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SURFACTANT PROTEIN D AS A SIGN OF EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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The purpose of this study is to evaluate and compare the levels of surfactant protein D in the blood serum of patients with chronic obstructive pulmonary disease with a stable course and those without chronic obstructive pulmonary disease. Materials and methods. The present study involved 122 patients with a confirmed diagnosis of chronic obstructive pulmonary disease (the main group) and 20 patients without this disease and other pulmonary or severe somatic diseases (the control group). This investigation was carried out at the Research Institute of Pulmonary Diseases. Spirometry was performed using a portable battery-powered ultrasound spirometer (Easy One; ndd Medical Technologies, Zurich, Switzerland). The SP-D level was determined in venous blood using a "sandwich" variant of solid-phase ELISA using a set of reagents from BioVendor (Czech Republic). Blood sampling and spirometry were performed simultaneously in patients with stable chronic obstructive pulmonary disease and healthy control groups in order to accurately correlate lung function with the level of SP-D in blood serum. Results. The mean age of the main and control groups was 55.9±4.4 and 53.3±2.8 years, respectively. Men made up 59.0% in the main group, women 41.0%, in the control group 55.0% and 45.0%, respectively. Smokers in the main and control groups amounted to 57.4% and 45.0%. FEV1, FVC and FEV1/FVC in patients with chronic obstructive pulmonary disease were lower than controls by 55.72% (p=0.001), 43.23% (p=0.001) and 35.28% (p=0.036), respectively. The level of SP-D in the blood serum of patients with chronic obstructive pulmonary disease and the control group was 372.68±98.16 ng/ml and 164.22±42.80 ng/ml (t=1.95, p=0.053), respectively. SP-D in smokers of the main group was 2.2 times (p=0.011) higher than the control level, and in non-smokers it was 2.5 times (p=0.053). During the exacerbation, the protein level was higher by 19.7% (p=0.042). In CLBL, SP-D correlated with FEV1 (r = −0.815; p<0.001), with FVC (r = −0.822; p<0.001), with FEV1/FVC (r = −0.644; p<0.001). Conclusion. SP-D can be characterized as a specific protein for the lungs, which can be analyzed in the blood as a biomarker for early diagnosis of acute exacerbations of chronic obstructive pulmonary disease and may reflect the severity of the disease.

Key words: chronic obstructive pulmonary disease, surfactant protein D, exacerbation, spirometry, correlation.

Introduction

Chronic obstructive pulmonary disease (COPD) is a growing global health problem and is the third leading cause of death worldwide [1]. In Azerbaijan, the prevalence of "previously diagnosed" COPD was 4.3% per 1000, and the prevalence of COPD "diagnosed by spirometry" was 37.5% per 1000 [2].

The main pathophysiological mechanism of COPD is irreversible airway obstruction with a progressive decrease in lung function, especially in patients with constant exposure to risk factors such as cigarette smoke, exposure to biomass smoke and air pollution, although in some patients lung dysfunction occurs more mildly [3]. Therefore, it is important to stop the decline in lung function in patients who do not meet the diagnostic criteria for COPD, but are at risk of progression (to COPD) to overt COPD [4-7]. In addition, early diagnosis of preclinical COPD may be crucial in patients with fixed obstruction according to spirometry without any symptoms or with only moderate symptoms before they develop a clinically significant deterioration, such as decreased lung function, worsening of symptoms or acute exacerbation [8, 9].

Most COPD patients are detected when they seek medical help with persistent symptoms (cough and/or shortness of breath) or frequent respiratory infections. In some cases, in patients with reduced lung function, the disease may be asymptomatic or patients may ignore their symptoms until the disease worsens and they are hospitalized [10]. Since spirometry is not recommended during an exacerbation, because it is difficult to perform, and measurements are not accurate enough, it is difficult to assess the condition of these patients.

The most widely used marker of COPD severity and progression is the volume of forced exhalation per second (FEV1), but it does not reflect the activity of the underlying disease [11]. It is reported that FEV1 correlates poorly with symptoms and deterioration of health, does not distinguish between the causes of air flow obstruction, i.e., small respiratory tract disease or emphysema [11].

