Реферати

НОВІ НЕІНВАЗИВНІ МАРКЕРИ ТЯЖКОСТІ ПЕРЕБІГУ ІДІОПАТИЧНОГО ФІБРОЗУ ЛЕГЕНЬ

Родіонова В.В., Карасьова О.В., Бех О.Е., Ткаченко В.А., Гордієнко Ю.А.

Ідіопатичний фіброз легень є важким, неухильно прогресуючим захворюванням, з-за недостатньої специфічності ознак та мінливого характеру перебігу якого важливим є пошук додаткових неінвазивних маркерів для встановлення діагнозу, визначення ступеня тяжкості та моніторингу ефективності лікування. Метою дослідження було визначення активності желатиназ А та В, а також комплексу прожелатиназа В/ліпокалін у хворих з помірним та тяжким перебігом ідіопатичного фіброзу легень. Встановлено, що підвищення активності желатиназ пов'язано з прогресуванням захворювання. Збільшення рівня активності прожелатинази В та її активної форми на різних етапах захворювання може слугувати покажчиком ступеня розвитку фіброзного процесу, тоді як активність желатинази А – його стадії. Зміни активності комплексу желатиназа В/ліпокалін відбивають клінічні особливості перебігу ідіопатичного фіброзу легень та пов'язані з тяжкістю захворювання.

Ключові слова: ідіопатичний фіброз легень, желатинази А та В, комплекс прожелатиназа В/ліпокалін.

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НОВЫЕ НЕИНВАЗИВНЫЕ МАРКЕРЫ ТЯЖЕСТИ ТЕЧЕНИЯ ИДИОПАТИЧЕСКОГО ФИБРОЗА ЛЕГКИХ

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Идиопатический фиброз легких является тяжелым, неуклонно прогрессирующим заболеванием, из-за недостаточной специфичности признаков и изменчивого характера течения которого важным является поиск дополнительных неинвазивных маркеров для постановки диагноза, определения степени тяжести и мониторинга эффективности лечения. Целью работы было определение активности желатиназ А и В, а также комплекса прожелатиназа В/липокалин у больных с умеренным и тяжелым течением идиопатического фиброза легких. Установлено, что повышение активности желатиназ связано с прогрессированием заболевания. Увеличение уровня активности прожелатиназы В и ее активной формы на разных этапах заболевания может служить маркером степени развития фиброзного процесса, тогда как активность желатиназы А – его стадии. Изменения активности комплекса прожелатиназа В/липокалин отображают клинические особенности течения идиопатического фиброза легких и связаны с тяжестью заболевания.

Ключевые слова: идиопатический фиброз легких, желатиназы А и В, комплекс прожелатиназа В/липокалин.

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PECULIARITIES OF THE IMMUNE STATUS IN INDUSTRIAL WORKERS WITH PNEUMOCONIOSIS IN COMBINATION WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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This article presents the results of the study on the status of humoral link in general immunity and functional activity of immune cells in workers of the mining and metallurgical industries with pneumoconiosis in combination with chronic obstructive pulmonary disease. It was found that in this category of patients humoral immunity indices were characterized by a significant increase in IgM (up to 4.5 g/l) and IgE (up to 465.6 IU/ml) compared with the control group, patients with pneumoconiosis and occupational chronic obstructive pulmonary disease. This indicated the formation of a pronounced "immune response" with the transformation of B-lymphocytes into plasma cells and stimulation of IgG secretion, promoting the prolongation of bronchopulmonary inflammation. Increased serum IgA (up to 2.8 g/l) is evidence of the simultaneous formation of "protective processes" in the respiratory tract. Reduction of spontaneous (up to 109.55 OU) and induced (up to 246.45 OU) activity of circulating immune complexes, as well as proliferative activity of lymphocytes (up to 1.29 OU) in the reaction of blast transformation of lymphocytes with mitogen coenzyme A increases the probability of recurrent disease. Increasing the content of complement (C3 component) to 1.24 g/l stimulates the production of histamine from mast cells and platelets that support phagocytosis, increase the permeability of vessel walls, spasm of smooth muscles, antigen-antibody reaction with the subsequent development of autoimmune processes in this category of patients.

