients with comorbid depression. It is also important that SSRIs, unlike traditional tricyclic antidepressants, are acceptable for the treatment of affective disorders in patients with cardiovascular disease in older age groups.

Key words:
Cardiovascular disease, comorbid depression, tricyclic antidepressants, selective serotonin reuptake inhibitors

For citation:
Odinaev Sh.F., Khalikova N.A., Faizieva D.Sh. Depression and cardiovascular disease. Eurasian Scientific and Medical Journal "Sino". 2024; 5(2): 14-22. https://doi.org/10.54538/2707-5265-2024-5-2-14-22

At present, arterial hypertension (AH) is considered as the most common noncommunicable disease, detected in 25-40% of the adult population. About 90-95% of all AH cases are represented by hypertension [1]. Despite obvious successes in its diagnosis and treatment, AH still remains a disease that determines not only disability but also high mortality of working-age people [2]. The contribution of blood pressure (BP) to mortality from cardiovascular diseases is great: life expectancy in men suffering from AH is 8-10 years shorter, in women by 5-6 years [3]. The prognosis in AH depends primarily on the degree of BP elevation, target organ damage and the presence of comorbidities [4]. Depression has become one of the most urgent problems of medicine in recent years. According to WHO estimates of causes of disability and mortality, depression currently ranks fourth in the combined estimates of causes of disability and mortality, and experts predict it will rank second in the coming years, second only to coronary heart disease (IHD) [5].

Research conducted over the last two decades in different countries has shown that depression is common in general practitioners. The prevalence of depressive dis-

DOI: 10.54538/2707-5265-2024-5-2-14-22

Депрессия и сердечно-сосудистые заболевания

Ш.Ф. Одинаев, Н.А. Халикова, Д.Ш. Файзиева

Кафедра внутренних болезней № 1, ГОУ «Таджикский государственный медицинский университет им. Абуали ибни Сино»

В обзоре литературы представлены современные данные о связи стресса, тревоги и депрессии с сердечно-сосудистыми заболеваниями. Отмечено, что тревожно-депрессивные расстройства выявляются более чем у половины больных артериальной гипертонией и ишемической болезнью сердца.

Аффективные расстройства у больных с сердечно-сосудистой патологией повышают риск осложнений основного заболевания и увеличивают смертность. Отмечено увеличение распространённости депрессии с возрастом.

Обсуждается возможная связь депрессии с повышенной сердечно-сосудистой заболеваемостью и смертностью. Указывается, что антидепрессанты нового поколения, такие как селективные ингибиторы обратного захвата серотонина (СИОЗС), лишены побочных эффектов, характерных для трициклических антидепрессантов, и могут использоваться для лечения сердечно-сосудистых пациентов с коморбидной депрессией.

Важно также, что СИОЗС, в отличие от традиционных трициклических антидепрессантов, приемлемы для лечения аффективных расстройств у пациентов с сердечно-сосудистыми заболеваниями в старших возрастных группах.

Ключевые слова: *сердечно-сосудистые заболевания, коморбидная депрессия, трициклические антидепрессанты, селективные ингибиторы обратного захвата серотонина*

Для цитирования: *Одинаев Ш.Ф., Халикова Н.А., Файзиева Д.Ш. Депрессия и сердечно-сосудистые заболевания. Евразийский научно-медицинский журнал «Сино». 2024; 5(2): 14-22. https://doi.org/ 10.54538/2707-5265-2024-5-2-14-22*

orders varies by country and region of residence, but average estimates suggest that depression occurs in 10-20% of primary care patients. Therefore, depression has now moved beyond the boundaries of neurology and psychiatry and can reasonably be regarded as a general medical problem [3, 6]. The relationship between stress, anxiety, depression and cardiovascular diseases has been known for a long time, however, only in recent decades this relationship has been confirmed from the perspective of evidence-based medicine [3, 4]. The findings of one of the first researches by R.Carney published in 1988 show that the mortality rate in patients with myocardial infarction and depression is 3-6 times higher than in patients with myocardial infarction with no signs of depression [7]. Further a number of studies by S.N. Tereshenko et al. (2009) confirmed the relationship between affective spectrum disorders and arterial hypertension. In large prospective studies it was shown that depression and anxiety are independent risk factors affecting

both the occurrence and development of arterial hypertension and survival prognosis of patients with cardiovascular pathology [8,]. It was noted that the frequency of depression and anxiety in arterial hypertension is much higher than in other psychosomatic diseases. It is shown that anxiety-depressive disorders are detected in more than 50% of patients and more often accompany arterial hypertension with crisis course, often complicated by strokes and myocardial infarctions. The comorbidity of depression and IHD ranges from 18 to 60% [8].

