In 39.99% of surveyed there a mutation in the promoter region of genes studied the GST (20.55 % in patients with tuberculosis and 16.44 % healthy), among them, more than half (64.81 %) are carriers of pathological 0/0 genotype GSTM1 gene haplotype, whereas the combination of a homozygous mutation of the GSTT1 0/0 occurs in 2.33 times less, and there is almost one in three (27.78 %) of the surveyed. 4.17 % of patients with pulmonary tuberculosis are carriers of abnormal genotypes of both isoforms of GST genes. So, the favorable combination of functional alleles in the haplotype is characterized by the frequent occurrence of lung VDTB to 26,09 % ($\chi^2$22 = 4,37 p = 0,037) in a mild clinical course, against the backdrop of a liquid co- and polymorbidity (on 31,01 %, (χ^2 = 5.53, p = 0.019) and 31,38 % (χ^2= 4,07, p = 0.044), respectively) and more likely to bacteriological 60 dose of 18,40 % (χ^2 = 3,59, p = 0.052) and 45,64 % (p = 0.002), respectively.

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DYNAMICS OF CYTOKINE REGULATION IN PATIENTS WITH DRUG RESISTANT TUBERCULOSIS

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Tuberculosis (TB) remains a global threat of mankind. One of the features of modern TB is the increase in the prevalence of drug-resistant mycobacteria (MBT) to TB drugs, which leads to a decrease of quality of care and, as a consequence, to increase of mortality. A global WHO survey stated that among all new TB cases 3.6% are multidrug-resistant tuberculosis (MDR); in 9.6% (8,1-11,2%) of all MDR cases, XDR is reported; 60.0% of all MDR cases in the world are diagnosed in Brazil, China, India and South Africa. The high level of primary MDR TB, exceeding 6.5%, was registered in Kazakhstan, Russia (Tomsk region), Uzbekistan, Estonia, Israel, China (Liaoning and Henan Province), Latvia, Lithuania, Ukraine (Donetsk region).

The study included 107 patients with pulmonary tuberculosis, with newly diagnosed pulmonary tuberculosis (NDTB), with preserved sensitivity to TB drugs; MDR and XDR TB patients. Clinical, radiological, bio- chemical, microscopic, microbiological, immune-enzymatic and statistical study (ANOVA and Pearson correlation) methods were used.

Pearson correlation analysis between pro- and anti-inflammatory cytokines showed that in patients with MDR TB there is a weak, negative correlation between the levels of IL-6/IL-10 and IL-18/IL-10 (r= -0.22, p<0,001, and r= -0.16, p<0.001, respectively). Production of IL-6 and IL-10 in TB patients is independent of drug resistance, but increases in response to increased synthesis of endotoxins by MBT; the magnitude of endogenous intoxication and cytotoxic hypoxia creates prerequisites for the development of drug resistant strains. The IL-18/IL-10 ratio in these patients characterizes the increase in severity of the patient's state, the spread of inflammation processes in the lungs and the development of drug resistance; there is a significant bulk of the Tx-lymphocyte type 2 (CD4+), which indicates the development of deep gap in cell-mediated immune response and prevalence of an ineffective anti-inflammation immune activation.

So, **c**omprehensive assessment of integral indices of endogenous intoxication and level of certain pro- and anti-inflammatory cytokines in the blood plasma of patients with MDR TB show a moderate endogenous intoxication, break down of the cellular component of the immune reactivity due to the formation conditions for the development of MBT resistance MBT, by increase of cytotoxic hypoxia and activation of „systemic inflammatory response" syndrome. Analysis of plasma concentration of IL-6, IL-10 and IL-18 in patients with multidrug-resistant TB proved, that their level depends on the nature of the resistance of MBT and correlate with the spread of the specific process in the lungs.

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THE METABOLIC SYNDROME IN HIV-INFECTED PATIENTS WHO ARE HAVING A HIGHLY ACTIVE ANTIRETROVIRAL THERAPY SESSION

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Highly active antiretroviral therapy (HAART) transforms an infection with the human immunodeficiency virus (HIV) from a rapidly progressive and uniformly fatal disease into a chronic manageable condition. However, some HAART schemes application, especially those including protease inhibitors (PIs), causes in the majority of HIV-infected patients an iatrogenic metabolic syndrome (MS) (increased waist size, impaired fasting glucose, hypertriglyceridemia, low plasma high-density lipoprotein concentration, and hypertension) that is associated with an increased risk of cardiovascular cases that are connected with a process of accelerated atherosclerosis, even in young HIV-infected people. Metabolic complications and abnormal fat distribution were frequently observed after a few years of antiretroviral therapy and, as the wide range of antiretroviral drugs is still expanding, and long-term metabolic changes are becoming more common throughout the world. Nearly 20% of HIV-infected patients receiving treatment meet the criteria for the third report of the expert group on the detection, evaluation and treatment of hypercholesterolemia in adults (Third Report of the Adult Treatment Panel for MS), and about 50% for two out of five diagnostic criteria.

The etiology and pathogenetic mechanisms of the HIV / HAART-associated metabolic syndrome remain not fully clarified. There is controversial evidence as to which class of antiretroviral drugs can cause such changes. PIs is the most likely class, but the use of nucleoside reverse transcriptase inhibitors (NSAIDs) may also be a potential risk factor. In addition, PIs and NIDDs can work synergistically, or an effective HIV treatment can contribute to the