

*Eshmuratov Sardor Eldorovich,**Uralov Rustam Sherbekovich**Samarkand State Medical University, Samarkand, Uzbekistan***CHRONIC STRESS AND DEPRESSION IN PATIENTS RHEUMATOID ARTHRITIS**

Abstract :Rheumatoid arthritis (RA) is an autoimmune rheumatic disease of unknown etiology that affects 0.5–2% of the adult population at the most working age of 35-55 years and is characterized by chronic erosive arthritis and systemic damage to internal organs. The chronic progressive course of the disease and the accompanying lifelong therapy are accompanied by the development of often irreversible complications and side effects, manifested in changes in appearance, sleep disorders, emotional lability, physical discomfort and significant limitation of functionality. Back in 1961, P.Kielholz He noted the importance of physical and psychological maladaptation and a marked decrease in activity in RA in the development of chronic depression, as well as in the formation of a "vicious circle" of mental and somatic sufferin. Characterological features allow some patients to calmly adapt to new living conditions, while others develop psychopathological conditions that require psychological and medical correction.

Key words: autoimmune rheumatic disease, systemic damage, physical discomfort

INTRODUCTION

However, it is erroneous to consider depressive disorders only as the consequences of a severe chronic disease – RA. The presence of depression increases the risk of developing a somatic disease. RA also often develops in people suffering from depression. In particular, according to some authors, 66% of patients RA had depressive disorders even before the onset of arthritis . Modern research shows that RA and depression often have a common provoking factor – chronic psychosocial stress or distress – in accordance with Selye's concept. Diathesis is stressful The model, which has been actively developed in recent years, allows not only to identify a number of common pathogenetic mechanisms of RA and depression, including chronic stress on the background of a specific predisposition, but also to comprehensively consider the effect of psychotropic therapy on both psychopathological symptoms and, possibly, on inflammatory processes and pain. The other, so-called biopsychosocial model, is also applicable to both RA and depression. Epidemiological studies show that mental disorders are more common more than 60% of RA patients. Anxiety-depressive spectrum disorders predominate among them. R.G.Frank et al. report that more than a third of RA patients have signs of "major" depression or dysthymia. In the works of other authors, the frequency of depressive disorders ranges from 11 to 65%, anxious – from, The prevalence of depression in the general population is 5-8%. According to G.Treharne et al. among inpatient RA patients, 11% had suicidal thoughts at least once in their life, including In the case of depression, suicidal ideation was present in 30% of patients. Depressive disorders often remain unrecognized and untreated in RA patients. This is because all attention is focused on the physical aspects of the disease, and depression and anxiety are seen as a normal response to a chronic illness. The detection of depression is also complicated by the fact that some symptoms RA coincides with the manifestations of depression (for example, chronic fatigue, motor retardation, weight loss, insomnia, decreased appetite). In particular, chronic fatigue syndrome It occurs in 80-93% of RA patients. Studies show that fatigue is more

correlated with the severity of pain and the presence of depression, rather than with the inflammatory activity of the disease. In addition, the diagnosis of depression is difficult because a rheumatologist, like doctors of other specialties, often does not have enough awareness and time to discuss with the patient anything other than the problems associated with a somatic disease.

MATERIALS AND METHODS OF RESEARCH

The presence of depression significantly worsens the clinical manifestations, dynamics and prognosis of RA. In patients with RA, due to the presence of depression, there is a more pronounced pain syndrome, a greater number of inflamed joints, and a higher degree of functional insufficiency than in patients with RA without depressive disorders. At the same time, depression can contribute

to inflammation, affect compliance with medical recommendations and, thus, the course and outcome of RA. D.C. Ang et al. showed that, independently Depending on other risk factors, the presence of depression leads to a twofold increase in the probability of premature death in the cohort of RA patients observed for 18 years. This may be due, among other reasons, to a violation of compliance. It is known that the presence of depression in patients with chronic somatic disease increases the risk of incompetence of therapy by three times. Perhaps this is due to the fact that depression leads to a loss of hope for recovery, does not allow you to realize the benefits and effectiveness of the prescribed treatment., It leads to social isolation and limited contact with a doctor. In particular, it was found that patients with RA and depression are more likely to feel terminally ill when compared with patients with RA without depression, even with low severity of systemic disease.