Key words: pneumoconiosis, chronic obstructive pulmonary disease, workers, immune status.

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Lung diseases of occupational causation occupy a leading place in the general structure of occupational diseases and are one of the most important causes of temporary or permanent disability. There is a steady trend of increasing cases of these diseases for the first identified and the number of people who were recognized as disabled as a result [10].

In the mining and metallurgical industry, the impact on the organism of workers of industrial contaminants is primarily the cause of pneumoconiosis (PC) and chronic obstructive pulmonary disease (COPD) [7, 10]. COPD of occupational causation is a disease that occurs due to long-term exposure to

industrial contaminants and is characterized by progressive bronchial obstruction due to diffuse lesions of the bronchial mucosa, remodulation of their wall and the formation of pulmonary insufficiency [1]. Considering PC as a primary, mostly interstitial, pulmonary fibrosis, this disease is assessed as a pathological condition based on inflammation of the lung parenchyma [1, 7]. Undoubtedly, changes in general immunity determine the main pathogenetic links of PC and COPD of occupational causation, affect the features of their clinical manifestations and course [3, 4, 6].

The combination of PC and COPD in industrial workers is a problem to solve which the assessment of the state of general immunity is considered important in terms of studying the pathogenesis of these diseases [1]. Determining the state of humoral immunity and functional activity of immune cells is the basis for justification and implementation of measures for timely diagnosis, treatment and prevention of PC in combination with COPD.

The purpose of the study was to consider the indices of humoral immunity and functional activity of immune cells in mining and metallurgical workers, patients with PC in combination with COPD, as criteria for diagnosing the disease and developing measures to treat and prevent it.

Materials and methods. 118 employees of the mining and metallurgical industry with occupational lung diseases were examined. Of these, 33 patients with PC in combination with COPD, 16 patients with PC and 69 patients with COPD of occupational causation. The control group (CG) included 10 healthy workers.

The following indicators were determined by immunoturbidimetry: content of serum immunoglobulins A, M, G (IgA, IgM, IgG), in g/l, by immunochemical with electro-chemiluminescence immunoassay (ECLIA) – content of total immunoglobulin E (IgE), in IU/ml (Cobas 6000; Roche Diagnostics, Switzerland), by the method of flow cytometry using monoclonal antibodies – the number of B-lymphocytes ((CD_3^-, CD_{19}^+), in% (flow cytometer instrument). The number and functional activity of immune cells were assessed by enzyme-linked immunosorbent assay (ELISA) (EUROIMMUM, Germany) and immunoturbidimetry (Cobas 6000, Roche Diagnostics, Switzerland). The content of circulating immune complexes (CICs) was determined: large, medium, small, in optical units, spontaneous and induced (stimulated) variant of their activity, in optical units,calculated the phagocytic index (PI) defined the C3 complement component and C4-2 complement component in g/l, the proliferative activity of lymphocytes in the leukocytes blast-transformation reaction (RBLT) with the mitogen of coenzyme A, in optical units, were also determined.

All employees provided written consent to conduct the study in accordance with the ethical principles of the Declaration of Helsinki Human Participation as an Object of Research and Awareness, with the permission of the Commission on Bioethics of the State Institution "UKRNDIPROMMED" (Protocol No. 93 of 30.04.2015).

Material processing was performed using a standard Microsoft Office Excel software package. The obtained data had a normal probability distribution and for their analysis were used mainly parametric criteria of Student and Fisher. The number of observations was sufficient to obtain unbiased estimates of the first two points: the arithmetic mean (M) and the standard deviation (m). Student's t-test was used to compare the meal values of quantitative indices under the condition of normal distribution. The significance level of $p<0.05$ with a reliability of 95% was considered reliable.