A large prospective (3-year) multi-purpose study on the influence of depression on the course and prognosis of cardiovascular diseases - COORDINATA (clinical and epidemiological programme to study depression in cardiological practice in patients with arterial hypertension and coronary heart disease) was conducted in Russia. 5038 patients with AH/IHD were studied. Significant anxiety symptoms occurred in 33% of patients, depressive disorders - in 38% of patients. The results of the study strongly suggest that the presence of depressive, anxiety and mixed anxiety-depressive symptoms in patients with AH/IHD increases the risk of cardiovascular accidents and death by 1.5-2 times [3].

Another large-scale epidemiological study COMPAS (Clinical Epidemiological Programme for the Study of Depression in the Practice of General Practitioners) included 10 541 patients. A high prevalence of depressive spectrum disorders (45.9%) and depressive states (23.8%) was found among patients in the general medical network [6].

In 2001, A.B. Smulevich found that IHD patients with signs of depression have a 1.5-6 times higher mortality rate than patients with IHD having no depressive manifestations [9].

In 2005, D.E. Bush, analysing in detail 17 studies providing for the assessment of the relationship between depression, found a high consistency of the results of these works, which indicated a threefold increase in mortality in patients with depression [10].

A significant effect of depression on the increase in the incidence of CVDs development was noted in a series of studies by A.B. Smulevich, and a significant reduction of affective disorders was noted as the somatic condition improved [9].

The results of meta-analysis of data from 10 large studies performed by L.R. Wulsin and V.M. Singal (2013) also indicate an increase in the level of mortality from cardiovascular diseases in persons suffering from depressive disorders, compared with that in the general population [11]. Moreover, it was found that the increased risk of CVDs mortality in individuals with affective disorders cannot be explained only by behavioural factors associated with depression, such as smoking, dietary errors, lack of physical activity, etc. [4].

A 4-year prospective study (Robinson R.G. 2003), which involved about 3000 people aged 55-85 years, showed that the presence of depression increases the risk of coronary death both in patients with IHD and in people without coronary pathology. The more pronounced depressive disorders are in patients with IHD, the greater is the risk of coronary disorders; in case of low severity of depression the risk increases by 1.6 times with pronounced depression by 3 times [12]. In 2018, G.V. Pogosova studied hypertensive patients in the presence of anxiety-depressive disorders and noted that depression more than 2 times increases the risk of developing the most formidable complication of hypertension - cerebral stroke and is an independent predictor of cardiovascular death [13, 14].

The importance of depression as a predictor of fatal cardiovascular complications is particularly clear in the population over 60 years of age. According to Grace S.L. (2005), in elderly patients with arterial hypertension, depression significantly increases the risk of myocardial infarction, stroke and death. In

men over 70 years of age, this probability increases twice as much [5]. The state report on the state of health of the population of the Russian Federation (2016) noted that the incidence of depression in the elderly (60-74 years old) is two times higher, and in persons over 75 years old - six times higher than in the young [15]. Depression is not only the leading cause of increased mortality from CVDs, but also significantly worsens the quality of life of patients. An increase in the prevalence of depression with age by 1.2 times for each decade of life was also noted in other studies [16]. Depressive disorders were observed in more than half of elderly patients with CVDs, and the highest rate was observed in chronic heart failure - 61%, stroke - 59%, IHD - 57% and arterial hypertension - 52% [6].

