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The state of immune homeostasis in patients with the comorbid course of asthma, obesity and diabetes mellitus type 2

Key words: *asthma, obesity, diabetes mellitus type 2, cellular immunity.*

In recent years the attention of researchers and doctors of different specialities is more and more attracted by the problem of comorbidity, which means a combination of several chronic diseases in one patient [6, 13]. Comorbidity is very common in therapeutic patients. For example, according to M. Fortin's data (2005), the prevalence of comorbidity ranges from 69 % in young (18–44 years) patients to 98 % in older ones (over 65), the number of chronic diseases vary from 2.8 in young cases to 6.4 in elderly ones.

It should be emphasized that the influence of comorbid pathology on clinical manifestations, diagnosis, prognosis and treatment of many diseases is complex and individual. The cooperation of diseases, age and drug pathomorphism significantly changes the course of the main disease, the character and severity of complications, affects the patient's life quality, restricts or aggravates the medical-diagnostic process [15–17].

Asthma is one of the most common diseases. Statistical data demonstrate a steady rise of the incidence rate, disability and mortality from this pathology in the whole world. According to international experts, at least 334 million people suffer from asthma, it making about 5 % of inhabitants of our planet [11]. GINA (2014) recommendations point out the necessity of the personalized approach with regard for individual peculiarities of the asthma course in each particular patient. The revised GINA version (2014) draws a parallel between the success in achieving control over asthma and comorbid states, which can influence difficulties in diagnosis and efficacy of the given therapy [14].

Studies during recent years have shown that diabetes mellitus type 2 (DM2T) on the one hand as well as obstruction of bronchial patency and reduction of the pulmonary function on the other hand can mutually potentiate each other. It has been found out that asthma and DM2T have common pathogenetic mechanisms (systemic inflammation, hypoxia and hereditary predisposition). Results of the research, conducted by O.M. Uriasyev and Yu.A. Panfilov (2008), show a significant part of metabolic syndrome in formation of the character of the clinical course of asthma. The presence of metabolic disorders, first of all obesity, increases bronchial hyperactivity and considerably aggravates the quality of life in patients with asthma [4, 5].

Other studies confirm an increasing rate of obesity that rises to the character of epidemic. Asthma and obesity are now examined in a relationship, since the both forms of pathology belong to «modernization diseases» [2].

The World Health Organization site, which characterizes the problem of obesity, gives the data that since 1980 the number of people with obesity in the whole world has almost doubled. In 2008 more than 1.4 milliard adults at the age of 20 and over were overweight, it making 35 % of the population. According to M. Vortmann's data (2008), 28–44 % of asthma patients have different degrees of obesity [12, 19]. Modern studies of asthma incidence rate in patients with different levels of an increased body mass index (BMI) have revealed a direct dependence of a rising rate of development of asthma upon an increasing BMI [7]. In patients with comorbidity of asthma and obesity the index of asthma control achievement remains rather low [3]. Obesity both increases the risk of asthma development and is the risk factor for persistence and severity of asthma symptoms [18].

But many pathogenetic, diagnostic and therapeutic aspects of this problem have remained unsolved by the present time. A particular emphasis in the pathogenesis

Table 1

Statistical characteristics of the study of immunological parameters in the groups studied

