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«Actual Problems Of Clinical And Theoretical Medicine»

the first 3, 6 and 12 hours there were determined for the level of enzymes activity: AST (aspartate aminotransferase), ALAT (alanin aminotransferase).

Results. In 17 patients (77%) there was observed polymorphocellular leukocytosis (mean value - 35±3,3××⟦10⟧^9/l, which indicates nonspecific reactions of an organism in response to myocardial damage. In 19 cases (86%) we found the shift of leukocyte formula to the left, at the expense of band neutrophils. In 21 patients (95%) we noted an increase in the activity of enzymes. The average of indicates were the next: 3 hours AST was 69.2±7,1 IU/l, ALAT – 55,2±4,3 IU/l, 6 hours AST – 57,1±3,2 IU/l, ALAT – 81,5±1,9, 12 hours – AST-105,4±9,1 IU/l, ALAT - 60,3±1,5 IU/l. However, it should be noted that only 18 patients (81%) had confirmed acute myocardial infarction (the diagnosis was based on clinical, objective, laboratory datas).

Conclusions. Thus, leukocytosis, neutrophilia with a pronounced left shift, hyperfermentemia AST, ALAT were found in almost all patients in acute coronary syndrome, indicating a lack of specificity of these indicators.

Melenevych A. Ya.

IMPLICATION OF INTERLEUKIN-18 IN THE PATHOGENESIS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN COMBINATION WITH HYPERTENSION

Research advisor: Kapustnyk V.A., Doctor of Medical Science
Department of internal and occupational diseases, Kharkiv National Medical University, Kharkiv, Ukraine

Actuality. Chronic obstructive pulmonary disease (COPD) patients are at a high risk of developing cardiovascular diseases. Airflow limitation is a predictor of future risks of hypertension and cardiovascular events.

Aim of the investigation was to reveal the implication of Interleukin-18 (IL-8) in the pathogenesis of COPD in combination with hypertension.

Materials and methods. A number of population studies have shown that airflow limitation as measured by the forced expiratory volume in one second (FEV1) or the FEV1/forced vital capacity ratio is a predictor of future risks of hypertension and cardiovascular events. The other review described that for every 10% decrease in FEV1, all-cause mortality increases by 14%, cardiovascular mortality increases by 28%, and nonfatal coronary

events increase by almost 20% (Yuki Imaizumi, Kazuo Eguchi, Kazuomi Kario, 2014).

Results. COPD is characterized by a low grade systemic inflammation, closely associated with its extrapulmonary determinations. The relation of inflammation with cardiovascular disease in patients with COPD is complex. Circulating mediators, such as cytokines and C-reactive protein are capable of inducing endothelial dysfunction that initiates and then contribute to the progression of atherosclerosis. The activation of endothelium results in increase endothelial permeability with uptake of oxidized low-density lipoprotein into the atherosclerotic plaque which leads to rupture and thrombus formation with the development of heart attacks and strokes. IL-18 belongs to the interleukin-1 cytokine family, is characterized by proinflammatory activity, and activates the specific immunity. IL-18 induces the production of interferon-γ (IFN-γ) in T cells, macrophages, and NK cells and promotes differentiation of T cells, underlying the development of proinflammatory and proatherogenic immune response.

Several studies have shown that serum IL-18 levels have negatively correlated with FEV1 in COPD patients. Overexpression of IL-18 and IFN-γ-inducing factor lead to severe emphysematous changes and chronic inflammatory changes characteristic of COPD.

Conclusion. Future studies of the role and mechanism of participation of IL-18 and other cytokines in the formation of COPD in combination with hypertension will open new opportunities for effective targeted methods of early prevention and treatment of comorbid pathology.