Results of the study and their discussion. It was found that the content of IgA, compared with the control group, (table 1) was higher by 27.2% in patients with PC in combination with COPD ($p<0.05$) and PC, in patients with COPD of occupational causation – by 13.6%. IgM content was the highest in patients with PC in combination with COPD by 4.5 times, in patients with PC and COPD, respectively – by 77.7% ($p<0.002$) and – by 22.2%. The IgG content in CG patients was identical to that of patients with PC in combination with COPD, higher by 12.9% than in patients with PC and by 17.0% lower than in patients with COPD. The IgE content, in comparison with CG, was the highest in patients with PC in combination with COPD by 5.47 times ($p<0.05$), in patients with PC and COPD, respectively by 34.4% and in 2.43 times ($p<0.01$). The amount of CD_3^-, CD_{19}^+ in CG exceeded the same indicator in patients with occupational lung pathology: in patients with PC in combination with COPD – by 28.7%, in patients with PC – by 68.8% ($p<0.002$) and in patients with COPD – by 30.0% ($p<0.05$).

When compared with patients with PC in combination with COPD, the IgA content was lower in patients with COPD by 12.0% and identical to patients with PC. The IgM content was lower in patients with PC – by 2.56 times, and in patients with COPD – by 3.7 times. The IgG content, on the contrary, was higher by 10.0% ($p<0.05$) in patients with PC and lower in patients with COPD of occupational causation – by 19.8% ($p<0.001$). The IgE content, in comparison with patients with PC in combination with COPD, was lower in patients with PC – by 4.06 times, and in patients with COPD – by 2.24 times. The relative amount of CD_3^-, CD_{19}^+ in patients with PC in combination with COPD was the same as in patients with COPD and was by 31.1% higher than in patients with PC.

Table 1

Indicators of humoral immunity in workers of the mining and metallurgical industry with occupational lung diseases (M±m)

Indices	Control group (n=10)	Patients with COPD of occupational causation		
		Pneumoconiosis in combination with COPD (n=33)	Pneumoconiosis (n=16)	COPD (n=69)
1	2	3	4	5
Immunoglobulin A (g/l)	2.2±0.2	2.8±0.2$^{\#}$	2.8±0.4	2.5±0.1
Immunoglobulin M (g/l)	0.9±0.1	4.1±2.0	1.6±0.2$^{\#}$	1.1±0.1
Immunoglobulin G (g/l)	12.4±0.8	12.7±0.5	14.0±0.4*	10.6±0.3$^{\#/*/**}$
Immunoglobulin E (IU/ml)	85.1±32.1	465.6±195.0$^{\#}$	114.4±40.1	207.6±31.9$^{\#}$
B-lymphocytes (CD_3^-, CD_{19}^+) (%)	13.0±1.4	10.1±1.0	7.7±1.0$^{\#}$	10.0±0.4$^{\#/**}$

Note: # – The difference is significant compared to the control group (p<0.05); * – The difference is significant in comparison with the group of patients with pneumoconiosis in combination with COPD (p <0.05); ** – The difference is significant in comparison with the group of patients with pneumoconiosis (p <0.05).

Comparison of humoral immunity with patients with PC revealed that the IgA content in patients with COPD was lower by 12.0%, the content of IgM and IgG also, respectively – by 45.4% and – by 32.0% (p<0.001). The content of IgE, on the contrary, was lower in patients with PC than in patients with COPD – by 814%. The relative amount of CD_3^-, CD_{19}^+ in patients with PC was lower than in patients with COPD of occupational causation – by 29.8% (p<0.05).

Table 2.

