

## AUTOIMUNE COMPONENT IN THE DEVELOPMENT OF CEBROVASCULAR INSUFFICIENCY IN CHILDREN WITH BRONCHIAL ASTHMA

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**Summary:** *Bronchial asthma (BA) remains one of the most serious diseases of our time. A number of studies have shown that this disease in a number of cases, especially with the threat of termination of pregnancy, acute and chronic diseases of mothers, accompanied by fetal hypoxia, originates in the ante- and postnatal period. In general, taking into account the peculiarities of the cellular tissue organization of the brain and cerebrovascular blood supply, prolonged hypoxic lesions increase the activity of the hypothalamic-diencephalic structures and the cerebral cortex. The existing respiratory disorders lead to hemodynamic and metabolic disorders of cerebral structures, emotional sphere and autonomic regulation. However, this issue in childhood requires further study. The aim is to study autoimmune processes in the pathogenesis of cerebrovascular insufficiency in children with BA. Materials and methods. We examined 121 patients with asthma aged 5 to 15 years in the period of exacerbation. To study the role of the autoimmune component in the development of cerebrovascular insufficiency and its relationship with the autoimmune process in the bronchopulmonary system in AD in children, we used a method for the quantitative determination of autoantibodies to lipopolysaccharide antigens (LA) of cerebral vessels and topographic structures of the brain, as well as to homologous LA bronchi and lung tissue. The results of the studies have shown that the first signs of cerebral hemodynamic disturbance are recorded already in patients with mild disease and are aggravated depending on the severity of BA. Conclusions. The most characteristic changes are an increase in the tone of small and medium vessels and impaired cerebral venous circulation. It was also found that the levels of autoantibodies to lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain correlate with an increase in the level of autoantibodies to lipopolysaccharide antigens of the trachea, bronchi and lung tissue and reflect the severity of AD in children.*

**Key words:** cerebrovascular insufficiency, children, bronchial asthma, lipopolysaccharide antigens, autoantibodies

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### Introduction.

In the last decade, many researchers pay attention to the growth and prevalence of bronchial asthma (BA) in children with an increase in the frequency of severe course and a high probability of secondary complications [3].

The multifactorial and multifaceted mechanisms of BA development in children

originate in the ante- and postnatal period. Conflict situations during pregnancy, the threat of termination, acute and chronic diseases accompanied by fetal hypoxia are observed in 70-85% of mothers with BA. Hypoxia leads to dysmetabolic processes in the central nervous system (CNS) and dysregulatory disorders of

the cortical-subcortical and spinal structures of the brain that regulate the respiratory complex, disrupting neurogenic regulation of bronchial smooth muscle tone and damaging the ciliated epithelium [1, 3, 4, 10].

This leads to increasing permeability of bronchi mucous membranes for allergens, development of sensitization, hyperreactivity of the bronchi, transient dysfunction of the immune system, changing the Th1 / Th2 ratio with an increase in the Th2 cytokine profile and development of an immune response for hypersensitivity of the immediate and delayed type [2, 3, 8, 11].

Taking into account peculiarities of the cellular tissue organization of the brain and cerebrovascular blood supply, prolonged hypoxic lesions of the central nervous system, especially in children with moderate and severe BA, increase the activity of hypothalamic-diencephalic structures and cerebral cortex. Respiratory disorders in BA lead to hemodynamic and metabolic disorders of the cerebral structures, emotional sphere and autonomic regulation. Clinically, this is manifested by psychoemotional, neurotic disorders, accompanied by complaints of increased fatigue, irritability, emotional lability, sleep disturbances, memory disorders, low efficiency, headaches. Dependence of the nature and severity of mental disorders on the severity and duration of BA is noted in children [6, 8, 12].

The pathogenetic characteristics of BA are determined by the productive type of recurrent inflammatory response, manifested by activation and unregulated proliferation of the interstitial connective stroma elements of the bronchopulmonary structures. Therefore, the main spectrum of immunological and immunopathological reactions in BA in children has a clear antigenic dependence on the inflammatory activated interstitial stroma of the bronchopulmonary system as well as on the effect of autoantibodies on cerebral vessels and cellular tissue structures of the brain, predetermining the development of cerebrovascular insufficiency in this contingent of children [2, 7].