A number of studies have described an association between SPD and COPD [12, 13]. It is believed that surfactant protein D (SP-D) plays an important role in the pathogenesis of COPD, which protects the lungs from oxidative and inflammatory stress, affecting efferocytosis and removing pathogenic microbes as part of the patient's innate immune system [14]. Surfactant protein B is a large hydrophilic glycoprotein belonging to the collectin family, mainly produced in the lungs by type II alveolar cells and non-fibrous cells Clara x12]. SPD contributes to the resolution of lung inflammation, is detected in serum and has the advantage of remaining stable for 6 months [12]. It was found that SPD was associated with a progressive decrease in lung function, and higher levels of SP-D in blood serum were found in severe cases of COPD with deterioration of health [15]. Exacerbations, i.e. worsening of the patient's respiratory symptoms, are a com-

mon cause of hospitalization and have been confirmed as an important cause of morbidity and high mortality associated with COPD. The diagnosis of exacerbations depends on the clinical picture of the patient complaining of a sharp change in symptoms. There are not enough published studies in the literature on the frequency of COPD exacerbations among the Azerbaijani population.

The purpose of this study was to evaluate and compare the levels of surfactant protein D in the blood serum of patients with COPD with a stable course and those examined without COPD.

Materials and methods

The present study involved 122 patients with a confirmed diagnosis of COPD (the main group) and 20 patients without this disease and other pulmonary or severe somatic diseases (the control group) on the basis of the Research Institute of Pulmonary Diseases. The research was conducted in accordance with the ethical principles of the Helsinki Declaration of the World Medical Association. All the examined patients were acquainted with the purpose of the study and expressed the patient's voluntary consent.

The criteria for inclusion of patients in the study were: patients of both sexes; patients registered with the previously specified ICD-10-AM codes; patients with cough, shortness of breath, sputum; age from 40 to 60 years; absence of severe somatic diseases; who agreed to participate in the study. Exclusion criteria from the study: age under 40 and over 60 years; patients with acute and chronic inflammatory diseases; severe pulmonary diseases: tuberculosis, alveolitis, cancer; severe somatic diseases. The selection of patients in the polyclinic was carried out during spontaneous routine or scheduled visits, when their visit to the doctor was not related to the study (with or without respiratory symptoms). Study participants completed COPD questionnaires (Chronic Airways Diseases, A Guide for Primary Care Physicians, 2005; COPD Assessment Test) and underwent a full examination, after which they were diagnosed with COPD. Spirometry was performed using a portable battery-powered ultrasound spirometer (Easy One; ndd Medical Technologies, Zurich, Switzerland). The level of SP-D was determined in venous blood using a "sandwich" variant of solid-phase ELISA using a set of reagents from BioVendor (Czech Republic). Blood sampling and spirometry were performed simultaneously in patients with stable COPD and healthy control groups in order to accurately correlate lung function with the level of SP-D in blood serum.

Statistical data analysis was carried out using the program "Statistica for Windows" v. 16.0 (USA). The average values (M) and standard deviation (SD) were used to describe the character of the distribution of features. For a comparative analysis of numerical data with their normal distribution, the Student's t-test was used for 2 independent samples. To compare the frequencies of a binary trait, the criterion $\chi 2$ was used. The relationship between the various indicators was determined by correlation analysis with the calculation of the Pearson correlation coefficient (r). $P<0.05$ was taken as the level of statistical significance.

Results and discussion

The analysis of demographic indicators between the patients of the main and control groups did not reveal significant differences (Table 1).

Table 1

Initial data of the study groups

Indicator	Main group (n=122)	Control group (n=20)		P=
Average age, years	55.9±4.4	53.3±2.8	t=0.50	0.619
Gender: Men, n (%) Women, n (%)	 72 (59.0) 50 (41.0)	 11 (55.0%) 9 (45.0)	χ^2=0.114	0.736
Smoking: Smokers, n (%) ex-smokers, n (%) non-smokers, n (%)	 70 (57.4) 10 (42.6) 42 (34.4)	 9 (45.0) 1 (5.0) 10 (50.0)	χ^2=1.066	0.302

Note: *t = Student's criterion*

The age of the examined varied from 40 to 60 years. The maximum number of patients with COPD who were under our supervision were aged from 56 to 60 years, which compared with the age group of 40-45 years was 41.7% more common ($p<0.05$), compared with the age group of 46-49 years by 16.6% ($p>0.05$), compared with the age group the group of 50-55 years by 2.7% ($p>0.05$). Concomitant diseases occurred in 73.8% of cases (n=90) in the main group and in 65.0% of cases (n=13) in the control group ($\chi 2=0.663$, $p=0.416$).Of the concomitant diseases, there were diseases of the cardiovascular and gastrointestinal systems. In particular, the proportion of arterial hypertension in the main group was 16.4%, in the control group – 30.0%, angina – 9.0% and 5.0%, atrial fibrillation – 7.4% and 5.0%, respectively. Of the diseases of the gastrointestinal tract in the main group, chronic gastritis was more common - 14.7% (control–10.0%) and cholecystitis - 12.3% (control – 15.0%).