Indices of functional activity of the cellular component of immunity in workers of the mining and metallurgical industry with occupational lung diseases (M±m)

Indices	Control group (n=10)	Patients with COPD of occupational causation		
		Pneumoconiosis in combination with COPD (n=33)	Pneumoconiosis (n=16)	COPD (n=69)
1	2	4	5	6
Functional activity of immune cells/CICs:				
– spontaneous (OU)	112.88±1.46	109.55±4.05	113.28±2.16	108.94±1.49
– induced (OU)	268.66±6.82	246.45±8.87$^{\#}$	251.46±8.67	253.33±5.90
Phagocytic index	2.28±0.08	2.2±0.04	2.2±0.07	2.3±0.03*
Proliferative activity of lymphocytes (RBLT) with the mitogen of coenzyme A (OU)	1.33±0.03	1.29±0.02	1.39±0.04	1.35±0.02*
Circulating immune complexes:				
– large (OU)	10.02±2.7	9.46±1.06	8.66±1.56	7.23±0.41*
– medium (OU)	73.41±8.50	89.06±1.77	89.57±2.57	82.72±1.25$^{*/**}$
– small (OU)	174.21±2.52	177.46±1.36	177.60±2.28	177.10±0.99
Complement (C3 component) (g/l)	1.16±0.07	1.24±0.04	1.15±0.04	1.21±0.02
Complement (C4-2 component) (g/l)	0.22±0.02	0.26±0.01	0.23±0.01*	0.27±0.01$^{*/**}$

Note: $^{\#}$ – The difference is significant compared to the control group (p<0.05); * – The difference is significant in comparison with the group of patients with pneumoconiosis in combination with COPD (p<0.05); ** – The difference is significant in comparison with the group of patients with pneumoconiosis (p<0.05)

The data in table 2 on the functional activity of some indicators of the cellular component of the general immunity indicate that in comparison with CG, spontaneous activity of immune cells in patients with PC in combination with COPD and COPD of occupational causation was lower, respectively – by 3.0% and by 3.5%. In patients with PC it was higher by 0.3%. The induced activity of immune cells was lower in all groups of sick workers: in patients with PC in combination with COPD – by 9.0% (p<0.05), in patients with PC – by 6.8% and in patients with COPD – by 6.0%. PI, in comparison with CG, did not differ significantly in patients with occupational lung pathology. The index of proliferative activity of lymphocytes in RBLT was lower in patients with PC in combination with COPD by 3.1%. However, in patients with PC and COPD, it exceeded the same indicator in CG by 4.5% and 1.5%, respectively. It was found that the number of CICs, compared to CG, was lower in sick workers: by 5.9% in patients with PC in combination with COPD, by 15.7% in patients with PC and by 38.6% – in patients with COPD. Another trend was found for the mean CICs, the number of which in these groups of patients was higher, respectively by 21.3%, 22.0% and 12.6%. A similar orientation was found for small CICs, the number of which was also higher than in CG, respectively by 1.8% in patients with PC in combination with COPD, by 1.9% – in patients with PC and by 1.6% – in patients with COPD. The content of complement (C3 component), in comparison with CG, in patients with

PC in combination with COPD and COPD of occupational causation was higher, by 6.9% and 4.3%, respectively. In patients with PC, this index was, on the contrary, lower by 0.9%. The complement content (C4-2 component) in sick workers was higher: in patients with PC in combination with COPD – by 18.2%, in patients with PC – by 4.5%, in patients with COPD – by 22.7% ($p<0.05$).

When compared with patients with PC in combination with COPD, the spontaneous activity of the CICs was higher in patients with PC by 3.4% and, conversely, lowers in patients with COPD – by 0.5%. The induced CICs activity in patients of this group was lower than in patients with PC and COPD, by 2.0% and 2.8%, respectively. PI in these groups did not differ much. The index of proliferative activity of lymphocytes in RBLT in patients with PC in combination with COPD was lower than in patients with PC by 7.7% ($p<0.05$) and by 4.5% than in patients with COPD. The content of large subpopulations of CICs in patients with PC in combination with COPD exceeded similar indices in patients with PC and COPD, by 9.2% and 30.8%, respectively ($p<0.05$). The content of mean CICs subpopulations in these patients did not differ from patients with PC and was 7.7% ($p<0.01$) higher than in patients with COPD. The content of small CICs subpopulations in patients with occupational lung diseases did not differ significantly. Compared with patients with PC in combination with COPD, the complement content (C3 component) in patients with PC was lower by 7.8%, and in patients with COPD of occupational etiology – by 2.5%. The content of the C4-2 complement component in this category of patients exceeded the same indice in patients with PC by 13.9% ($p<0.05$) and, conversely, was lower than in patients with COPD by 3.8%.