In this regard, it is necessary to further study the pathogenetic aspects of BA in children as well as the influence of the autoimmune component in the development of cerebrovascular insufficiency with different severity of BA. Lack of information systematization on cerebrovascular insufficiency and mechanisms of its realization in children with BA in the available literature makes these studies the most relevant.

**The aim** of this work is to study the autoimmune component in the development of cerebrovascular insufficiency in children with BA.

#### **Material and methods of research**

An immunological study of blood serum was carried out in 121 children with BA at the age of 5 to 15 years in the period of the disease exacerbation.

At the present stage we have formed groups, depending on the severity of the disease course: with a mild course - 45 children, with moderate - 39 and severe - 37 children, in accordance with the main provisions of the International Consensus and the report of the National Institute of Heart, Lung, Blood (USA) together with the WHO - Global Initiative For Asthma ("GINA-2013"). The comparison group consisted of 25 healthy children. All children under study were randomized by sex, age, severity of the disease.

The study used lipopolysaccharide antigens from homologous cells of cerebral vessels, cellular tissue structures of the brain, trachea, bronchi, lung tissue obtained from sectional images from accidentally died children with I (0) blood group 2-4 hours from the moment of death according to V. D. Yakovenko et al. [5]

In accordance with the methodology the tested antigens had a chemical composition, represented by components of mainly lipopolysaccharides. There were no proteins in the antigenic preparations; peptides were detected only in the form of traces.

To elucidate the immunopathological role of the autoimmune component in the development of cerebrovascular insufficiency and its relationship with the autoimmune process in the bronchopulmonary system in BA in children,

we used the method of quantitative determination of autoantibodies to lipopolysaccharide antigens of cerebral vessels and topographic structures of the brain, as well as to homologous lipopolysaccharide antigens of bronchi and lung tissue in the Wanier nephelometric reaction modified by V.V. Kvirikadze et al. [5].

The state of the cerebral blood circulation in children with BA was studied and angiocerebral disorders were assessed, using rheoencephalography, which allows to characterize the elastic properties of cerebral vessels, and radiocirculoencephalography, which gives the most complete picture of the state of cerebral blood flow.

Rheoencephalography (REG) was recorded on a 4-channel rheograph connected to a 4-channel VNIIMIO electroencephalograph (type 4-M). REG was recorded in the frontomastoidal and occipitomastoidal abductions, characterizing the state of the cerebral blood flow in the carotid and vertebrobasilar vascular basins. [5].

The data of radiocirculoencephalography (RCEG) were recorded on a three-channel radiograph "Gamma" (Hungary) using hippuran J-131 as a radioactive indicator. Cerebral blood flow was investigated and evaluated in all areas of the hemispheres. The quantitative analysis of the RCEG-indicator was based on the data of its first derivative, which allows one to judge the state of the arterial, capillary and venous phases of cerebral blood flow.

The obtained data were processed statistically by the method of variation series according to the Student's t-test at ( $p < 0,05$ ).

### **Results and its discussion**

The results of the studies have shown that the examined children with BA have an autoimmune control of the lipopolysaccharide antigens of the cerebral vessels and the corresponding parts of the brain, increasing dependence on the disease severity (Table 1).

In the group of children with mild asthma, the level of autoantibodies to lipopolysaccharide antigens of the thoracic aorta, common and internal carotid arteries, hemolysate was found at weakly positive values, indicating the "trigger" value of autoimmune reactions of the extracerebral great vessels in the formation of cerebrovascular insufficiency. In the group of children with mild BA, the autoimmune control of lipopolysaccharide antigens of cerebral vessels is observed at weakly positive values of common carotid, internal carotid, basilar and anterior cerebral arteries; veins: basilar, common basilar; cerebral cortex: frontal, temporal, parietal. The rest of the cerebral vessels and veins did not fall under the autoimmune control of the body (Table 1).

