RECURRENT BRONCHITIS IN CHILDREN: STATE OF THE PROBLEM

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Great interest of many researchers and practitioners to the problem of recurrent bronchitis is displayed not only by high frequency of occurrence in young children, but also by a number of vexed questions about classification, the difficulties of differential diagnosis, the possibility of modifying into other nosological forms at later stages of life.

Today, there are some changes in the clinic of the disease, which depend on several etiological factors, that cause airway hyperreactivity. A variety of categories in children with recurrent bronchitis (RB) is due to the multifactorial nature of the disease.

The diseases of the respiratory system still remain an actual problem of pediatrics and occupy one of the first places in the structure of childhood morbidity. One of the prevalence lesions of the lower respiratory tract are bronchitis, which develop in 50% of children [1,3]. The frequency of recurrent bronchitis is 16.4 cases per 1000 children [5]. In modern classification it is noted, that RB usually occurs in children of the first 4–5 years of life. According to many authors, this nosology is most often registered under 7 years old [1,4].

Recurrent bronchitis is a multifactorial, environmentally dependent disease, the main pathogenetic link of which is recurrent inflammation in the mucous membrane of the bronchial tree, which is caused by a decrease in local protection factors and the general immunological resistance of the organism. It should be noted, that respiratory viral infections with repeated episodes of the broncho-obstructive syndrome, form bronchial hyperreactivity, which determines the probability of phenotypic realization of asthma in younger children [4].

In the basis of the age limit are features of the respiratory and immune systems, which are largely connected with the processes of their development and maturation. According to the literature, intensive processes of growth and differentiation of lung tissue elements take place in the first years of a child's life, and morphogenesis is considered completed by 6–7 years old [1, 2]. At the age of 1–6 years, the immune response to infectious antigens is reoriented from the prevalence of the Th2–way of the response, which is typical for younger children, to the Th1 response, typical for the infectious process in adults [3].

The biocenosis of the upper respiratory tract in babies is also in formation process. At that, the microbial paysage is very unstable, polymorphic, dependent on the environment and varies with the age of the child, becomes similar of an adult only at the age of 5–8 years [5]. The lower respiratory tract in younger children is often involved in respiratory infections with the development of bronchial obstruction syndrome, which determines the severity of patient status and often causes hospitalization. According to the literature, the frequency of expressed clinical signs of bronchial obstruction in case of recurrent bronchitis is 70–80% [1].

In the foreign literature isolated phenotypes, that have both clinical and prognostic value. By debut and duration, there is a transitory obstruction, which is observed in children only in the first three years of life, and persistent, which is observed in the first 6 years of life. The development of obstructive bronchitis in children after three years of life is considered as late starting and is divided into atopic and non-atopic phenotype, that is, in combination with and without atopy [4, 5]. Analysis of the information, obtained during long-time observation showed, that lung function in children with transitory obstruction was changed from birth, even before the first episode. The risk of developing asthma in this group is not great. In contrast, lung function in children with persistent obstruction at birth is intact and, at the same time, there is a high risk of developing asthma [4, 5].

Research in recent years separated the most significant factors ,which induce the development of recurrent bronchitis in children: age-related features of the respiratory tract and chest; respiratory infection (respiratory syncytial viruses, influenza and parainfluenza viruses, adenovirus, mycoplasma); features of local immunity; social factors (passive smoking, early attendance of kindergarten); environmental factors; aggravated obstetric, perinatal anamnesis, hereditary and allergic anamnesis, comorbidity of ENT, and other foci of chronic infection, thymomegaly [2, 5].

Currently, there is a consensus about the main role of infection in development and exacerbation of recurrent bronchitis. The greatest importance in the etiology of bronchitis from infectious factors are viruses (95% of cases) and viral-bacterial associations.

The bacterial nature of the disease (pneumococcus, hemophilic bacillus) is second, then fungi and protozoa, and in some cases may be atypical flora (mycoplasma, chlamydia, pneumocysts, legionella). The leading place among viruses is occupied by respiratory syncytial (RS) viruses, adenoviruses. More seldom, enteroviruses, morbilli virus, cytomegaloviruses, coronarovirus, ECHO viruses, Coxsackie viruses cause of bronchitis [1, 3].

There are reports, that children with atopic phenotype of obstructive syndrome are genetically predisposed to the persistent course of some viral and atypical (RS viruses, adenoviruses, parainfluenza, bokaviruses) infections [3,4]. The respiratory viruses damage the ciliated epithelium of mucous membrane in the respiratory tract, increase its penetrability to allergens, toxic substances, and the sensitivity of the irritating receptors of submucous tissues of the bronchi, which causes an increase in bronchial hyperreactivity [1, 2, 4].

The development of inflammation, which can be caused by infectious, toxic, allergic, and physical effects, takes the main place in the pathogenesis of airway hyperreactivity. The cytokine system is the initiator of a cascade of reactions, which leads to releasing of new mediators and cell migration to the inflammatory focus, transforming the "vicious circle", predisposing to prolonged bronchial obstruction and superinfection [1]. Inflammatory processes that develops by viruses, lead to edema of the mucous membrane, hypersecretion of viscous mucus, disturbance of mucociliary transport [2].

Therefore, disturbance in the drainage function of the bronchial tree conduces to colonization with pathogenic microflora [1, 2]. Besides, viruses inhibit the activity of macrophages and reduce local nonspecific immunity. As a result, conditions are created for bacterial or mixed superinfection (viral-bacterial), that leads to development of protracted inflammatory process [2]. Currently, the question is being discussed: is asthma realized by recurrent respiratory tract infection, or are children predisposed to bronchial asthma, because they have an altered level of interferon or another cytokine response [3, 4]? Great importance in the development of RB is given to ante- and perinatal factors. In children born by mothers with preeclampsia, hypertension, diabetes, risk of developing early transient, persistent obstruction, antibiotics during parturition can cause both early transient and persistent obstruction is increased [1, 5].

Thus, given data evidences, that the etiopathogenesis of RB is complex, not all of its links have been studied enough. Further research in this way will optimize the criteria for predicting the risk of RB in children.

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