As a result of adipokine level assessment, it was revealed that adiponectin in the main group was significantly lower (6.633 ± 0.016 ng/ml, $p<0.001$) than in the comparison group (7.977 ± 0.046 ng/ml, $p<0.001$). The leptin level was significantly higher in case of comorbidity of AG and T2D as compared to hypertensive patients without T2D (16.346 ± 0.142 ng/ml and 12.306 ± 0.185 ng/ml, respectively, $p<0.001$). Patients with hypertension and diabetes demonstrated close relationship between changes in adipokine levels and increase in body mass index (BMI).

Patients with BMI of 25-34.9 kg/m^2 had significantly higher leptin levels (17.766 ± 0.085 ng/ml) as compared to patients with normal body weight (13.080 ± 0.149 ng/ml), which may indicate the presence of leptin resistance in patients with excessive weight and first-degree obesity. The adiponectin level in patients with normal body weight was significantly lower than in patients with BMI 25-34.9 kg/m^2 (6.315 ± 0.022 and 6.770 ± 0.013 ng/ml, respectively, $p<0.001$), which can be regarded as its counter-regulatory increase at the initial stages of weight gain. In patients with hypertension without T2D, the adipokine imbalance was similar to the group of patients with concomitant diabetes. However, in patients with hypertension without T2D, but with IR, adiponectin tended to decrease, but decrease thereof was not significant (unlike the comorbidity group). At the same time, the other adipose tissue hormone, leptin, was significantly higher in patients with IR (13.307 ± 0.428 vs. 12.089 ± 0.198 ng /ml, $p <0.05$).

Conclusions: Based on the analysis provided, the relationship between adipokine levels and the development of IR in case of hypertension was revealed. Patients with hypertension and concomitant T2D are characterized by hyperleptinemia and hypoadiponectinemia, the severity of which differs depending on BMI.

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ASPECTS OF COMORBIDITY OF DEPRESSIVE DISORDERS AND SOMATIC DISEASES

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Depressive disorders are a condition that is often found in primary medical practice. It is proved that with somatic diseases the prevalence of depressive disorders is from 22 to 33% (pathology of the cardiovascular, endocrine system, neurological diseases, oncopathology, obstetric and gynecological conditions). The prevalence of depressive disorders in the population is currently increasing.

Common risk factors for depressive disorders relate to patients with somatic diseases. These are genetic, family, personality factors, as well as

negative life events (job loss or worsening social status). Additional risk factors are previous depressive episodes and certain types of somatic illness. Severe depressive disorders occur in patients with severe somatic diseases that are accompanied by severe pain or lead to disability.

We can distinguish the following main provisions regarding depressive disorders in somatic patients:

in many patients, depressive disorders do not develop, which indicates that somatic pathology does not explain the development of depression; depressive disorders accompanying the course of somatic diseases worsen psychosocial functioning and often complicate medical rehabilitation and treatment; treatment of depressive disorders with somatic pathology must be treated simultaneously; untimely started treatment of depressive disorder worsens the prognosis of both depression and somatic disease; suicide in depressive disorders is higher in certain somatic diseases (cancer, AIDS).

Depression develops in more than 50% of patients in the acute period after a stroke. Often, depressive disorders occur in patients with damage to the anterior left hemisphere of the brain, with dysphasia, episodes of major depression or a history of cerebrovascular disease.

About half of depressive disorders in patients with Parkinson's disease meet the criteria for major depression. Depressive disorders in Parkinson's disease may be predictors of future dementia. More pronounced intellectual decline, especially with damage to the frontal lobes of the brain.

For patients suffering from heart disease, there is a high risk of developing depressive disorders. Somatic symptoms, such as chest pain and palpitations, can be significantly expressed in depressive disorders. Often these symptoms are regarded as manifestations of a cardiovascular disease, which greatly complicates the treatment of the disease.

There are numerous hypotheses that explain the biological mechanisms that can cause somatic illness to cause depressive disorder. For example, in a somatic disease, the level of neurotransmitters in the brain (for example, serotonin) often decreases, the immune system weakens, and depressive symptoms develop as a result of side effects of drugs used in the treatment of somatic disease. Drugs that can cause depressive symptoms include: anticonvulsants (phenobarbitone), antihypertensive drugs (lipophilic beta-blockers), antiarrhythmic drugs (digitalis), some antibiotics, lipid-lowering drugs (statins), chemotherapy for cancer (methotrexate), H2-blockers (cimetidine).

A patient survey is a key point in the diagnosis of suspected depressive disorder in somatic patients. It is important to collect a complete medical history, then move on to specific issues and conduct appropriate screening studies.

Some patients do not feel depression, describe their condition as somatic discomfort. Sometimes depressive symptoms can be minimally

expressed and the doctor's attention is focused on somatic disease. Denying the presence of somatic pathology can lead to an underestimation of the accompanying psychopathological symptom. Patients with somatic diseases are reluctant to report depressed mood, fearing that this could adversely affect treatment. Such behavior is especially likely in cases where relatives require the creation of an atmosphere of optimism. If patients want to discuss their bad mood, relatives and healthcare providers should not interrupt and underestimate their symptoms.

The presence of a serious somatic pathology can mask a depressive disorder, because many symptoms (fatigue, loss of appetite) occur in both conditions.

Generally, depressive disorders are successfully treated by primary care physicians. But sometimes, some patients may require consultation or treatment in a specialized hospital.

Antidepressants must be used to treat any depressive state. It is important to suggest a possible interaction between antidepressants and drugs that are often used in the treatment of somatic diseases. Doctors should be aware of interactions that induce or inhibit liver enzymes involved in the metabolism of drugs, such as cytochrome P 450. Side adverse effects can also result from the interaction of selective serotonin reuptake inhibitors (SSRIs) with heterocyclic antidepressants, monoamine oxidase inhibitors (MAOIs), theophylline, β-blockers, antipsychotic drugs. During the treatment of depression ву MAOI, hypertensive reactions may occur while using vasoconstrictors, stimulants, sympathomimetics, drugs, and eating foods rich ву tyramine.

Thus, depressive disorders are often found in people with concomitant somatic pathology and complicate the course of both conditions. It is important to know that treatment of depressive disorders in somatic patients is effective and should start from the moment of diagnosis. Close collaboration between medical specialists is necessary to improve the diagnosis, treatment and further prevention of depressive disorders in somatic patients.

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THE USE OF HERB ALTHEA OFFICINALIS ROOT IN PATIENTS WITH COPD AND COEXISTING HYPOACIDIC GASTRITIS

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Inroduction. Althea officinalis belongs to malvaceae family, the use of plants from the Malvaceae family for herbal therapy is very common in the Middle East. Althaea officinalis is native to Asia, Europe and United States of