There is also autoimmune control for children with mild BA at the level of weakly positive values for the cellular tissue structures of the frontal, parietal cortex, as well as the basal ganglia, pons varoli. Weakly positive values of the level of autoantibodies to lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain in children with mild asthma make it possible to predict these changes as reversible in the cerebrovascular insufficiency development (Table 1). In groups of children with moderate and especially severe BA, there was an increase in the level of autoantibodies to positive values to lipopolysaccharide antigens of cerebral vessels and brain regions located in the zone of their vascularization. It should be noted that the pathological changes in the cerebral vessels and the corresponding areas of the brain are not equal. Lipopolysaccharide antigens of the basilar, anterior cerebral, middle and posterior cerebral arteries; veins: basilar and general basilar, as well as to the cellular tissue structures of the temporal, parietal and occipital cortex, as well as to the cellular tissue structures of the basal ganglia and the pons varoli are under autoimmune control most often (Table 1).

Table 1. Quantitative determination of autoantibodies to homologous lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain in children with BA in the period of exacerbation ( $M \pm m$ ), conv. Units

Vascular and tissue antigens	BA Severity			Healthy children (control group) n = 25
	Light n = 45	Moderate severity n = 39	Severe n = 37	
1	2	3	4	5
Aorta : Chest	0,125±0,013* E <sub>x</sub> =0,19	0,142±0,024* E <sub>x</sub> =0,21	0,189±0,028* <sup>^</sup> E <sub>x</sub> =0,22	0,023±0,004
abdominal	0,068±0,12 E <sub>x</sub> =0,20	0,097±0,015* E <sub>x</sub> =0,21	0,118±0,021* E <sub>x</sub> =0,22	0,026±0,006
Arteries: Common carotid	0,124±0,021* E <sub>x</sub> =0,16	0,146±0,025* E <sub>x</sub> =0,18	0,164±0,029* <sup>^</sup> E <sub>x</sub> =0,23	0,019±0,003
External carotid	0,057±0,009 E <sub>x</sub> =0,17	0,085±0,014* E <sub>x</sub> =0,23	0,116±0,021* <sup>^</sup> E <sub>x</sub> =0,24	0,020±0,004
Internal carotid	0,123±0,018* E <sub>x</sub> =0,17	0,133±0,021* E <sub>x</sub> =0,24	0,161±0,021* <sup>^</sup> E <sub>x</sub> =0,26	0,024±0,003
Basilar	0,125±0,020* E <sub>x</sub> =0,23	0,162±0,028* E <sub>x</sub> =0,28	0,288±0,045* <sup>^</sup> E <sub>x</sub> =0,32	0,033±0,007
anterior cerebral	0,127±0,024* E <sub>x</sub> =0,25	0,148±0,029* E <sub>x</sub> =0,22	0,269±0,048* <sup>^</sup> E <sub>x</sub> =0,21	0,029±0,005
middle cerebral	0,126±0,017* E <sub>x</sub> =0,16	0,136±0,023* E <sub>x</sub> =0,19	0,273±0,036* <sup>^</sup> E <sub>x</sub> =0,23	0,024±0,003
posterior cerebral	0,125±0,022* E <sub>x</sub> =0,21	0,162±0,032* E <sub>x</sub> =0,27	0,187±0,029* <sup>^</sup> E <sub>x</sub> =0,25	0,031±0,006
Veins: basilar	0,124±0,023* E <sub>x</sub> =0,24	0,169±0,32* <sup>^</sup> E <sub>x</sub> =0,31	0,285±0,047* <sup>^</sup> E <sub>x</sub> =0,27	0,036±0,007
common basilar	0,125±0,021* E <sub>x</sub> =0,22	0,152±0,029* E <sub>x</sub> =0,31	0,224±0,038* <sup>^</sup> E <sub>x</sub> =0,25	0,028±0,004
Superior vena cava	0,076±0,013 E <sub>x</sub> =0,19	0,094±0,016* E <sub>x</sub> =0,21	0,128±0,022* <sup>^</sup> E <sub>x</sub> =0,29	0,034±0,006
Cortex: frontal	0,123±0,021* E <sub>x</sub> =0,24	0,147±0,027* E <sub>x</sub> =0,26	0,169±0,033* <sup>^</sup> E <sub>x</sub> =0,27	0,023±0,004
temporal	0,079±0,015 E <sub>x</sub> =0,19	0,102±0,018 E <sub>x</sub> =0,21	0,119±0,021* <sup>^</sup> E <sub>x</sub> =0,23	0,026±0,006
parietal	0,124±0,023* E <sub>x</sub> =0,22	0,157±0,029* E <sub>x</sub> =0,18	0,237±0,045* <sup>^</sup> E <sub>x</sub> =0,24	0,028±0,005
occipital	0,073±0,011 E <sub>x</sub> =0,16	0,096±0,017 E <sub>x</sub> =0,19	0,175±0,034* <sup>^</sup> E <sub>x</sub> =0,20	0,032±0,006