The mechanisms of the negative impact of depression on the course and prognosis of CVDs are being actively studied and have not yet been definitively established. Accumulated data suggest several possible variants elucidating the relationship between depression and increased cardiovascular morbidity and mortality. At the present stage, changes in serotoninergic systems are considered as the most promising subject of research into the interaction between depression and cardiovascular diseases. The study of the molecular basis of the pathogenesis of depression has revealed that in depression patients there is a deficiency of monoamines and, above all, of the 'good mood' mediator serotonin. Serotonin plays a key role in many psychological and physiological processes, including the regulation of appetite, mood, anxiety, cognitive function and wakefulness, as well as the contractility of vascular smooth muscle [17]. Studies of anxiety-depressive states have revealed many abnormal changes in the function of serotonin reuptake, transport and binding to central (brain) and peripheral receptors [5, 18], as well as impaired platelet activation [17, 19]. Moreover, reduced serotoninergic function in the central nervous system is associated with impaired hypothalamic-pituitary-adrenal stress response, which leads to an increased risk of IHD and death [8]. In the supposed connection of affective disorders with inflammatory processes, the greatest attention is paid to the increase in the levels of proinflammatory cytokines - interleukins (IL-1 and IL-2) in depression.

IL-1 and IL-2 stimulate corticotropin-releasing hormone secretion, which leads to increased release of adrenocorticotropic hormone and glucocorticoids [20]. It is known that immune inflammation plays an important role in most stages of atherosclerosis from the initial accumulation of leukocytes to the formation of atherosclerotic plaques [21]. Angiotensin-converting enzyme (ACE) is a membrane-bound endopeptidase. It is involved in the metabolism of numerous small peptides, such as angiotensin II or bradykinin generation, which play an important role in the regulation of vascular tone and cardiac function. In addition, angiotensin II has a proinflammatory effect on the vascular wall, inducing the production of cytokinins and adhesion molecules, aggravating endothelial dysfunction and promoting atherogenesis [22].

Modern studies have shown that the same allelic combination of two genes - ACE ID/DD genotype and G protein (GBPTT) genotype - is simultaneously associated with increased risk of myocardial infarction and predisposition to depression, which may be one of the explanations for the comorbidity of depression with CVDs [20, 23]. In addition, the data obtained suggest that a single nucleotide polymorphism in the promoter region of the ACE gene (254291) may be a common pathophysiological link between depression and CVDs [14].

Moreover, it has recently been shown that ACE inhibitors, which are one of the key classes of drugs in the treatment of CVDs, can have

antidepressant effects, which indirectly confirms the common pathogenetic mechanisms underlying CVDs and depression [24].

Excessive production of aldosterone by the adrenal cortex, which plays an important role in the formation of AH, IHD and CHF, is no less characteristic feature of depression, its, sort of, business card, because in depressed patients, even without cardiovascular pathology, the blood level of this mineralocorticoid is significantly increased. This allowed to consider hyperaldosteronism as a marker of depression [9].

The linking pathogenetic link between IHD and depression may be the functional state of platelets. D.L. Musselman (1998) first suggested that platelet dysfunction is a direct reaction to psychological stress. An increase in markers of platelet reactivity with an increase in their aggregation activity was found in patients with depression. Serotonin has been shown to be a platelet agonist and potentiates the effect of other agonists. It has also been shown that stress leads to haemoconcentration due to plasma reduction [18]. An association between increased blood viscosity and the risk of cardiovascular complications has been revealed [25].

There is evidence that in patients with IHD, coronary vasospasm is more often observed in endothelial dysfunction. When IHD is associated with depression, the possibility of serotonin - mediated stimulation of endothelial nitric oxide (NO) secretion and coronary dilatation is reduced [26].

Depression interferes with the fulfilment of medical recommendations on lifestyle changes, diet, smoking cessation, and motor activity. It limits contacts with other people, leads to psychological disadaptation of patients and, in general, sharply reduces adherence to treatment and, in turn, is an important and independent risk factor for adverse outcomes in cardiovascular diseases [9,13].

The first drugs that were used as specific agents for the treatment of depression appeared in the late 1950s. For a long time, monoamine oxidase inhibitors (MAOIs) and tricyclic antidepressants were the two main 'typical' groups of antidepressants. Initially, it was believed that the main mechanism of their antidepressant action was the activation of noradrenergic transmission. Over time, their influence on central serotoninergic processes, namely their ability to inhibit neuronal serotonin reuptake, began to play a major role in the mechanism of antidepressant action.

