



## Cytokine Profile in the Prognosis of Coronary Heart Disease Comorbidity in Chronic Obstructive Pulmonary Disease

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<b>ABSTRACT</b>	The objective of the to determine the relationship between cytokine levels and the clinical course of CHD with COPD.
<b>Keywords:</b>	cytokine levels

**Introduction.** Chronic obstructive pulmonary disease (COPD) is a multifactorial widespread disease with a complex mechanism of formation of individual phenotypes, which has an important medical and social significance due to its high morbidity and mortality, as well as long periods of temporary disability, frequent disability and high cost of treatment [1, 9, 13]. Among individuals older than 40 years COPD occurs in 8.2-10.1 % of cases and in 14.2 % of those older than 65, combined with cardiovascular diseases in 56.6-71.4 % of cases [8, 12]. In 10 years, one in four COPD patients

becomes disabled, with a life expectancy of about 8 years [1, 4, 5]. COPD kills 2.75 million people every year and by 2022 it could be the third leading cause of human death after stroke and myocardial infarction [2, 3, 16, 18].

COPD patients have a high prevalence of heart failure, arterial hypertension (AH), arrhythmias and myocardial ischaemia [6, 7, 15]. On average, among patients with COPD, AH is diagnosed in 62.2% of cases, coronary heart disease (CHD) in 27%, carotid atherosclerosis in 43.6% and chronic heart failure (CHF) in 23.6% [9, 14, 22]. According to large studies, COPD

patients have a 2-3-fold increased risk of mortality from cardiovascular disease [8, 9, 10]. In patients with COPD, the course of CHD is difficult to detect [13, 14, 15]. The clinical course of CHD in patients with COPD can manifest itself in three main clinical variants: angina (11%-43%), bronchoobstructive (10%-24%), pain-free (47-88.4%) [22, 28]. For patients with COPD in combination with CHD, the most characteristic feature is prolongation of myocardial ischaemia-free time [23, 25, 30]. With chronic hypoxia, the threshold of pain sensitivity increases and this contributes to the atypical course of CHD in patients with COPD. In the work of E.N. Chicherina noted that a mild degree of bronchoobstruction in every fourth COPD patient is accompanied by myocardial ischemia, and a moderate degree in every third patient, with painless episodes predominating [27, 29].

CHD is one of the most common CVDs caused by atherosclerotic lesions of coronary arteries and is characterized by a clinically undulating course with alternating phases of exacerbation and remission [10, 12, 28]. In pathogenesis of CHD one of the hypotheses is the inflammatory theory, in which local and systemic inflammatory processes play an important role in the process of formation, damage of stable ASD with subsequent occlusion by thrombotic deposits in CA lumen and development of CHD [20, 24].

According to the literature, more and more evidence is accumulated, indicating the importance of inflammatory processes in the vascular wall as a factor in the development and destabilization of atherosclerotic process and the associated earlier, more frequent development of CVD and their various complications. In atherosclerotic process the main marker of inflammation are cytokines, their imbalance is manifested by the increase of proinflammatory interleukins (tumour necrosis factor (TNF- $\alpha$ ), interleukin-1 $\beta$  (IL-1 $\beta$ ), IL-6) and decrease of anti-inflammatory interleukins (IL-4, IL-8 and IL-10). In particular, hyperproduction of proinflammatory cytokines IL-1 $\beta$ , IL-6, TNF- $\alpha$ , contribute to the early progression of CHD and lead to the development of acute cardiovascular complications (ACV).

Studies taking into account such factors as cytokine imbalance in the development of CHD in COPD have not been previously studied [26, 27].

Thus, unfavourable environmental, demographic processes, close anatomical and functional connection of the heart and lungs lead to a frequent combination of COPD and CHD, which may be the cause of earlier development of acute coronary syndrome, acute myocardial infarction and sudden cardiac death.

**Objective:** To determine the relationship between cytokine levels (IL-1 $\beta$ , IL-6; IL-10, TNF- $\alpha$ ) and the clinical course of CHD with COPD.

**The material and methods:** The present study is based on the follow-up of the patients with CHD hospitalized in the period from 2021 to 2022 in the intensive care and emergency departments #1, #2 of Samarkand Branch of Republican Scientific Center of Emergency Medical Care (SF RSC EMC).