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1	2	3	4	5
Brain tissue structures: large hemispheres	0,076±0,013 E <sub>x</sub> =0,25	0,098±0,015* E <sub>x</sub> =0,28	0,156±0,032*^ E <sub>x</sub> =0,26	0,029±0,005
Basal ganglia	0,123±0,025* E <sub>x</sub> =0,29	0,148±0,026* E <sub>x</sub> =0,32	0,184±0,033*^ E <sub>x</sub> =0,27	0,025±0,006
Hypothalamus	0,078±0,014 E <sub>x</sub> =0,18	0,111±0,017*^ E <sub>x</sub> =0,28	0,121±0,021*^ E <sub>x</sub> =0,31	0,027±0,004
Pons Varolii	0,124±0,023* E <sub>x</sub> =0,25	0,135±0,025* E <sub>x</sub> =0,33	0,169±0,032*^ E <sub>x</sub> =0,24	0,031±0,005
Medulla	0,059±0,007 E <sub>x</sub> =0,21	0,109±0,014*^ E <sub>x</sub> =0,23	0,121±0,021*^ E <sub>x</sub> =0,20	0,024±0,003
Cerebellum	0,065±0,012 E <sub>x</sub> =0,21	0,084±0,018* E <sub>x</sub> =0,32	0,119±0,021*^ E <sub>x</sub> =0,25	0,033±0,007
Hemolysate	0,154±0,023* E <sub>x</sub> =0,32	0,267±0,043*^ E <sub>x</sub> =0,28	0,384±0,056*^ E <sub>x</sub> =0,33	0,042±0,008

Note:

1. Negative reaction in the values of  $Q_{\phi} = 0,0004-0,1236$ ;  
slightly positive reaction in the values of  $Q_{\phi} = 0,1237-0,1633$ ;  
positive reaction in the values of  $Q_{\phi} = 0,1634-0,6411$ ;  
sharply positive reaction in the values of  $Q_{\phi} = 0,6412-1,4248$ ;
2. \* - significant differences in indicators from the group of healthy children ( $p < 0,05$ );
3. ^ - significant differences in indicators from the group of children with mild BA ( $p < 0,05$ );
4. E<sub>x</sub> – indicator of the sample distribution normality ( $E_x = 0$ ).

Thus, the results of the immunological studies have shown that one of the leading links in the pathogenesis of cerebrovascular insufficiency is the increased autoimmune control of cerebral vessels and tissue structures of the brain, accompanied by a complex of clinical symptoms, aggravating the severity of the course of the disease. The presence of autoaggression against cerebral vessels and brain tissue indicates the development of cerebral vasculitis, which leads to cerebral circulatory disorders and the development of these clinical symptoms [9].

The state of blood circulation and the elastic properties of the cerebral vessels were studied using rheoencephalography (REG). During the REG study, it has been found that changes in cerebral hemodynamics in the vertebrobasilar and carotid basins are unidirectional, therefore, the REG in the frontomastoidal and occipitomastoidal abductions was characterized simultaneously.