Traditional antidepressants are widely used not only in psychiatry, but also for the treatment of a number of somatic diseases with comorbid depression [27]. However, their use in somatic patients has been extremely limited due to their cardiotoxic effects. A number of tricyclic antidepressants have pronounced cholinolytic activity, which makes their use in prostatic hypertrophy, bowel and bladder atony, glaucoma and cardiovascular diseases difficult. Undesirable effects when prescribing tricyclic antidepressants are mainly due to the fact that they inhibit simultaneously reversible neuronal capture of different neurotransmitter amines (norepinephrine, dopamine, serotonin). According to a number of authors [28] from 20 to 60% of patients with arterial hypertension interrupt treatment with antidepressants mainly because of their side effects, such as orthostatic hypotension, intracardiac conduction disturbance, dizziness, dry mouth, impaired accommodation, urinary retention, associated with the pronounced cholinolytic effect of tricyclic antidepressants. In addition, unfavourable interaction of drugs of this group with beta-adrenoblockers, cardiac glycosides, antiarrhythmic agents has been noted, which also limits their use in patients with cardiovascular diseases.

New generation antidepressants - selective serotonin reuptake inhibitors (SSRIs) are

devoid of the side effects characteristic of tricyclic antidepressants, and their own side effects are rare and usually do not require drug withdrawal [28, 29].

The data of experimental studies indicate: administration of SSRIs significantly increases the amplitude of synaptic plasticity, promotes normalisation of brain metabolism, activates neuronal vital activity and its functioning. It has been shown that SSRIs increase the reactivity of the hypothalamic-pituitary-adrenal system in response to stress, increasing the number and length of apical dendrites of pyramidal cells and increasing the volume of hypocapm. This mechanism is believed to underlie the resulting anti-anxiety and anti-stress action [30].

The results of studies on the use of SSRIs in hypertensive patients are of interest [31-33]. It has been shown that the addition of drugs to the therapy recommended for the treatment of AH, in addition to the correction of the psychological status of patients, provides improvement of the general clinical condition of patients, tolerability of therapy and stabilization of blood pressure levels. Long-term (6 months and more) use of SSRIs in patients with affective disorders promotes not only regression of structural and geometric indicators of left ventricular remodelling, but also normalization of its diastolic function, which allows the use of antidepressants in the complex treatment of AH [34].

Analysis of literature data and results of a number of studies have shown that therapy of depressive-anxiety disorders in patients with IHD and CHF is reasonable. It is important that SSRIs, unlike traditional antidepressants, can be used for the treatment of CVDs in elderly patients due to the absence of sedation, orthostatic hypotension and other adverse reactions [16].

The study of anxiety-depressive states in patients with CVDs (AH, IHD and with cerebral circulatory disorders) indicates that comorbid conditions are detected quite often. They often contribute to a faster progression of the underlying disease, dominate in clinical manifestations and are often the cause of its various complications.

It seems to us that a more in-depth study of anxiety-depressive states in cardiovascular diseases is a promising direction, since timely diagnosis and timely treatment and preventive measures contribute to a more favourable course of the underlying disease, prevent various complications, and improve the quality of life of patients. Complex treatment of comorbid conditions contributes to shortening the patient's stay in hospital, prevents premature loss of working capacity, disability, thereby prolonging life expectancy.

It should be noted that this problem is devoted to a relatively small number of works that study the features of comorbid conditions in diseases of the cardiovascular system, especially in older age groups. In Tajikistan, such studies have hardly been conducted. New antidepressants - selective serotonin reuptake inhibitors (SSRIs) have shown high efficacy in the treatment of this category of patients. However, they also need more in-depth study of their pharmacological properties in the treatment of anxiety-depressive states in elderly and senile patients.

The given data of the literature testify to the high prevalence of affective disorders in patients with diseases of the cardiovascular system in general, and in ischaemic heart disease and arterial hypertension in particular. Depressive states in this category of patients worsen the clinical course of the underlying disease, contribute to more frequent occurrence of its various complications, worsen the quality and shorten life expectancy. The use of selective serotonin reuptake inhibitors, which are safe and well tolerated in combination with the main groups of cardiovascular drugs, is a promising direction and dictates the expediency of a deeper study of this issue.