At the time of examination, there was no exacerbation in patients with COPD and spirometry was performed in patients (Table 2).

Lung function indicators were significantly lower in patients with COPD than in the control group. Thus, the indicators of FEV1, FVC and FEV1/FVC in patients with COPD were lower than the control values by 55.72% ($p=0.001$), 43.23% ($p=0.001$) and 35.28% ($p=0.036$), respectively.

In 44 (36.1%) patients, stage I COPD was clas-

sified (forced expiratory volume in 1 s (FEV1) is more than 80% of the proper value), in 39 (32.0%) - stage II (FEV1 >50% and <80%), in 8 (6.5%) patients - stage III severity diseases (FEV1 >30% and <50%)

Determination of the concentration of surfactant protein D (SP-D) in the blood serum of patients of the main and control groups showed a significant increase in protein in patients of the main group: the level of SP-D in the serum of patients with COPD and the control group was 372.68±98.16 ng/ml and 164.22±42.80 ng/ml (t=1.95, p=0.053), respectively. Serum levels of SP-D were highest in smokers in the main group (Table 3).

Table 2
Comparison of indicators of pulmonary function in study groups

Indicator	Main group (n=122)	Control group (n=20)	t=	P
FEV1, l/sec	1.78±0.44	4.02±0.50	3.36	0.001
FVC, l/sec	3.48±0.07	6.13±0.53	4.96	0.001
FEV1/ FVC, %	58.12±10.14	89.80±11.02	2.12	0.036

Notes: *FEV1 is the volume of forced exhalation in 1 s; FVC - forced vital capacity of the lungs*

Table 3
The level of surfactant protein D in smoker patients

Patients	n	Main group	n	Control group	T	P
smokers	70	334.22±68.74	9	154.0±10.45	2.59	0.011
non-smokers	42	190.84±56.21	10	76.53±13.11	1.98	0.053

As can be seen from Table 3, the value of SP-D in the smokers of the main group was 2.2 times (p=0.011) higher than the control level, and in non–smoking patients - 2.5 times (p=0.053). At the same time, there was no airway obstruction in smokers.

The average level of SP-D in the group of patients with stage I COPD was 308.14±80.22 ng/ml, in patients with stage II and III COPD - 366.21±77.11 ng/ml and 394.0±45.72 ng/ml, respectively. As can be seen, the concentration of this protein increased in parallel with the stages of COPD.

In the course of the study, 37 (30.3%) patients had episodic exacerbation of COPD. The level of SP-D was determined in these patients at the time of exacerbation, which averaged 397.08±20.15 ng/ml. Comparative analysis showed a significant increase in this protein compared to the level in patients without COPD exacerbation (318.71±32.43 ng/ml, n=85) by 19.7% (t=2.05, p=0.042).

A correlation analysis was carried out between the level of SPD and the functional parameters of the lungs in both study groups. Strong negative correlations were revealed between the level of SP-D and FEV1 in the main group (r=-0.815; p<0.001) and in the control group (r=-0.704; p<0.001), between the level of SP-D and FVC in the main group (r=− 0.822; p <0.001) and in the control group (r=-0.843; p <0.001), as well as between the level of SP-D and FEV1/FVC in the main group (r=-0.644; p <0.001) and in the control group (r=-0.726; p <0.001).

Significantly elevated mean serum levels of SP-D were found in patients with COPD compared to patients without COPD. SP-D is one of the few lung-specific proteins that can be evaluated in peripheral blood and can be a useful marker in tracking the progression of the disease and the health status of patients with COPD. It was found that surfactant protein D is mainly produced by type II pneumocytes and Clara cells in the lungs [16]. SPD is a multimeric glycoprotein that is part of the collagen-containing C-type lectin or collectins [7]. It promotes homeostasis of pulmonary surfactants and plays a crucial role in pulmonary innate immunity [7, 10]. An increase in the level of SP-D in the blood serum is associated with smoking and chronic inflammatory conditions, in particular, COPD [17].

According to the results of this study, the concentration of SP-D in the blood of COPD patients significantly exceeded the concentration in the blood of the examined control group. It should be noted that the exact mechanism of increasing the level of SP-D in blood serum is still unclear. The level of SP-D in blood serum increases in blood circulation in response to lung pathology, which may explain higher levels during episodes of acute exacerbation as a result of increased intrapulmonary inflammation, since previous studies have shown that the severity of COPD exacerbations correlates with inflammation of the respiratory tract [6, 7].