Analysis of the REG data shows an increase in the tone of the vascular wall of arterial vessels of small and medium calibers, as evidenced by the lengthening of the anacrotic REG phase, an increase in the REG wave propagation velocity, an increase in the dirotic index, and a change in the tonic tension of the arterial vessels of the brain. Signs of cerebral venous outflow obstruction of blood were also identified: presystolic waves, two-stage anacrot, appearance of an additional wave at the end of the descending frequency of the REG curve.

When analyzing the data, the RCEG revealed three types of changes in cerebral hemodynamics in the examined patients. The first type is the closest to the norm. Evaluation of the temporal parameters of the curves showed some shifts towards an increase in the duration of all phases of cerebral blood flow. In the second type of cerebrovascular disorders, more pronounced changes in

cerebral blood flow were observed. Against the background of an increase in the total time of cerebral hemodynamics, a differentiated duration lengthening of the capillary phase was noted, indicating predominant changes in the microcirculation system. The third type of cerebral blood flow disorders was characterized by further deterioration in the state of cerebral hemodynamics. A significant increase in the duration of the venous phase was revealed along with the lengthening of the arterial and capillary phases.

Analysis of the study results shows that the severity of cerebrovascular disorders in children with BA depends on the severity of the disease and the increase in the level of autoantibodies to lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain.

In 65% of patients with mild asthma, there was an increase in the tone of arterial vessels, mainly of medium and small caliber. Signs of decreased vascular tone were noted in 10% of cases. Normal REG was registered in 25% of patients in this clinical group. The first and second types of RCEG were the most typical for children with a mild course of the disease.

An increase in the tone of small-caliber arterial vessels and a decrease in the duration of the catacrotic phase of REG were noted in 84.95% of cases in children with moderate bronchial asthma as compared with the group with mild BA. In addition, difficulties in cerebral venous outflow of blood were identified in 15.05% of cases. In this group the second and third types of RCEG dominated.

The REG study of patients with severe BA revealed the highest severity of cerebral hemodynamic disorders. A further increase in the tonic tension of small-caliber arterial vessels, a decrease in the duration of the catacrotic phase of REG were noted in children of this group as compared with the group of patients with a moderate course, and the most pronounced signs of obstruction of venous outflow were revealed. This group was dominated by the third type of RCEH. It should be noted that in most cases, changes in the vertebrobasilar vascular system prevailed. These data correlate with an increase in the level of autoantibodies to lipopolysaccharide

antigens of the basilar artery and veins: basilar and common basilar (Table 1).

The results of the studies have shown that the first signs of cerebral hemodynamic disturbance are recorded already in patients with a mild course of the disease and are aggravated depending on the severity of the course of bronchial asthma. The most characteristic rheoencephalographic changes in BA in children are an increase in the tone of small and medium vessels and impaired cerebral venous circulation. Assessment of the RCEG data, which allows to trace individual phases of cerebral blood flow, also revealed predominant changes in the microcirculation system and insufficient vascularization of cerebral structures with obstruction of venous outflow. This also correlates with the level of autoimmune reactions to lipopolysaccharide antigens of the basilar, common basilar and superior vena cava.

The leading role in the BA pathogenesis in children belongs to autoimmune reactions in the cellular tissue structures of the bronchopulmonary system. Tissue antigens, which are lipopolysaccharides of the cytoplasmic membranes of the cellular structures of the trachea, bronchi and lung tissue, by chemical composition, were used in their detection. Their antigenic composition includes collagen II and V types presented in cerebral vessels and cellular tissue structures of the brain tissue.

Autoimmune reactions in the cellular tissue structures of the bronchopulmonary system lead to their cytolysis and the release of lysosomal enzymes, which, affecting the structures of type III and V collagen, open the determinants of collagen  $\alpha$ -chains, and it acquires greater antigenicity. Autoimmune control of the cellular tissue structures of the bronchopulmonary system in children with BA leads to the development of autoimmune reactions in the cerebrovascular vessels and cellular tissue structures of the brain.