In PC patients, spontaneous CICs activity was by 4.0% higher than in COPD patients, however, induced CICs activity was lower by 0.7%. PI did not differ significantly in patients with PC and COPD, the proliferative activity of lymphocytes in RBLT in patients with PC was higher than in patients with COPD of occupational causation by 2.9%. The content of different CICs subpopulations in patients with PC was higher than in patients with COPD: large – by 19.7%, medium – by 8.3% ($p<0.02$), small – by 0.3%. The complement content in patients with PC was lower than in patients with COPD, respectively by 5.1 % – C3 component and 17.4 % ($p<0.002$) – C4-2 component.

Studies have shown that humoral immunity indices of workers in the mining and metallurgical industries with PC in combination with COPD are characterized by a significant increase in IgM: by 4.5 times compared to CG, from 2.6 to 3.7 times in comparison with patients with PC and COPD of occupational causation with a simultaneous increase in IgE – by 5.4 times compared to CG, from 2.2 to 4.1 times in comparison with patients with PC and COPD of occupational causation. These changes indicate the presence of processes for mechanisms formation on the expressed "immune response" with simultaneous transformation of B-lymphocytes into plasma cells and stimulation of IgG secretion [5]. That is, the invented shifts of the humoral link of the general immunity stimulate the prolongation of chronic, mainly infectious, bronchopulmonary inflammation. An increase in IgE in the blood indicates the "trigger" of allergic reactions, which are realized by the tendency to develop bronchial hyperreactivity and subsequent atopy. Increased serum IgA forms "protective processes" in the respiratory tract. There is a tendency to decrease the spontaneous and induced activity of the CICs and the proliferative activity of lymphocytes in RBLT, which is evidence of chronic systemic inflammation and the formation of a tendency to its recurrent course. Increased complement (C3 component) is a predictor that stimulates the production of histamine from mast cells and platelets, supporting phagocytosis, increasing vascular permeability, increasing smooth muscle spasm, leukocyte chemotaxis and antigen-antibody reaction [2]. An increase in the complement content (C4-2 component) is a sign of a significant risk of occurrence and progression of infectious inflammation in sick workers. The obtained data expand the idea of the pathogenetic mechanisms of PC formation in combination with COPD and supplement the results of previous studies on the pathogenesis of dust pathology of the lungs in industrial workers [2, 5, 9].

Conclusions

1. According to the results of the study it was established that at the present stage an increase in IgM and IgE, which is a sign of high activity of chronic inflammation in the bronchopulmonary system with the presence of an allergic component, is a specific feature of the state of the humoral part of the immune system in workers of the mining and metallurgical industry with PC in combination with COPD.

2. In terms of functional activity of immune cells in patients with PC in combination with COPD, the tendency to decrease the spontaneous and induced activity of CICs and proliferative activity of lymphocytes in RBLT is a predictor of chronic systemic inflammation and the formation of predisposition to its recurrent course.

3. Increased complement content (C3 component) stimulates the activation and subsequent hyperproduction of inflammatory mediators, initiation of autoimmune mechanisms that cause prolongation of systemic inflammation with a predisposition to its infectious complications.

Prospects for further research lie in the fact that the obtained data are an important component in further research aimed at developing a scientifically sound system of measures for timely diagnosis, treatment and prevention of PC in combination with COPD in workers of the mining and metallurgical industries.