There is another explanation for the increased level of SP-D in the blood serum during episodes of exacerbation, due to its important role in innate immunity. SPD is responsible for protecting the host's lungs from microorganisms, contributing to opsonization, neutralization, agglutination and enhanced phagocytosis or lysis of inhaled pulmonary pathogens. It also modulates lung inflammation, reducing the formation of oxidative radicals and increasing the clearance of apoptotic and necrotic cells [9, 10, 16]. Thus, the pulmonary expression of SP-D may increase to protect the lungs from pathogens and regulate the inflammatory response in the respiratory tract, where high serum levels of SP-D may reflect increased expression in the lungs [17]. However, the expression of SP-D in the lungs during acute exacerbations has not been analyzed, since more comprehensive studies are required to evaluate this theory.

Consequently, the elevated serum levels of SP-

D in COPD patients in this study may be related to the most common theory, which suggests that SP-D, a hydrophilic protein, moves from the lung to the systemic circulation. This process is probably regulated by changes in alveolar-capillary permeability [12, 16].

Our data, together with the results of other studies presented in the literature, indicate that the levels of SP-D in blood serum may reflect the activity of the disease. Since SP-D is produced mainly in the lungs and moves into the systemic circulation when the permeability of the alveolar-capillary barrier is impaired, serum SP-D has been proposed as a potential biomarker of epithelial integrity in COPD and tracking the progression of the disease [12, 16, 17].

We found an inverse correlation between serum SP-D concentrations and lung function (FEV1% of normal; FVC% of normal; FEV/FVC). It is assumed that surfactant proteins diffuse from the alveoli into the bloodstream in such a way that it reflects oxygen saturation of the blood and lung damage [3]. Our results are consistent with the data of S. Winkler et al. [[12], who reported that serum SPD negatively correlates with FEV1% and FEV1/FVC in patients and healthy smokers,

According to the results obtained by us, the concentration of SP-D in smokers with COPD was statistically significantly higher in comparison with smokers of the main group. Interesting results were listed in a study by M.Y. Lv et al. [17]. The authors showed that the cigarette was not the only factor contributing to the increased occurrence of SP-D in COPD. According to WHO statistics, the incidence of COPD in smokers can reach more than 50%, and cigarettes are one of the recognized etiological factors [1]. In a study by M.Y. Lv et al. [17] SP-D expression decreased in the lung tissue of a mouse model of COPD caused by a cigarette, but there was no significant difference in smoking rates in clinical statistics, which suggests that cigarettes were not the only factor affecting SP-D. Cigarettes, being an important cause of COPD, can directly destroy the structure of SPs by themselves, and can also enhance the destruction of alveolar epithelial cells, causing the initiation of an inflammatory reaction and oxidative stress, increasing the likelihood of recurrence of respiratory tract infection and affecting the expression of SPs, thereby contributing to the progression of COPD.

Conclusion

Based on the results obtained, SP-D can be characterized as a specific protein for the lungs, which can be analyzed in the blood as a candidate biomarker for early diagnosis of acute exacerbations of COPD and may reflect the severity of the disease.

Prospects for further research consist in the development of new methods for the diagnosis and treatment of COPD.

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Реферат

СУРФАКТАНТНИЙ БІЛОК D ЯК ОЗНАКА ЗАГОСТРЕННЯ ХРОНІЧНОЇ ОБСТРУКТИВНОЇ ХВОРОБИ ЛЕГЕНЬ

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Ключові слова: хронічна обструктивна хвороба легень, сурфактантний білок D, загострення, спірометрія, кореляція.

Мета дослідження - оцінка та порівняння рівнів сурфактантного білка D у сироватці крові у пацієнтів з хронічним обструктивним захворюванням легень зі стабільним перебігом та обстежених без цього захворювання. Матеріал та методи дослідження. Обстежено 122 пацієнти з хронічним обструктивним захворюванням легень (основна група) та 20 пацієнтів без цього захворювання та інших легеневих та тяжких соматичних захворювань (контрольна група). Спірометрію проводили за допомогою портатив-