A rank correlation analysis was carried out to prove that there is a direct connection between autoimmune reactions to antigens of lipopolysaccharides of cellular tissue structures of the bronchopulmonary system

and lipopolysaccharide antigens of cerebral vessels and brain structures.

The first rank sample represented the degree of deviation from the standard (t-test) of the titer of autoantibodies to lipopolysaccharide antigens of the trachea, bronchi and lung tissue in patients of the three study groups. Thus, in children with mild BA, the level of autoantibodies to tracheal lipopolysaccharide antigens ranged from 0.065-0.0116 conventional units; to bronchial antigens from 0.097-0.123 conventional units; lung tissue - 0.226-0.244 conventional units. In children with moderate BA, the level of autoantibodies to tracheal lipopolysaccharide antigens ranged from 0.114-0.133 conventional units; to bronchial antigens from 0.224-0.308 conventional units; lung tissue - 0.264-0.311 conventional units. In children with severe BA the level of autoantibodies to tracheal lipopolysaccharide antigens ranged from 0.134-0.272 conventional units; to bronchial antigens from 0.258-0.325 conventional units; lung tissue - 0.303-0.339 conventional units. The second sample represented the level of autoantibodies to lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain in children of the studied groups. After ranking the values of the t-criterion in each sample a rank correlation was carried out. As a result, it has been found that there is a direct reliable relationship between the two autoimmune processes. The rank correlation coefficient had the values  $P_s = 0.67$  ( $p < 0.05$ ).

Thus, the results of the studies have shown that in the pathogenesis of cerebrovascular insufficiency in children with BA there is an autoimmune control of cerebral vessels and cellular tissue structures of the brain, the severity of which depends on the severity of the course of the disease.

### Conclusions

1. An immune diagnostic has been developed, which includes determination of autoantibodies to homologous lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain. This

allows to diagnose the degree of cerebrovascular insufficiency in children with BA with high accuracy.

2. Indicators of autoantibodies level to lipopolysaccharide antigens of arteries, venous vessels and cellular tissue structures of the brain, makes it possible to detect lesions of blood vessels and tissue areas of the brain in cerebrovascular insufficiency in children with BA.

3. Levels of autoantibodies to lipopolysaccharide antigens of cerebral vessels and cellular tissue structures of the brain correlate with an increase in the level of autoantibodies to lipopolysaccharide antigens of the trachea, bronchi and lung tissue and reflect the severity of BA in children.

4. It is necessary to differentially include immunomodulatory drugs and correctors of hemodynamic disorders in the complex protocol of BA therapy in children to correct autoimmune reactions of cerebral vessels and cellular tissue structures of the brain.

### Prospects for further research

The research is promising for improving immunological diagnostics, development and production of tissue antigens and their use in diagnostics of secondary complications and developing new approaches to pathogenetic therapy of the disease.

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## ЗНАЧЕННЯ АВТОІМУННИХ ПОРУШЕНЬ У ФОРМУВАННІ ЦЕРЕБРОВАСКУЛЯРНОЇ НЕДОСТАТНОСТІ У ДІТЕЙ ПРИ БРОНХІАЛЬНІЙ АСТМІ