Indexes	(Me[Q25–Q75])								p
	Control group		Asthma		Asthma + DM2T		Asthma + O		
	Median	Lower quartile-top quartile	Median	Lower quartile-top quartile	Median	Lower quartile-top quartile	Median	Lower quartile-top quartile	
WBC, x10^9/l	6,00	6,00–6,00	6,92	6,70–7,45	4,300	4,075–4,800	8,512	7,900–9,100	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
Number of lymphocytes	2,200	2,100–2,300	2,00	1,90–2,100	1,310	0,980–1,383	1,215	0,900–1,300	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
CD3, %	60,00	59,00–61,00	60,00	50,00–65,00	36,00	33,00–39,250	55,561	54,00–59,00	p_{1-2} = 0,476 p_{1-3} = 0,001 p_{1-4} = 0,001
CD22, %	20,00	19,00–21,00	22,00	18,00–27,00	15,00	11,00–18,00	18,491	15,00–22,00	p_{1-2} = 0,272 p_{1-3} = 0,001 p_{1-4} = 0,24
O-cells, %	24,00	23,00–25,00	27,00	24,00–29,00	31,00	30,00–33,00	25,140	23,00–27,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,045
CD16, %	21,00	21,00–22,00	18,00	16,00–19,00	14,00	12,750–16,00	22,991	22,00–24,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
CD4, %	40,00	39,00–41,00	36,00	35,00–37,00	30,00	26,750–34,00	32,921	32,00–34,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
CD8, %	22,00	21,00–23,00	20,00	18,00–21,00	21,00	20,00–23,00	22,342	21,00–23,00	p_{1-2} = 0,001 p_{1-3} = 0,245 p_{1-4} = 0,264
CD4/CD8	1,818	1,773–1,884	1,833	1,667–1,950	1,377	1,215–1,547	1,477	1,417–1,524	p_{1-2} = 0,942 p_{1-3} = 0,001 p_{1-4} = 0,001
% phagocytes neutrophils	60,00	50,00–70,00	60,00	50,00–70,00	36,00	32,00–40,00	46,544	45,00–48,00	p_{1-2} = 0,605 p_{1-3} = 0,001 p_{1-4} = 0,001
NBT-test, %	10,00	9,00–10,500	15,00	14,00–17,00	15,00	14,00–16,00	20,702	18,00–21,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
IgE, ME/ml	60,00	50,00–65,00	140,00	120,00–170,00	60,00	46,750–82,500	238,772	170,00–290,00	p_{1-2} = 0,001 p_{1-3} = 0,611 p_{1-4} = 0,001
IgA, g/l	2,900	2,800–3,00	4,100	3,100–4,900	3,450	3,100–3,725	2,952	2,340–3,560	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,794
IgM, g/l	1,500	1,450–1,600	1,900	1,700–1,900	1,670	1,450–1,800	1,590	1,450–1,760	p_{1-2} = 0,001 p_{1-3} = 0,017 p_{1-4} = 0,137
IgG, g/l	13,00	12,00–14,00	19,00	18,00–20,00	23,00	19,00–25,00	19,939	18,00–22,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001
CIC, %	93,00	92,00–94,00	91,00	89,00–93,00	79,00	77,00–81,00	89,070	87,00–91,00	p_{1-2} = 0,003 p_{1-3} = 0,001 p_{1-4} = 0,001
Lymphocytic antibodies	3,00	2,00–4,00	11,00	10,00–13,00	21,00	19,00–22,00	15,482	14,00–17,00	p_{1-2} = 0,001 p_{1-3} = 0,001 p_{1-4} = 0,001

Note: p_{1-2} – the level of statistical significance between the control group and the patients with asthma; p_{1-3} – the level of statistical significance between the control group and the patients with asthma + DMT2; p_{1-4} – the level of statistical significance between the control group and the patients with asthma + O; Me – median.

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of aggravation of the asthma course in cases of DM2T and/or obesity is laid upon hyperglycaemic and hypoglycaemic hormones. Also interesting is a dysfunction of the system of interleukins in a combination of asthma with endocrine pathology, which manifests itself by an imbalance of Th1/Th2 differentiation of CD4+-cells [1].

The aim of the present research consisted in studying the state of cellular and humoral immunity, factors of nonspecific defence in asthma, DM2T, obesity and their combination, as well as revealing of peculiarities in the regulation of immune response in patients with a comorbid course of this pathology.

Materials and methods

The diagnosis of asthma was made according to the international consensus paper GINA 2014. The diagnosis of DM2T was verified in compliance with international protocols. The total number of examined patients with asthma was 269. Three groups of cases were singled out: group 1 – patients with asthma (asthma, n = 61); group 2 – patients with asthma and DM2T (asthma + DM2T, n = 94); group 3 – patients with asthma and obesity (asthma + O, n = 114). The control group included 21 somatically healthy persons. The patients' age averaged 53.1 ± 2.7 years. The average age in the control group was 48.1 ± 1.4 years.

