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MODERN APPROACHES TO THE USE OF MUCOLYTIC AND BRONCHODILATOR AGENTS IN ACUTE RESPIRATORY VIRAL INFECTIONS WITH BRONCHIAL OBSTRUCTION IN CHILDREN

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Annotation

In children with acute respiratory viral infections, the lower respiratory tract is often involved, leading to the development of bronchial obstruction. The use of the combined preparation Ascoril Expectorant (bromhexine + salbutamol + guaifenesin), due to its combined bronchodilator and mucolytic effects, results in rapid regression of disease symptoms, prevents progression to a more severe and prolonged course, and subsequently reduces the need for antibiotic therapy. The clinical efficacy and favorable safety profile of Ascoril Expectorant allow it to be recommended as the drug of choice for children over 2 years of age with mild to moderate bronchial obstruction associated with ARVI.

Keywords

bromhexine, salbutamol, guaifenesin, Ascoril

Acute respiratory viral infections (ARVIs) account for more than half of all acute infectious diseases in children, and during epidemic periods, the proportion of ARVIs rises to 80–90%. The highest incidence rates of ARVIs are observed in preschool-aged children. Young children experience ARVIs on average 3–4 times per year, with 30–40% of children in this age group experiencing 6–8 episodes annually. Children who begin attending preschool institutions in their first year contract ARVIs 1.5 times more often than peers who remain at home. The frequency of ARVIs decreases with age; school-aged children experience an average of 2–3 episodes per year.

ARVIs occur year-round, although the highest number of cases is observed from early autumn to late spring.

In children with respiratory diseases, especially in early childhood, the lower respiratory tract is often involved in the pathological process, leading to the development of obstructive syndrome (OS). Several phenotypes of bronchial obstruction exist, with both clinical and prognostic significance. Transient obstruction is observed only in the first three years of life, while persistent obstruction primarily occurs in the first six years. OS can develop in children after three years of age (late-onset), in which case atopic and non-atopic phenotypes are distinguished, i.e., OS with or without atopy. Long-term follow-up data indicate that children with transient obstruction exhibit altered lung function from birth, even before the first obstruction episode. In contrast, children with persistent obstruction and a high risk of developing bronchial asthma (BA) have normal lung function at birth, with obstructive changes developing between ages 4–6. One significant factor predisposing children to virus-induced

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obstructive symptoms in early life may be a reduced airway lumen due to impaired fetal development in the antenatal period.

Obstructive syndrome in preterm children with lung disease is usually associated with a combination of lung immaturity, oxygen therapy, and ventilatory support. This primarily concerns infants with low birth weight and severe neonatal respiratory disease. However, even preterm children without neonatal illnesses may demonstrate reduced respiratory function later in life. It is suggested that preterm birth may predispose children to an obstructive pattern. Testing healthy preterm children in their second year of life shows that lung function does not normalize during the period of maximal lung growth. The mechanism of persistent reduced lung function in preterm children is not fully understood. It may result from a narrower airway lumen and decreased lung tissue elasticity, leading secondarily to altered alveolarization of the lung parenchyma.

Respiratory viral infections in early life may be a more important risk factor for bronchial obstruction than atopy. During the autumn–winter period, severe cases of bronchiolitis in children under two years are often caused by respiratory syncytial virus (RSV) and exhibit a typical seasonal pattern. Severe bronchiolitis is associated with a 30–40% likelihood of developing asthma. RSV combined with human bocavirus leads to more severe disease.

The second most common etiological factor of bronchiolitis is rhinovirus (RV). Moderate to severe RV infection increases the risk of developing asthma. Many other viral infections—such as influenza, parainfluenza, coronavirus, enterovirus, adenovirus, human metapneumovirus, and bocavirus—affect small bronchi and bronchioles, presenting clinically as bronchiolitis. In neonates with atopy risk, moderate or severe obstruction due to RV infection is a significant factor not only for recurrent obstruction episodes by age three but also for asthma development by age six. Studies indicate that the leading risk factors for recurrent bronchial obstruction after acute bronchiolitis are RV infection and a family history of asthma. Children with RV infection treated with oral glucocorticosteroids had a significantly lower probability of developing recurrent obstruction.

Investigation of the balance between Th1- and Th2-type immune responses showed that in atopic children, peripheral mononuclear cells incubated with RV produce interleukin-10 (IL-10), whereas in non-atopic children, they produce gamma-interferon and IL-12. Reduced mononuclear cell capacity to produce gamma-interferon and IL-12 decreases viral clearance and may promote asthma exacerbation by supporting Th2-type inflammation and insufficient Th1 antiviral response. Currently, it is debated whether recurrent respiratory infections cause airway damage leading to asthma or whether children are predisposed to BA due to altered interferon levels or other cytokine responses.

Understanding postnatal processes requires knowledge of antenatal development. Airway branching occurs in the first half of pregnancy, and antenatal factors may influence airway caliber. The ADAM33 gene plays a key role in antenatal lung development, particularly in airway branching and determining airway caliber.

Maternal atopy is associated with impaired lung function in newborns, although the mechanism is unclear. Children born to mothers with preeclampsia, hypertension, or diabetes have an

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increased risk of early transient, persistent, or late-onset obstruction. Antibiotic administration during labor may contribute to both early transient and persistent obstruction.