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**Резюме:** Бронхіальна астма (БА) залишається одним з важких захворювань сучасності. У ряді досліджень показано, що дане захворювання в ряді випадків, особливо в разі загрози переривання вагітності, гострих і хронічних захворювань вагітних, що супроводжуються гіпоксією плода бере свій початок ще в анте- і постнатальному періоді. В цілому, враховуючи особливості клітиннотканьової організації мозку і цереброваскулярного кровопостачання тривалі гіпоксичні ураження посилюють активність гіпоталамо-діенцефальних структур і кори головного мозку. Наявні дихальні розлади призводять до гемодинамічних і метаболічних порушень церебральних структур, емоційної сфери і вегетативної регуляції. Однак, дане питання в дитячому віці вимагає подальшого вивчення. **Мета** - вивчення автоімунних процесів в патогенезі цереброваскулярної недостатності у дітей, хворих на бронхіальну астму. **Матеріали та методи.** Обстежено 121 пацієнт з БА у віці від 5 до 15 років в періоді загострення. Для вивчення ролі автоімунного компонента в розвитку цереброваскулярної недостатності та зв'язку його з автоімунним процесом в бронхолегеневій системі при БА у дітей, використовувалася метод кількісного визначення аутоантитіл до ліпополісахаридних антигенів (ЛПА) церебральних судин і топографічних структур головного мозку, а так само до гомологічних ЛПА трахеї, бронхів і легеневої тканини. **Результати** проведених досліджень показали, що перші ознаки порушення церебральної гемодинаміки реєструються вже у хворих з легким перебігом захворювання та поглиблюються в залежності від ступеня тяжкості перебігу БА. **Висновки.** Найбільш характерними змінами є підвищення тонуусу дрібних і середніх судин і порушення венозного мозкового кровообігу. Також встановлено, що рівні аутоантитіл до ліпополісахаридних антигенів церебральних судин і клітиннотканьових структур головного мозку корелюють з наростанням рівня аутоантитіл до ліпополісахаридних антигенів трахеї, бронхів і легеневої тканини і відображають тяжкість перебігу БА у дітей.

**Ключові слова:** цереброваскулярна недостатність, діти, бронхіальна астма, ліпополісахаридні антигени, аутоантитіла

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## ЗНАЧЕНИЕ АУТОИММУННЫХ НАРУШЕНИЙ В ФОРМИРОВАНИИ ЦЕРЕБРОВАСКУЛЯРНОЙ НЕДОСТАТОЧНОСТИ У ДЕТЕЙ ПРИ БРОНХИАЛЬНОЙ АСТМЕ

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**Резюме.** Бронхиальная астма (БА) остается одним из тяжелых заболеваний современности. В ряде исследований показано, что данное заболевание в ряде случаев, особенно при угрозе прерывания беременности, острых и хронических заболеваниях матерей, сопровождающиеся гипоксией плода берет свое начало еще в анте- и постнатальном периоде. В целом, учитывая особенности клеточнотканевой организации мозга и цереброваскулярного кровоснабжения длительные гипоксические поражения усиливают активность гипоталамо-диэнцефальных структур и коры головного мозга. Имеющиеся дыхательные расстройства приводят к гемодинамическим и метаболическим нарушениям церебральных структур, эмоциональной сферы и вегетативной регуляции. Однако, данный вопрос в детском возрасте требует дальнейшего изучения. **Цель** – изучение аутоиммунных процессов в патогенезе цереброваскулярной недостаточности у детей, больных БА. **Материалы и методы.** Обследован 121 пациент с БА в возрасте от 5 до 15 лет в периоде обострения. Для изучения роли аутоиммунного компонента в развитии цереброваскулярной недостаточности и связи его с аутоиммунным процессом в бронхолегочной системе при БА у детей, использовался метод количественного определения аутоантител к липополисахаридным антигенам (ЛА) церебральных сосудов и топографических структур головного мозга, а так же к гомологичным ЛА трахеи, бронхов и легочной ткани. **Результаты** проведенных исследований показали, что первые признаки нарушения церебральной гемодинамики регистрируются уже у больных с легким течением заболевания и усугубляются в зависимости от степени тяжести течения БА. **Выводы.** Наиболее характерными изменениями являются повышение тонуса мелких и средних сосудов и нарушение венозного мозгового кровообращения. Также установлено, что уровни аутоантител к липополисахаридным антигенам церебральных сосудов и клеточнотканевых структур головного мозга коррелируют с нарастанием уровня аутоантител к липополисахаридным антигенам трахеи, бронхов и легочной ткани и отражают тяжесть течения БА у детей.

**Ключевые слова:** цереброваскулярная недостаточность, дети, бронхиальная астма, липополисахаридные антигены, аутоантитела

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**Конфлікт інтересів:** відсутній.

**Конфликт интересов:** отсутствует.