The 88 patients with CHD and COPD were examined. They were divided into 2 groups depending on sex. Group 1 included 46 (54,8%) male patients with CHD and COPD, mean age was 58,8 $\pm$ 5,29 years. The second comparison group consisted of 42 (45.2%) female patients with CHD and COPD, mean age of which was 55.9 $\pm$ 4.22 years (Fig. 3.1). The control group consisted of 50 virtually healthy volunteers.

All patients underwent history taking, anthropometry, general clinical examination, assessment of clinical and hemodynamic data, standard general clinical and biochemical tests (interleukins). Besides standard electrocardiography (ECG) in 12 leads, all patients underwent echocardiography (EchoCG) on Mindray device in M- and B-mode in accordance with the recommendations of American Association of Echocardiography. ESH/ESC (2015) and RCO/WHO (2014) recommendations were used for the diagnosis of CHD. Statistical processing of the results was performed using Statistica 6.1 software.

**Results.** Analysis of the results showed that the pro-inflammatory interleukins IL-6, IL-1 $\beta$ , TNF- $\alpha$  were statistically significantly higher in male

patients with IHD and COPD compared to female patients with IHD and COPD (Figure 1).

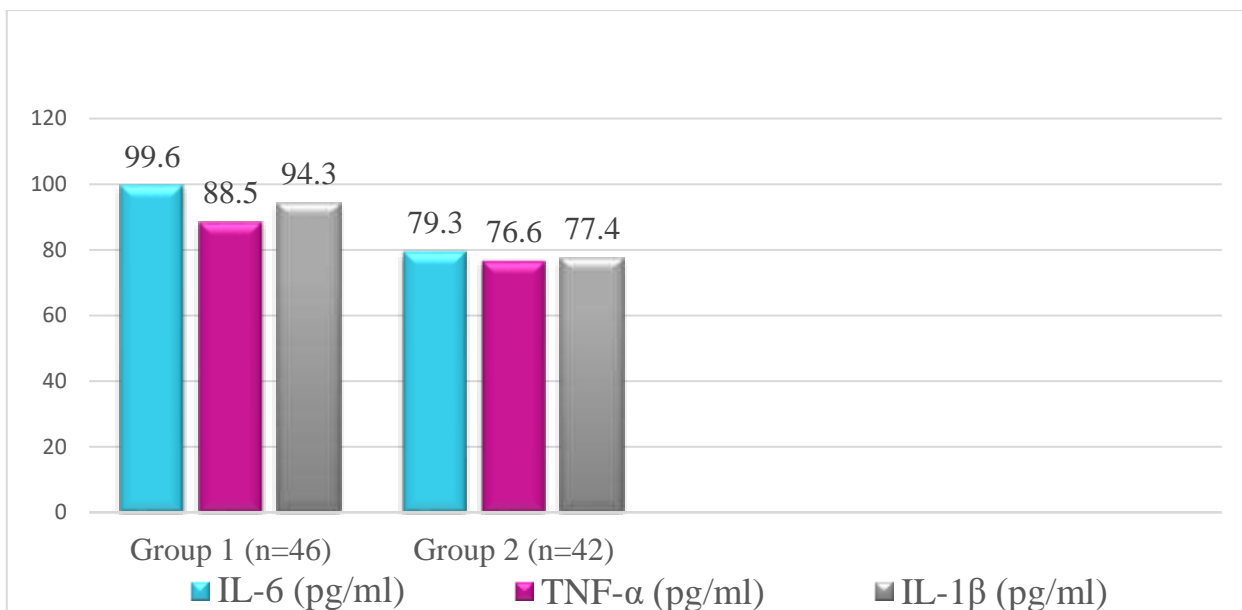


Figure 1. Pro-inflammatory interleukin scores in patients with CHD and COPD as a function of gender

In patients with CHD in polymorbidity with COPD, irrespective of gender, increased levels of IL-6, TNF- $\alpha$  and IL-1 $\beta$  were observed from the first day of the disease and normalisation of pro-inflammatory interleukin levels was noted by day 14 of the disease.