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Реферати

ОСОБЛИВОСТІ ІМУННОГО СТАТУСУ У ПРОМИСЛОВИХ ПРАЦІВНИКІВ, ХВОРИХ НА ПНЕВМОКОНІОЗ У ПОЄДНАННІ З ХРОНІЧНИМ ОБСТРУКТИВНИМ ЗАХВОРЮВАННЯМ ЛЕГЕНЬ

Рубцов Р.В.

В статті викладено результати досліджень щодо вивчення стану гуморальної ланки загального імунітету та функціональної активності імунних клітин у працівників гірничорудної та металургійної промисловості, хворих на пневмоконіоз у поєднанні з хронічним обструктивним захворюванням легень. Встановлено, що у цієї категорії хворих показники гуморального імунітету характеризуються суттєвим збільшенням IgM (до 4,5 г/л) та IgE (до 465,6 МО/мл), у порівнянні з контрольною групою, хворими на пневмоконіоз та хронічне обструктивне захворювання легень професійної етіології, вказуючи на формування вираженої «імунної відповіді» з трансформацією В-лімфоцитів у плазматичні клітини та стимуляцією секреції IgG, сприяючи пролонгації бронхолегеневого запалення. Збільшений вміст у сироватці крові IgA (до 2,8 г/л) є свідченням одночасного формування «захисних процесів» у дихальних шляхах. Зменшення спонтанної (до 109,55 опт.од.) та індукованої (до 246,45 опт.од.) активності циркулюючих імунних комплексів, а також проліферативної активності лімфоцитів (до 1,29 опт. од.) у реакції бластної трансформації лімфоцитів з мітогеном коензіму А посилює вірогідність рецидивуючого перебігу хвороби. Збільшення вмісту комплементу (С3 компоненту) до 1,24 г/л стимулює продукцію гістаміну з опасистих клітин та тромбоцитів, які підтримують фагоцитоз, посилюють проникність стінок судин, спазм гладкої мускулатури, реакцію антиген-антитіло з подальшим розвитком автоімунних процесів у цієї категорії хворих.

Ключові слова: пневмоконіоз, хронічне обструктивне захворювання легень, працівники, імунний статус.

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ОСОБЕННОСТИ ИММУННОГО СТАТУСА У ПРОМЫШЛЕННЫХ РАБОЧИХ С ПНЕВМОКОНИОЗОМ В СОЧЕТАНИИ С ХРОНИЧЕСКИМ ОБСТРУКТИВНЫМ ЗАБОЛЕВАНИЕМ ЛЕГКИХ

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В статье изложены результаты изучения гуморального звена общего иммунитета и функциональной активности иммунных клеток у рабочих горнорудной и металлургической промышленности с пневмокониозом в сочетании с хроническим обструктивным заболеванием легких. Установлено, что у этой категории больных увеличено содержание IgM (до 4,5 г/л), IgE (до 465,6 МО/мл), в сравнении с контрольной группой, больными пневмокониозом и хроническим обструктивным заболеванием легких профессиональной этиологии, указывая на формирование выраженного «иммунного ответа» с трансформацией В-лимфоцитов в плазматические клетки, стимуляцией секреции IgG, способствуя пролонгации бронхолегочного воспаления. Повышенное содержание в сыворотке крови IgA (до 2,8 г/л) отражает формирование «защитных процессов» в дыхательных путях. Уменьшение спонтанной (до 109,55 опт.ед.) и индуцированной (до 246,45 опт.ед.) активности циркулирующих иммунных комплексов, а также пролиферативной активности лимфоцитов (до 1,29 опт. ед.) в реакции бластной трансформации лимфоцитов с митогеном коэнзима А, усиливает вероятность рецидивирующего течения болезни. Увеличение содержания комплемента (С3 компонента) до 1,24 г/л стимулирует продукцию гистамина тучными клетками и тромбоцитами, вызывая фагоцитоз, усиливая проницаемость стенок сосудов, спазм гладкой мускулатуры, реакцию антиген-антитело с развитием аутоиммунных процессов у этой категории больных.

Ключевые слова: пневмокониоз, хроническое обструктивное заболевание легких, рабочие, иммунный статус.

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