All patients underwent a complex examination, which included collection of patients' complaints, study of their case histories, physical examination with study of their BMI, additional examination with clinical and biochemical studies, ECG and computed spirometry.

The cellular component of immunity was studied with help of «Anti-CD» erythrocytic diagnosticums. Quantification of subpopulations of T- and B-lymphocytes was performed using plaque reaction with erythrocytes, on which monoclonal antibodies against such receptors as CD3 (T-lymphocytes), CD4 (T-helpers), CD8 (T-suppressors and cytotoxic lymphocytes), CD16 (NK, natural killer cells) and CD22 (B-lymphocytes) were adsorbed. The findings of the study were assessed in an optic microscope with the immersion system.

The study of the humoral component of immunity included quantification of the number of immunoglobulins A, M and G, revealing of the complement activity and the level of circulating immune complexes. The technique of enzyme immunoassay (EIA), described by H. Fricrnel and G. Holzheielt in 1984, was used for quantification of the number of immunoglobulins in blood serum. Immunoenzymatic detection of immunoglobulins was made by the indirect noncompetitive technique of EIA. In order to detect the serum complement the assessment of its activity by 50% haemolysis with Reznikov spectrophotometric method (1967) was used. The content of CIC in blood serum was determined nephelometrically (D.A. Grinevich, A.N. Alferova, 1981).

The complex of immunological studies included revealing of parameters of phagocytic activity of neutrophils (N.V. Vasilyev, Yu.V. Odintsov, 1972), the Nitro Blue Tetrazolium (NBT) test. The method of evaluation of phagocytic activity of neutrophils is based on assessment of the absorptive capacity of blood neutrophils versus the test culture after their joint incubation by using an optical microscope. The NBT test was used for assessing the state of oxygen-dependent mechanisms of bactericidal power of neutrophils.

The findings were statistically treated via SPSS19 (IBM, USA) software tool. Medians and interquartile intervals were computed. The significance of median differences was estimated based on the Mann-Whitney U-test and the Kruskal-Wallis H-test. The relation between the signs was assessed with the Spearman's rank correlation coefficient. The Shapiro-Wilk test was used for analysing the normality of data distribution.

Results and discussion

Our analysis of immune homeostasis indices (Fig. 1) revealed reliable changes in values of both cellular and humoral immunity in all groups of the examined cases. Changes of cellular immunity were more pronounced in the group of examined patients asthma + DM2T, with a particular decrease of CD4% and CD22%. The group of asthma + O demonstrated a marked reduction of CD4%. In both groups the above changes developed against a background of reliably significant lymphopenia. A considerable inhibition of the percentage of phagocytizing neutrophils only in the group of asthma + DM2T confirms development of secondary immunodeficiency in DM2T too. All 3 groups revealed an increased NBT-test, especially manifested in the group of asthma + O, it indirectly pointing to an insufficiency of the phagocytic component. The obtained data showed that obesity can be regarded as the trigger for development of deficiency in the cellular and phagocytic components of immunity, and there are significant signs of cellular immunodeficiency in DM2T caused by this particular pathology, this fact is confirmed by the literature data [10]. The humoral component of immunity in all groups revealed a higher level of IgG, which was most pronounced in the group of patients with asthma + DM2T and there were demonstrated a long-term course of the inflammatory process of the autoimmune character. Having analysed the duration of disease in all groups of patients, we received the following data: on an average, in the group of patients with asthma + DM2T Me = 29.8, Q25–Q75 = 26.7–32.15 years, while in the groups of asthma and asthma + O it was less, respectively, by a factor of 1.8 and 1.5. Our analysis of the level of CIC revealed their increased formation in patients with asthma + DM2T and asthma + O, while in isolated asthma this index virtually was within its normal values. These data confirmed the development of an inflammatory process of the autoimmune character in asthma + DM2T and DM2T + O, while in isolated asthma the above pathological process was under the «control» of therapy. For example, in patients from the group of asthma + DM2T the number of exacerbations averaged 3.6 ± 0.46 times versus 1.3 ± 0.02 times in the group with asthma and 2.87 ± 0.32 times in the group with asthma + O. All examined groups demonstrated an increase of lymphocytotoxic antibodies, more pronounced in patients with

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asthma + DM2T, this fact just pointed to activity of autoimmune processes [8].