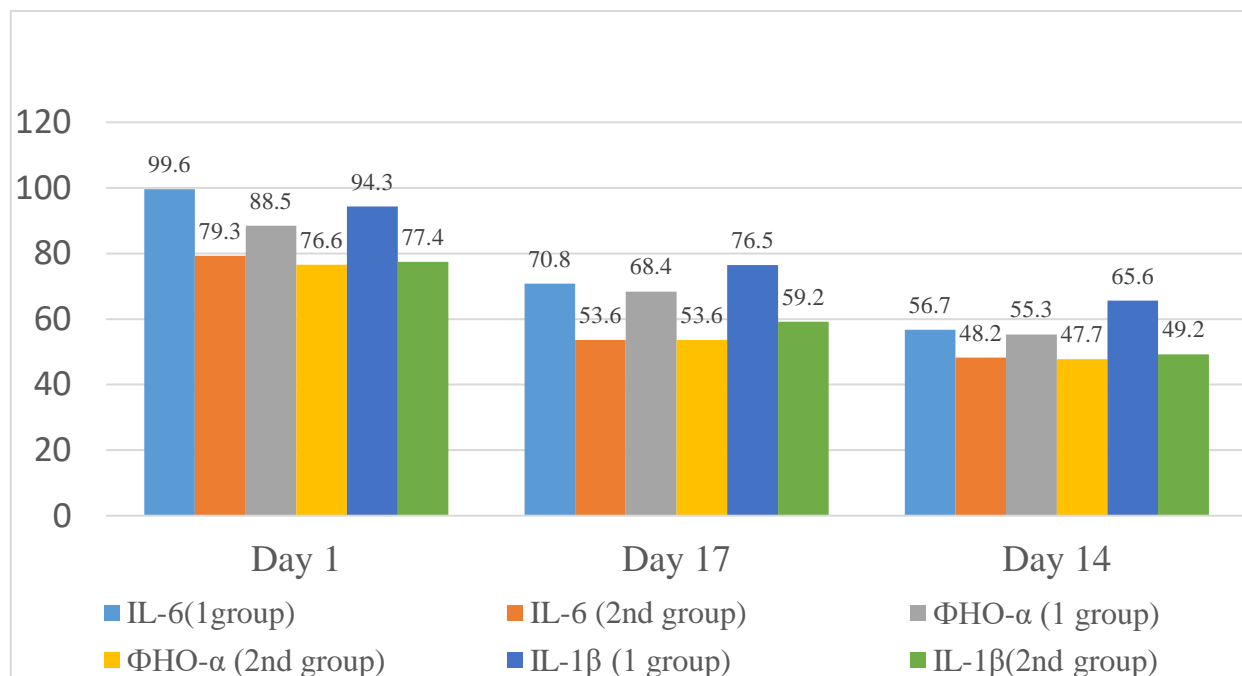
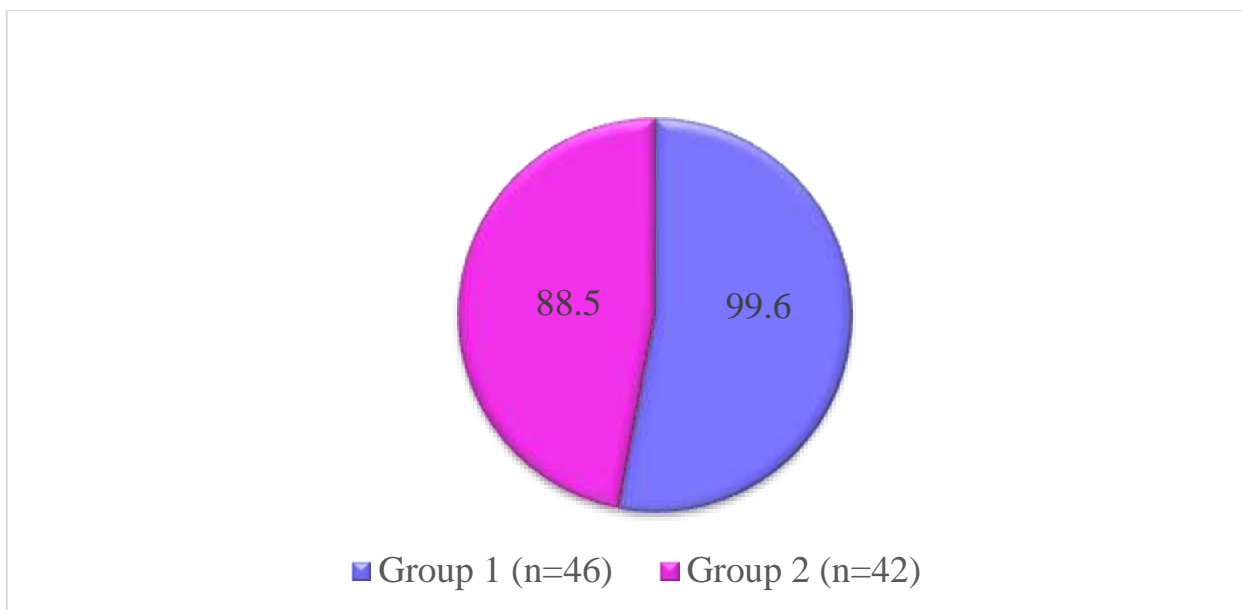
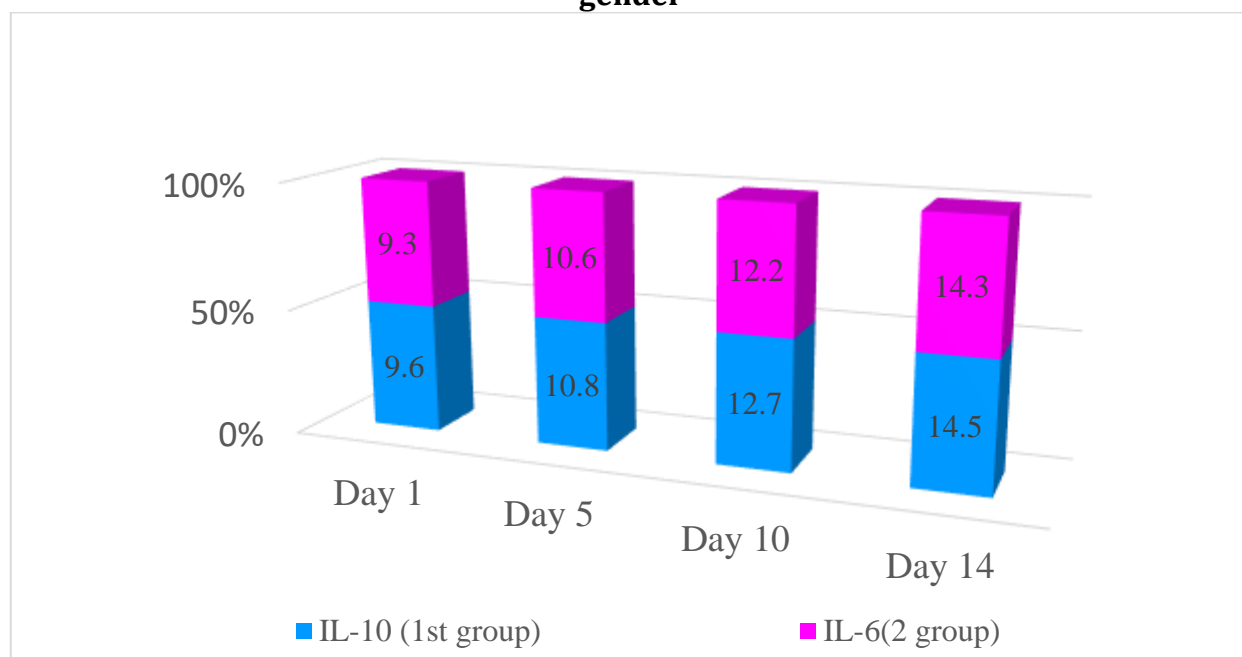


Figure 2. Dynamics of pro-inflammatory interleukin scores in patients with CHD and COPD as a function of sex over 14 days

In the study of anti-inflammatory interleukin levels, the anti-inflammatory interleukin IL-10 was found to be slightly higher in female patients with CHD and COPD compared to male patients with CHD and COPD (Figure 2).



**Figure 1. Anti-inflammatory interleukin scores in patients with CHD and COPD as a function of gender**



***Patients with IBS in polymorbidity with COPD, irrespective of gender, had low levels of IL-10 from the first day of illness, and normalisation of anti-inflammatory interleukin levels was seen on day 14 of the disease.***

**Conclusion.** The study revealed the relationship between proinflammatory interleukin concentrations and the severe course of CHD with COPD, which allows its use as an additional laboratory indicator in these pathologies, and allows the identification of

groups at low and high risk of developing different complications. At the same time, patients with high blood expression of TNF- $\alpha$ , IL-6 and IL-1 $\beta$  are predicted to have a complicated course of the disease. The early identification of patients with CHD and COPD at

high risk of complications will allow more active treatment (medication and/or surgery) of such patients.

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