REFERENCES

1. Kozhokar K.G., Urvantseva I.A., Nikolaev K.Yu. The influence of psychosocial factors on the development of coronary heart disease and acute coronary syndrome. Cardiovascular therapy and prevention. 2016; 15(3): 58-62. (in Rus.).
2. Yuldashev R.N., Yunusova Z.V. Depressive disorders and their correction in patients with cardiovascular diseases. Medicine of Kyrgyzstan. 2018; 1: 36-40. (in Rus.).
3. Chazov E.I., Oganov R.G., Pogosova G.V., Deev A.D., Shalnova S.A. and others. Depressive symptoms worsen the prognosis in patients with arterial hypertension and coronary heart disease: the first results of the prospective stage of the Russian multicenter study COORDINATA. Cardiology. 2007; 10: . (in Rus.).
4. Ivanov S.V. Depression and cardiovascular pathology. Cardiology. 2009; 7-8: 115-120. (in Rus.).
5. Grace S.L., Abbey S.E., Kapral M.K. et al. Effect of depression on five-year mortality after an acute coronary syndrome. Am J Cardiol 2005; 96: 1179-1185.
6. Oganov R.G., Olbinskaya L.I., Smulevich A.B., Vein A.M. et al. Depression and depressive spectrum disorders in general medical practice / Results of the COMPASS program / / Cardiology. 2004; 1: 48-54. (in Rus.).
7. Glassman A.H. Depression and cardiovascular comorbidity. Dialogues Clin Neurosci 2007; 9: 9.
8. Chazov E.I., Oganov R.G., Pogosova G.V., Shalnova S.A., Romasenko L.V. Clinical and epidemiological program for studying depression in cardiological practice in patients with arterial hypertension and coronary heart disease (COORDINATE): results of a multicenter study. Cardiology. 2007; 3: 28-37. (in Rus.).
9. Smulevich A.B. Depression in general medicine. M. 2001(in Rus.).
10. Carney R.M., Rich M.W., Freedland K.E. et al. Major depressive disorder predicts cardiac in patients with coronary artery disease. Psychosom Med. 1988; 50: 627-633.
11. Wulsin L.R., Singal B.M. Do depressive symptoms increase the risk for the onset of coronary disease? A systematic quantitative review. Psychosom Med. 2013; 65: 201-210.
12. Scherrer J.F., Xian H., Bucholz K.K. et al. A twin study of depression symptoms, hypertension, and heart disease in middle-aged men. Psychosom Med. 2013; 65: 548-557.
13. Pogosova N.V., Oganov R.G. Clinical and epidemiological program for studying psychosocial risk factors in cardiological practice in patients with arterial hypertension and coronary heart disease (COMETA): first results of a Russian multicenter study. Cardiology. 2018; 58(9): 47-58. (in Rus.).
14. Pogosova G.V. Depression is a risk factor for the development of coronary heart disease and a predictor of coronary death. Cardiology. 2012; 12: 4-11. (in Rus.).
15. Gridina S.A., Povetkin S.V. Comparative assessment of the effect of free and fixed combinations of antihypertensive drugs on the quality of life of patients with arterial hypertension at high and very high risk of cardiovascular complications. Cardiology. 2016; 3: 25-29. (in Rus.).
16. Bondy B. Common genetic factors for depression and cardiovascular disease. Dialogues Clin Neurosci. 2007; 9: 19-28.
17. Marzari C., Maggi S., Manzato E. et al. Depressive symptoms and development of coronary heart disease events: the Italian longitudinal study on aging; Gerontol A. Biol Sci Med Sol. 2005; 60: 85-92.
18. Nemeroff C.B., Musselman D.L. Are platelets the link between depression and ischemic heart disease? Am Heart J. 2012; 140: 57-62.

