



Characteristics Of Immune Status of Children with Bronchiectasis Disease and Chronic Deforming Endo bronchitis

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Annotation. In recent years there has been an active study of immunological mechanisms in the development of BEB and CDEB, the participation of some cellular, humoral and other factors in the progression of the disease has been proved. The problem of BED and ChDED as an immunodeficiency state is widely discussed in the literature.

Keywords: *immunological mechanisms, bronchopulmonary system, non-specific respiratory diseases, T-system*

Immunological status of the patient is characterised by insufficiency of the local immunity system in combination with imbalance of the population of immunocompetent cells in the blood. The insufficiency of the humoral immunity link as a cause of BED and ChDED formation was pointed out that the ratio of local and systemic immunity indicators determines the course of the inflammatory process in the bronchopulmonary system. In this case, a special role in the development and course of nonspecific respiratory diseases is assigned to disorders of local lung defence.

At the same time, despite the progress made in the field of theoretical and clinical pulmonology, many questions concerning the pathogenesis of BED and ChDED remain unresolved. The results of studies conducted in children with BED in the exacerbation phase of the disease have shown that the immune system undergoes certain changes. In connection with the above-mentioned, we conducted a study on the state of the immune system in children with BED (Table 1).

In BED, when there is a prolonged inflammatory process in the lungs, the more expression of changes in the thymus gland, leading to the depletion of adaptive forces of the body. The study of the T-system showed that the number of CD3+ lymphocytes in the peripheral blood of sick children in the groups with BEB and ChDED ranged from 43 to 46%, averaging $44.5\pm2.3\%$, which is significantly lower than the control (p<0.001). Children of the control group have an average of 61.5 ± 2.2 CD3+ lymphocytes in 1 µl of peripheral blood. In sick children this index is 1.9 times lower than the control value

Analysed indicators	Practical	Period of	disease							
	healthy	exacerbation, n=245		D	р	D.				
	children	CDEB n=82	BEB	P	Ρ1	P ₂				
	n=22 (I)	(II)	n=163 (III)							
Lymphocytes, %	34,6±2,3	33,6±0,2	32,8±0,2	<0,01	<0,05	<0,01				
CD3 ⁺ , %	$61,5\pm 2,2$	43,7±3,2	46,8±1,5	<0,001	<0,001	<0,001				
CD4 ⁺ , %	39,2±2,1	24,7±1,9	22,8±1,0	<0,001	<0,001	<0,001				
CD8 ⁺ , %	19,5±1,8	$15,4{\pm}1,4$	14,0±1,0	<0,01	<0,01	<0,05				
IRI (CD4/ CD8)	2,0±0,2	1,6±0,03	1,6±0,06	>0,05	>0,05	>0,05				

Table 1.Immunological parameters of children with BED and ChDED (M±m)



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CD16 ⁺ , %	10,2±1,3	16,7±0,5	18,3±0,6	<0,001	<0,001	<0,01
Fagotsitoz, %	58,5±2,3	$48,7{\pm}1,4$	45,6±1,6	<0,001	<0,001	<0,01
CD20 ⁺ , %	16,4±0,5	35,3±1,4	37,8±1,7	<0,001	<0,001	<0,01
CIK	1,01±0,1	1,8±0,3	2,5±0,4	<0,05	<0,01	<0,01
ASLO	1,2±0,1	4,3±0,2	6,2±0,3	<0,01	<0,01	<0,001

Note: P - reliability of differences between groups I and II; P1 - reliability of differences between groups I and III; P2 - reliability of differences between groups II and III.

When studying the number of T-helper cells (CD4+), their profound deficiency was revealed. Thus, if in the blood of practically healthy children circulates from 34 to 45% of T-helpers with an average value of $39,2\Box2,1\%$, the blood of children with BED and ChDED contains from 22 to 30% with an average value of $24,7\Box1,9\%$ and $22,8\pm1,0\%$, which is 1,5 times lower than the values of the control group (P<0,001), while the absolute values of CD4+-cells were 2,3 times lower than the control values (P<0,01). Another group of regulatory T-lymphocytes - T-suppressors (CD8+)-cytotoxic lymphocytes are able to inhibit strong and protracted immunological reactions. It has been found that 16 to 23% of T-suppressor/cytotoxic lymphocytes circulate in the blood of practically healthy children, with an average value of $19.5\pm1.8\%$. In children with BEB the relative value of CD8+ - lymphocytes was increased compared to the control up to $15,4\Box1,4\%$ and $14,0\pm1,0$ with individual variations from 20 to 32%. Against the background of suppression of CD4+ receptor expression and increase of CD8+, the value of immunoregulatory index (IRI) decreased by 49.5%. Changes in CD3+,CD4+,CD8+ - lymphocytes were more pronounced in the group of children with bronchiectatic disease than in children with deforming bronchitis (P<0.05; P<0.01).