Attention is drawn by maximally high values of IgE in patients with asthma + O; this fact demonstrates a high allergization of patients with obesity and is confirmed by data from other researchers [9].

Conclusions

As a result of the conducted clinical-immunological studies it is shown that the comorbid course of asthma with obesity and DM2T is based upon a progressing chronic inflammation of the autoimmune character. Patients with the combined pathology reveal a lower level of cells with T-cell activity and a compensatory increased content of cytotoxic T-lymphocytes, it resulting in an imbalance of the cellular component of the immune system and a lower immunoregulatory index down to Me = 1.38, Q25–Q75 = 1.21–1.54. The revealed signs of secondary immunodeficiency in asthma and DM2T (deficiencies in the cellular and phagocytic components) do not provide the necessary level of pathogenetic immune responses, particularly the production of IgE.

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СТАН ІМУННОГО ГОМЕОСТАЗУ У ХВОРИХ З КОМОРБІДНИМ ПЕРЕБІГОМ БРОНХІАЛЬНОЇ АСТМИ, ОЖИРІННЯМ ТА ЦУКРОВИМ ДІАБЕТОМ 2-ГО ТИПУ

Г.В. Єрьоменко

Резюме

Коморбідність бронхіальної астми (БА), цукрового діабету 2-го типу (ЦД2Т) і ожиріння (О) залишається актуальною проблемою сучасної медицини.

Мета: дослідити стан клітинного, гуморального імунітету, факторів неспецифічного захисту при БА та в поєднанні з ЦД2Т та О.

Матеріали та методи дослідження. Обстежено 269 хворих на БА, виділено 3 групи: перша група – хворі з БА (n = 61), друга група – хворі з БА і ЦД2Т (n = 94), третя група - хворі з БА та О (n = 114).

Стандартні методи дослідження на БА, визначення клітинної ланки імунітету проводили з використанням еритроцитарних діагностикумів «Анти-CD», визначення кількості субпопуляцій Т- і В-лімфоцитів – за допомогою реакції розеткоутворення з еритроцитами. Оцінку результатів дослідження проводили в світловому мікроскопі з імерсійною системою. Дослідження гуморальної ланки імунітету включало визначення кількості імуноглобулінів А, М, G, активності комплементу, рівня циркулюючих імунних комплексів (ЦІК). Для кількісного визначення імуноглобулінів у сироватці крові був використаний метод імуноферментного аналізу (ІФА). Вміст ЦІК в сироватці крові визначали нефелометричним методом. У комплекс імунологічних досліджень входило визначення параметрів фагоцитарної активності нейтрофілів, НСТ-тест.

Результати. Виявлені достовірні зміни як показників клітинного, так і гуморального імунітету у всіх групах досліджених хворих. Зміни клітинного імунітету більш виражені в групі обстежуваних БА + ЦД2Т, особливо відзначено зниження кількості CD4 і CD22. У групі БА + О мало місце виражене зниження кількості CD4. В обох групах зазначені зміни розвивалися на тлі достовірно значущої лімфопенії. Значне зниження відсотка фагоцитуючих нейтрофілів лише в групі хворих на БА + ЦД2Т також підтверджує розвиток вторинного імунодефіциту при ЦД2Т. У всіх 3 групах відзначено підвищення показників НСТ-тесту, найбільш виражене в групі БА + О, що побічно вказує на недостатність фагоцитарної ланки.