19.Robinson R.G. Poststroke depression: prevalence, diagnosis, treatment and disease progression. Biol Psychiatr. 2003; 54: 376-387.
20. Brasier A.R., Recinos Eledresi M.S. Vascular inflammation and the renin-angiotensin system. Arterioscler Thromb Vase Biol. 2002; 22: 1257-1266.
21.Me Caffery J.M., Frasure-Smith N. et al. Common genetic vulnerability to depressive symptoms and coronary artery disease: a review and development of candidate genes related to inflammation and serotonin. Psychosom Med. 2006; 68: 187-200.
22.Bush D.E. Post-Myocardial Infarction Depression. Evidence report/technology assessment, no. 123. Ref Type: Serial (Book, Monograph).5-1-2005.
23. Ohira T., Iso H., Satoh S. et al. Prospective study of depression symptoms and risk of stroke among Japanese. Stroke. 2013; 32: 903-908.
24. Musselman D.L., Evans D. L., Nemeroff C.B. The relationship of depression to cardiovascular disease: epidemiology, biology and treatment. Arch Gen Psychiatry. 1998; 55: 580-592.
25. Schlienger R.G., Fischer L.M., Jick H., Meier C.R. Current use of serotonin reuptake inhibitors and risk of acute myocardial infarction. Drug Saf 2004; 27: 1157-1165.
26. Waly E. H. Hypertension and dyslipidemia among type ii diabetic patients and related risk factors and complications. Egypt. J. Community Med. 2018; 36: 31-43.
27. Gavrilov D.V. et al. Accuracy of cardiovascular risk assessment in everyday clinical practice. Preventive medicine. 2021; 24(4): 69-75. (in Rus.).
28.Naumova L.A., Osipova O.N. Comorbidity: mechanisms of pathogenesis, clinical significance. Modern problems of science and education. 2016; 5: 105-105. (in Rus.).
29.Skibitsky V.V. Arterial hypertension and depressive disorders: possibilities of using combined antihypertensive and psychocorrective pharmacotherapy. Arterial hypertension. 2016; 22(5): 505-518. (in Rus.).
30. Naber C.K., Husing J., Wolfhard U. et al. Interaction of the ACE D allele and the GNB3 825T allele in myocardial infarction. Hypertension. 2017; 36: 986-989.
31.Iskenderov B.G., Budagovskaya Z.M., Sisina O.N. The effect of a fixed combination of perindopril and amlodipine on intrarenal hemodynamics and the functional state of the kidneys in patients with essential arterial hypertension. Therapeutic archive. 2013; 5: 78-83. (in Rus.).
32. Kroeze W.K., Kristiansen K., Roth B.L. Molecular biology of serotonin receptors structure and function at the molecular level. Curr Top Med Chern. 2002; 2: 507-528.
33. Lett K.S., Blumenthal J.A., Babyäk M.A. et al. Depression as a risk factor for coronary artery disease: evidence, mechanisms, and treatment. Psychosom Med. 2004; 66: 305-315.
34. Monster T.B., Johnsen S.P., Olsen M.L. et al. Antidepressants and risk of first-time hospitalization for myocardial infarction: a population-based case-control study. Am. J Med. 2004; 117: 732-737.

FINANCING

There was no financial support.

ФИНАНСИРОВАНИЕ

Финансовой поддержки не было.

CONFLICT OF INTEREST

The authors declare no conflict of interest

КОНФЛИКТ ИНТЕРЕСОВ

Авторы заявляют об отсутствии конфликта интересов.

INFORMATION ABOUT THE AUTHORS:

***Odinaev Shukhrat Farkhodovich –** Doctor of Medical Sciences, Associate Professor, Head of the Department of Internal Diseases N1, State Educational Institution "Avicenna Tajik State Medical University".
E-mail: nnnn70@mail.ru
https://orcid.org/0000-0002-4188-5955

Khalikova Nargis Abdurashidovna– Senior Lecturer, Department of Internal Diseases N1, State Educational Institution "Avicenna Tajik State Medical University".
E-mail: nargis.a.khalikova.01@gmail.com

Faizieva Dilafruz Shamsidinovna – Senior Lecturer, Department of Internal Diseases N1, State Educational Institution "Avicenna Tajik State Medical University".
E-mail: faizieva.90@gmail.com

ИНФОРМАЦИЯ ОБ АВТОРАХ:

***Одинаев Шухрат Фарходович** – доктор медицинских наук, доцент, заведующий кафедрой внутренних болезней №1 ГОУ «Таджикский государственный медицинский университет им. Абуали ибни Сино».
E-mail: nnnn70@mail.ru
https://orcid.org/0000-0002-4188-5955

Халикова Наргис Абдурашидовна – старший преподаватель кафедры внутренних болезней №1 ГОУ «Таджикский государственный медицинский университет им. Абуали ибни Сино».
E-mail: nargis.a.khalikova.01@gmail.com

Файзиева Дилафруз Шамсидиновна – старший преподаватель кафедры внутренних болезней №1 ГОУ «Таджикский государственный медицинский университет им. Абуали ибни Сино».
E-mail: faizieva.90@gmail.com

*** Автор для корреспонденции.**