ного ультразвукового спірометра з батарейним живленням. Рівень SP-D визначали в сироватковій крові методом ІФА тест-набором фірми «BioVendor». Результати. Середній вік основної та контрольної групи становив 55,9±4,4 та 53,3±2,8 років відповідно. Чоловіки становили в основній групі 59,0%, жінки 41,0%; у контрольній групі 55,0% та 45,0% відповідно. Курці в основній та контрольній групах склали 57,4% та 45,0%. Показники ОФВ1, ФЖЕЛ та ОФВ1/ФЖЕЛ у пацієнтів з хронічним обструктивним захворюванням легень були нижчими за контрольні на 55,72% (p=0,001), 43,23% (p=0,001) та на 35,28% (p=0,036) відповідно. Рівень SP-D у сироватці крові у пацієнтів з хронічним обструктивним захворюванням легень та групи контролю становив 372.68±98.16 нг/мл та 164,22±42,80 нг/мл (t=1,95, p=0,053) відповідно. SP-D у курців основної групи у 2,2 рази (p=0.011) перевищував контрольний рівень, а у пацієнтів, що не палять, – у 2,5 рази (p=0,053). При загостренні хронічного обструктивного захворювання легень рівень білка був вище на 19,7% (p=0,042). При хронічному обструктивному захворюванні легень SP-D корелював з ОФВ1 (r = −0,815; p <0,001), з ФЖЕЛ (r = − 0,822; p <0,001), з ОФВ1/ФЖЕЛ (r = −0,644; p <0,001). Висновок. В патогенезі хронічного обструктивного захворювання легень не виключена роль сурфактантного білка D (SP-D). SP-D може характеризуватись як специфічний білок легень як біомаркер-кандидат для ранньої діагностики гострих загострень хронічного обструктивного захворювання легень, і може відображати тяжкість захворювання.

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АСОЦІАЦІЇ СТРУКТУРНО-ФУНКЦІОНАЛЬНОГО СТАНУ МІОКАРДА ТА РІВНІВ СИРОВАТКОВОГО NT-PROBNP ТА ST2 У ПАЦІЄНТІВ З ПОЄДНАНОЮ КАРДІАЛЬНОЮ ПАТОЛОГІЄЮ

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Ураження серця при гіпертонічній хворобі представляє собою сукупність змін лівого шлуночка, лівого передсердя та коронарних артерій в результаті хронічного підвищення артеріального тиску. Артеріальна гіпертензія збільшує навантаження на серце, викликаючи структурно-функціональні зміни в міокарді. Безперечний практичний інтерес викликають асоціації клініко-лабораторних та інструментальних показників із рівнями різних біомаркерів, які характеризують специфічність та тяжкість системних процесів, що відбуваються в серцево-судинній системі і також можуть бути використані для передбачення прогнозу захворювань. Метою дослідження було вивчити асоціації структурно-функціонального стану міокарда та рівнів сироваткових NT-proBNP та ST2 у пацієнтів з гіпертонічною хворобою з/без хронічної коронарної хвороби. Матеріали і методи дослідження. У дослідження було включено 118 пацієнтів з гіпертонічною хворобою II стадії з/без хронічної коронарної хвороби (ХКХ). Усім пацієнтам додатково проводили визначення основних показників структурно-функціонального стану міокарда за даними ехокардіографії, а також визначали вміст біомаркерів NT-proBNP та ST2 у плазмі крові на 2-3 день перебування в стаціонарі на тлі підбору оптимальної терапії. Результати дослідження та їх обговорення. Отримані дані свідчили, що відносно низький рівень NT-proBNP асоціювався з суттєвим збільшенням величини правого передсердя та індексу правого передсердя і частоти випадків з концентричною гіпертрофією лівого шлуночка порівняно з проміжним і відносно високим рівнями нейрогормону. У пацієнтів з відносно низьким рівнем нейрогормону спостерігали достовірне збільшення величини кінцевого діастолічного розміру та лівого передсердя порівняно лише з проміжним рівнем. Результати аналізу змін показників ехокардіографії залежно від рівня ST2 у плазмі продемонстрували повну відсутність будь-яких достовірних змін між виділеними групами.

Ключові слова: гіпертонічна хвороба, NT-proBNP, ST2, структурно-функціональний стан міокарда, ехокардіографія.

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Вступ

Гіпертонічна хвороба (ГХ), як хронічне захворювання, є не лише причиною втрати здоров'я, але й основним та незалежним фактором ризику розвитку іншої серцево-судинної патології, включаючи ішемічну хворобу серця, серцеву недостатність та інсульт [15]. Менше половини пацієнтів з гіпертонією знають про свій стан, а багато інших знають, але не лікуються або лікуються неадекватно, хоча успішне лікування гіпертонії зменшує глобальний тягар захворювань і смертності [13].

Ураження серця при гіпертонічній хворобі представляє собою сукупність змін лівого шлуночка, лівого передсердя та коронарних артерій в результаті хронічного підвищення артеріального тиску [12]. Артеріальна гіпертензія збільшує навантаження на серце, викликаючи структурно-функціональні зміни в міокарді [12, 14]. Ці зміни включають гіпертрофію лівого шлуночка, яка може прогресувати аж до розвитку серцевої не-