Висновки. В результаті проведених клініко-імунологічних досліджень показано, що в основі коморбідного перебігу БА з О та ЦД2Т лежить прогресуюче хронічне запалення автоімунного характеру. У пацієнтів з поєднаною патологією відзначається зниження рівня клітин з Т-хелперною активністю і компенсаторне збільшення вмісту цитотоксичних Т-лімфоцитів, що призводить до дисбалансу клітинної ланки імунної системи і зниження імунорегуляторного індексу. Виявлені ознаки вторинного імунодефіциту при БА і ЦД2Т (дефіцит клітинної і фагоцитарної ланок) не забезпечують належний рівень патогенетичних імунологічних реакцій, зокрема вироблення IgE.

Ключові слова: бронхіальна астма, цукровий діабет 2-го типу, ожиріння, клітинний імунітет.

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СОСТОЯНИЕ ИММУННОГО ГОМЕОСТАЗА У БОЛЬНЫХ С КОМОРБИДНЫМ ТЕЧЕНИЕМ БРОНХИАЛЬНОЙ АСТМЫ, ОЖИРЕНИЯ И САХАРНОГО ДИАБЕТА 2-ГО ТИПА

Г.В. Еременко

Резюме

Коморбидность бронхиальной астмы (БА), сахарного диабета 2-го типа (СД2Т) и ожирения (О) остается актуальной проблемой современной медицины.

Цель: исследовать состояние клеточного, гуморального иммунитета, факторов неспецифической защиты при БА, СД2Т, О и их сочетании.

Объект исследования. Обследовано 269 пациентов с БА, выделено 3 группы: первая группа - больные БА (n=61), вторая группа – больные БА и СД2Т (n=94), третья группа – больные с БА и О (n = 114).

Материалы и методы исследования. Определение клеточного звена иммунитета проводилось с использованием эритроцитарных диагностикумов «Анти-CD». Определение количества субпопуляций Т- и В-лимфоцитов проводилось с помощью реакции розеткообразования с эритроцитами. Оценку результатов исследования проводили в световом микроскопе с иммерсионной системой. Исследование гуморального звена иммунитета включало определение количества иммуноглобулинов А, М, G, активности комплемента, уровня циркулирующих иммунных комплексов (ЦИК). Для количественного определения иммуноглобулинов в сыворотке крови был использован метод иммуноферментного анализа (ИФА). Содержание ЦИК в сыворотке крови определяли нефелометрически. В комплекс иммунологических исследований входило определение параметров фагоцитарной активности нейтрофилов, НСТ-тест.

Результаты. Отмечены достоверные изменения показателей как клеточного, так и гуморального иммунитета у всех групп исследованных больных. Изменения клеточного иммунитета более выражены в группе обследуемых БА + СД2Т, особенно отмечено снижение уровня CD4 и CD22. В группе БА + О имело место выраженное снижение количества CD4. В обеих группах указанные изменения развивались на фоне достоверно значимой лимфопении. Значительное подавление процента фагоцитирующих нейтрофилов лишь в группе больных БА + СД2Т также подтверждает развитие вторичного иммунодефицита при СД2Т. Во всех 3 группах отмечено повышение показателей НСТ-теста, наиболее выраженное в группе БА + О, что косвенно указывает на недостаточность фагоцитарного звена.

Выводы. В результате проведенных клинико-иммунологических исследований показано, что в основе коморбидного течения БА с О и СД2Т лежит прогрессирующее хроническое воспаление аутоиммунного характера. У пациентов с сочетанной патологией отмечается снижение уровня клеток с Т-хелперной активностью и компенсаторное увеличение содержания цитотоксических Т-лимфоцитов, что приводит к дисбалансу клеточного звена иммунной системы и снижению иммунорегуляторного индекса. Выявленные признаки вторичного иммунодефицита при БА и СД2Т (дефицит клеточного и фагоцитарного звеньев) не обеспечивают должный уровень патогенетических иммунологических реакций, в частности выработку IgE.

Ключевые слова: бронхиальная астма, сахарный диабет 2-го типа, ожирение, клеточный иммунитет.

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