In the group of healthy children, 7 to 15% of natural killer cells (CD16+) circulate - with an average relative value of $10.2\pm1.3\%$ in 1 µl. The study of phagocytic function in sick children, with BED and ChDED showed a disruption of these processes and confirms the increase in shifts with the activation of the inflammatory process. One of the reasons for the transition of acute pneumonia to prolonged pneumonia and to BED and ChDED, is a decrease in the functional activity of leukocytes, overstimulation of the functional state of neutrophils.

In the children we observed, phagocytosis was significantly reduced (48.7±1.4% and 45.6±1.6) with the norm of 58.5±2.3%, (P<0.01; P<0, 001). Theoretical and experimental works proved that reticuloplasmocytic tissue belongs to the immunocompetent system of the organism and plasma cells are the main producers of antibodies, the absence of qualitative differences in the picture of reticuloplasmocytic hyperplasia, bone marrow in patients in the phase of exacerbation and out of exacerbation of BED and ChDED. This indicates a prolonged, possibly permanent strain of the immunocompetent system in BEB and CDEB, in particular humoral immunity. This is proved by our data on the state of humoral immunity. The study of humoral immunity indices in 62 children with BED showed an increase in IgA 1,9±0,08 and IgM 1,6 \pm 0,6 in comparison with the norm (1,18 \pm 0,07; 1,18 \pm 0,07, respectively). There was also an increase in the amount of IgG 10,0±0,03 with the norm 9,03±0,21 (P<0,001). CIC concentration in children with BEB was significantly higher than in practically healthy children $(2.4\pm0.3 \text{ and } 2.5\pm0.4 \text{ vs. } 1.1\pm0.1, P<0.01; P<0.001)$, which confirms the high activity of the inflammatory process. When analysing CICs depending on the clinical form of BED and chronic deforming endobronchitis, the highest CIC levels were found in bronchiectatic disease. The analysis of our results revealed a sharp increase of ASL to lung antigen in children with BEB and CDEB, especially with BEB 6,5±0,3%, in comparison with children with deforming endobronchitis 5,2 \pm 0,4, while in children of practically healthy children this index is 1,1 \pm 0,1% (P<0,01). It should be noted that the content of antigen-binding lymphocytes during the exacerbation of BED and ChDED clearly reflects the severity of the pathological process, in the dynamics of ASL allows to assess the effectiveness of the therapy. Our studies have shown



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that the content of antigen-binding lymphocytes reflects the severity of the pathological process, and the dynamics of the index allows us to assess the effectiveness of therapy. Thus, the increase in the content of antigen-binding lymphocytes quantitatively characterises the development of autoimmune reaction. In patients of children with BED and ChDED in the phase of exacerbation of the disease in the blood are detected ASL, reacting not only with antigens of the pathogen, but also with antigens of lung tissue. In this case, a pronounced autoimmune reaction with a 3-4-fold increase in the blood content of ASL was observed.

Summarising the obtained results, it can be noted that the exacerbation phase of BED and ChDED in children is marked by profound disorders of immune status characterised by persistent T-cell immunodeficiency. The decrease in the level of T-lymphocytes in peripheral blood is apparently caused by redistribution of immunocompetent cells to the affected tissues of the lung, liver, thymus gland, vascular endothelium, as well as by the synthesis of immunosuppressive factors and, as a result, by increased intake of immature forms of immunocytes into the blood. The degree of cellular link depression was the higher, the more severe the pathological process in the lungs. The loss of functional activity of immunocompetent cells in CNCDL can be associated with the imbalance of oxidant and antioxidant systems. Analysis of new data on the pathogenesis of CHB, BED and ChDED allows us to conclude that a significant and prolonged increase in the intensity of POL is the most important mechanism of bronchopulmonary inflammation, reduces the functional activity of lymphocytes, causes the formation of immunodeficiency and, as a consequence, the severe course of the disease. Immunological reaction in childhood CNSLD has a clearly defined pathogenetic role in the development of various clinical manifestations of the disease, and therefore were registered in all studied nosological forms as a manifestation of secondary immunodeficiency state and autoimmune process in varying degrees of severity. Modern immunocorrective influence of secondary immune-dependent diseases has five main directions, among which the leading is correction by immune system hormones (thymic peptides, myelopeptides, etc.) and physical methods (laser irradiation) and application of ozone, etc.

Thus, concluding the chapter we can say that as a result of the conducted examinations we have established clinical and radiological, functional and bronchological features of chronic nonspecific lung diseases, which have their own peculiarities in the phase of exacerbation of the disease. In particular, in children with malformations of the bronchopulmonary system clinical symptoms of the disease in most cases appear only after the layering of infection, and in acquired forms of IBD it occurs after a prolonged inflammatory process in the lungs. In addition, in acquired forms of the disease, clinical and radiological manifestations of the disease are more uniform, while in congenital forms they depend on the type of malformation. As the term of the disease increases, the number of children with purulent endobronchitis difficult to be treated by traditional methods increases, which dictates the need for early diagnosis and treatment depending on the cause of the disease. In the study of patients with chronic non-specific lung diseases in different age groups, changes in lipid peroxidation, antioxidant system and immunological status were found, more pronounced in children with BED, indicating serious violations in the pathogenesis of the disease, which should be taken into account when carrying out therapeutic measures in patients.

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