In addition, refusal of treatment is one of the frequent manifestations of suicidal behavior in rheumatology practice, characteristic of patients suffering from depression. Along with this, depression is often associated with cognitive impairments, and patients sometimes forget or do not They understand the importance of taking medications regularly, which leads to a decrease in the effectiveness of therapy and a worse prognosis. In addition to the negative consequences directly related to the somatic condition, depression contributes to a decrease in working capacity, loss of work and an increase in the cost of treating patients. RA. In the work conducted on 356 patients with inflammatory rheumatic diseases, noted that the severity of depression, along with the severity of the underlying disease and physical activity, are independent predictors of disability. A significant proportion of RA patients (25-40%) lose their ability to work in the first five years of the disease. The presence of anxiety-depressive symptoms, along with a severe course of rheumatic disease, increases disability from 25 to 50%.

THE RESULTS AND THEIR DISCUSSION

According to various authors, from 25 to 57% of RA patients have sleep disorders, one of the causes of which, along with high inflammatory activity and pain, are anxiety and depressive disorders. A special psychopathological problem in RA

is the prevalence and severity of cognitive impairments, which, according to some authors, are detected in 30% of RA patients. For comparison, among healthy people matched by age and gender, these disorders were detected only in 7.5% of cases. The pathogenetic mechanisms of cognitive disorders in RA have not been established to date. They are usually unrelated. with the duration of RA, the use of glucocorticoids (GK), the degree of disability. However, it is known that quite often cognitive impairments in RA are caused by the presence of depression. and with adequate therapy, depressive disorders become significantly less pronounced. It is also known that cardiovascular diseases (CVD), including arterial hypertension (AH), are also a risk factor for cognitive impairment. Considering the frequent occurrence of hypertension in RA patients,

we can expect an increase in the number of cognitive disorders associated with a combination of depression and hypertension. Taking into account the well-known fact that the pathogenesis of Alzheimer's disease, characterized by severe cognitive impairment, is based on intrathecal hyperproduction of proinflammatory cytokines (CC), in particular tumor necrosis factor- α (TNF- α), as well as recently obtained data that perispinal extractal weekly 6-month administration of a TNF receptor inhibitor-alpha – etanercept in patients with Alzheimer's disease led to rapid and sustained recovery of cognitive functions, It can be assumed that TNF- α hyperproduction, characteristic of RA, may underlie cognitive impairments in RA. The causes of depressive disorders in RA patients, as well as the causes of the development of RA itself, are ambiguous. It should be noted that depression, like RA, is a chronic multifactorial disease, in the development of which psychosocial, neuroimmune and neuroendocrine factors are involved. Many researchers note the influence of environmental factors acting on the immune and neuroendocrine systems of the body on the occurrence and provocation of RA exacerbations. These factors include, first of all, chronic stress, which precedes the development or exacerbation of RA in 30-87% patients. On the other hand, the modern consideration of the pathogenesis of depression is based on the diathesis-stress model, which emphasizes the role of provoking stress factors in the development of various variants of depression, depending on the nature of the predisposition. Moderate-intensity chronic daily stress is considered a more important predictor than RA activity showed that The presence of depression in RA is not related to the duration and severity of the disease, but significantly correlates with the presence of chronic stress, lack of social support, and degree of functional disability. It has been established that moderate-intensity chronic stress leads to pro-inflammatory shifts in the neuroendocrine system due to the lack of an adequate response from the hypothalamic-pituitary-adrenal system (HPA). On the other hand, acute pronounced stresses, accompanied by an increased release of neurotransmitters and stressful hormones are often associated with a decrease in the activity of the disease. Depressive disorders are also most often provoked by prolonged stress factors. Apparently, moderate -intensity chronic stress is a common pathogenetic

factor leading to the development of systemic rheumatic disease and depressive disorder.

Thus, in conditions of inflammation, causing pain and depression, the central nervous system is initially designed to perform an adaptive role, forcing the body

to function in a more "gentle" mode with limited physical and emotional activity. RA is characterized by a consistently high level of pro-inflammatory CK, when pain and depression become chronic, and the lack of timely and adequate treatment

can lead to irreversible consequences in the form of early disability and an unfavorable outcome.

CONCLUSIONS

Depressive disorders in RA are widespread. Perhaps this is due to the fact that these diseases are characterized by similar causes and mechanisms of development with the significance of chronic stress, as well as the systemic pathogenetic and clinical manifestations, which determines the need for a systematic approach to complex treatment. Further study of predictors, pathogenesis features, and dynamics of anxiety-depressive spectrum disorders in RA patients, as well as Improving psychopharmacological and psychotherapeutic strategies will help not only reduce the contribution of depression to the overall picture of the disease and improve the quality of life of patients, but also significantly improve the prognosis of systemic disease.

LITERATURE:

1. Ravshanova, M., Ibragimov, K., Uralov, R., Xasanov, F., Islamova, K., Abdushukurova, K., ... & Axmedov, I. (2024). Clinical and Immunological Characteristics of Patients with

- Rheumatoid Arthritis on Synthetic DMARDS Therapy. *Frontiers of Global Science*, 2(1), 41-47.
2. Ibragimov, K., Sulstonov, I., & Ravshanova, M. (2024). The Effectiveness of the Combination Therapy with biologic DMARDS in Rheumatoid Arthritis. *Frontiers of Global Science*, 2(1), 17-24.
 3. Ziyadullaev, S. K., Sulstonov, I. I., Dushanova, G. A., & Akbarovna, K. S. (2021). The Effectiveness Of Pharmacotherapy For Dmards With Ra Depending On The C3435t Polymorphism Of The Mdr1 Gene. *Int. J. of Aquatic Science*, 12(3), 2908-2916.
 4. Sobirov, A., & Sulstonov, I. (2024). COMPREHENSIVE ANALYSIS OF CLINICAL NEUROPSYCHOLOGICAL AND NEUROIMAGING ASPECTS OF ALZHEIMER'S DISEASE. *Frontiers of Global Science*, 2(1), 25-29.
 5. Islomovich, S. I. (2024). FEATURES OF THE COURSE OF PREGNANCY IN RHEUMATOID ARTHRITIS. *International journal of medical sciences*, 4(10), 77-84.
 6. Islomovich, S. I. (2024). Gender characteristics of the current rheumatoid arthritis. *International journal of medical sciences*, 4(10), 3-8.
 7. Khasan, I., Ibrat, A., Islomovich, S. I., Sukhrobovna, R. M., & Shuxrat, Z. The Association Between Cardiovascular Disease and Conventional DMARDs in Patients with Rheumatoid Arthritis. *International journal of health sciences*, 6(S8), 5053-5059.
 8. Тоиров, Э. С., Ахмедов, И. А., & Султонов, И. И. (2020). Дисбаланс нервной и эндокринной системы при ревматоидном артрите. *Journal of cardiorespiratory research*, 1(2), 73-76.
 9. Хамраева, Н. А., Султонов, И. И., & Хасанов, Ф. Ш. У. (2019). Кожные проявления у больших системной красной волчанкой. *Вопросы науки и образования*, (28 (77)), 128-131.
 10. Sulstonov, I. I., Kh, Z. S., Ruzybakieva, M. R., Kireev, V. V., Aripova, T. U., & Suyarov, A. A. (2021). Pharmacogenetic Aspects of Drug Resistance in Rheumatoid Arthritis. *Annals of the Romanian Society for Cell Biology*, 25(3), 4147-4150.
 11. Тоиров, А. Э., Султонов, И. И., & Тоиров, Э. С. (2020). ЗНАЧЕНИЕ ДИСФУНКЦИИ ПОЧЕК У БОЛЬНЫХ ОСТРЫМ ИНФАРКТОМ МИОКАРДА НА ФОНЕ САХАРНОГО ДИАБЕТА 2-ГО ТИПА. *Вестник науки и образования*, (9-3 (87)), 86-91.
 12. Kireev, V. V., Sulstonov, I. I., Ziyadullaev, S. K., Suyarov, A. A., Aripova, T. U., Usmanbekova, K. T., & Nasretdinova, M. T. (2021). Genetic Engineered Preparations-An Innovative Approach in the Treatment of Rheumatoid Arthritis. *Annals of the Romanian Society for Cell Biology*, 25(3), 4114-4119.
 13. Намраева, Н. А., Султонов, И. И., & Hasanov, F. S. (2020). Systemic lupus erythematosus treatment strategy. *Journal of Critical Reviews*, 7(9), 269-270.
 14. Иргашева, У. З., Султонов, И. И., & Тоиров, Д. Р. (2013). Признаки дебюта системной красной волчанки. *Академический журнал Западной Сибири*, 9(1), 15-15.