Maternal smoking negatively affects fetal lung development, reducing IL-4 and gamma-interferon levels while increasing mononuclear cell proliferation in response to dust allergens. Umbilical cord blood studies show maternal smoking increases IL-13 and reduces gamma-interferon mRNA response after stimulation, also affecting TNF-alpha production. Epidemiological studies confirm that maternal smoking and atopy are associated with bronchiolitis in infants. Maternal smoking significantly influences newborn immune response and anatomical development of the lower airways (alveolar underdevelopment).

RSV bronchiolitis is associated with increased Th2 cytokine expression or reduced Th1 response, supporting the hypothesis that RSV may induce asthma. Bronchial hyperreactivity persists after bronchiolitis, explaining predisposition to obstruction later. Detection of RSV-specific IgE in children suggests early RSV infection may trigger a Th2-type immune response predisposing to asthma.

Genetic studies show polymorphisms in IL-8, IL-10, and toll-like receptor (TLR) genes correlate with RSV infection severity. Asthma development in otherwise healthy children may result from prior exposure to adverse genetic and antenatal factors during RSV bronchiolitis, making bronchiolitis a marker rather than a direct cause of subsequent respiratory problems. Subsequent viral infections in these children may lead to complications and severe obstruction, requiring urgent and adequate treatment.

There is a hypothesis that early viral exposure may prevent asthma development later. Early attendance at organized childcare is associated with more frequent obstruction, but asthma develops less often by age six compared to children who remain at home. Most asthma exacerbations are virus-associated. Early diagnosis of BA is challenging due to variability and non-specific clinical manifestations and limitations of functional testing. In preschool and school-aged children, allergen exposure, sensitization, and viral infections interact.

The non-atopic phenotype of bronchial obstruction presents the greatest diagnostic difficulty. Severe adenoviral infection may cause prolonged obstruction in previously healthy children. Respiratory viruses (RV, RSV, metapneumovirus, influenza) affect the lower airway epithelium, provoking local immune responses and antiviral defense via interferons, chemotaxis, and NK-cell activation. The airway epithelium is central to respiratory disorders, producing secretions containing nonspecific and specific protective factors, responding to immune signals, and regulating innate and adaptive immunity, including Th2 inflammation, structural airway changes, and angiogenesis.

Regardless of predisposing factors, recurrent respiratory infections with bronchial obstruction contribute to bronchial hyperreactivity, promoting recurrent and chronic bronchitis or asthma risk. Optimizing diagnostic and therapeutic strategies to timely correct airway obstruction is a key pediatric and pulmonology task.

Given the diversity and severity of clinical symptoms, treatment of children with ARVIs uses drugs targeting multiple components of the pathological process. Mucokinetic agents (ambroxol,

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acetylcysteine, carbocisteine) play an important role in managing cough by thinning mucus, increasing its liquid fraction, stimulating ciliary function, and promoting surfactant production. These drugs optimize the rheological properties of airway secretions, supporting mucociliary clearance recovery.

The cornerstone of pharmacological therapy for bronchial obstruction in respiratory infections is bronchodilators. In mild obstruction with tenacious mucus during ARVI, combined medications containing mucolytics and bronchodilators are effective. The combined preparation Ascoril provides bronchodilatory and expectorant effects, shortens cough duration, converts cough to productive, and enhances mucociliary transport.

Components of Ascoril – bromhexine + salbutamol + guaifenesin – exert bronchodilator, mucolytic, and expectorant effects. Salbutamol, a fast-acting beta-2 agonist, provides bronchodilation. Bromhexine hydrochloride and its active metabolite ambroxol depolymerize and break down mucoproteins and mucopolysaccharides in mucus, stimulating secretory cells. Guaifenesin enhances the liquid fraction of mucus, reduces surface tension and adhesiveness, alleviates anxiety, psychogenic and autonomic symptoms, and improves sleep. Menthol (racemethol) provides mild spasmolytic and weak antiseptic effects.

Clinical data show Ascoril Expectorant in children aged 2–10 with mild or moderate obstruction during ARVI significantly improves disease course, as assessed by physicians and parent questionnaires. ARVIs in these children involved various respiratory regions (laryngotracheitis, bronchitis). Dosage: children under 6 years, 5 mL (1 tsp) 3 times/day; children 6–10 years, 5–10 mL (1–2 tsp) 3 times/day. Best outcomes were observed with early initiation (first days of illness), treatment duration 7–10 days depending on symptom regression.

Short-acting beta-2 agonists (salbutamol) in Ascoril are justified because over 60% of children show impaired bronchial patency via bronchophony. High-frequency oscillations (>5000 Hz) and spirometry deviations indicating mild/moderate obstruction were seen in >50% of children. Clinical signs of obstruction included brief dyspnea episodes, low-productive cough, and minor lung wheezing in 15% of children. Compared to control children receiving only bromhexine, those receiving the combination showed earlier positive dynamics: by day 2–3, cough became wet, sputum cleared more easily, and by day 6–7, most symptoms resolved ($p < 0.05$).

The presence of a short-acting bronchodilator and complementary mucolytic, sedative, and anti-inflammatory components contributed to pronounced clinical efficacy, with cough resolution 3–4 days earlier than the control group ($p < 0.05$).

Symptom improvement was accompanied by better sleep, increased activity, and normalized emotional state. Total clinical symptom scores confirmed the statistically significant effectiveness of Ascoril Expectorant ($p < 0.05$).

Three children (4%) in the Ascoril group required antibiotics due to insufficient response, but the need was 2.5 times lower than in the control group (3 children, 10%; $p < 0.05$).

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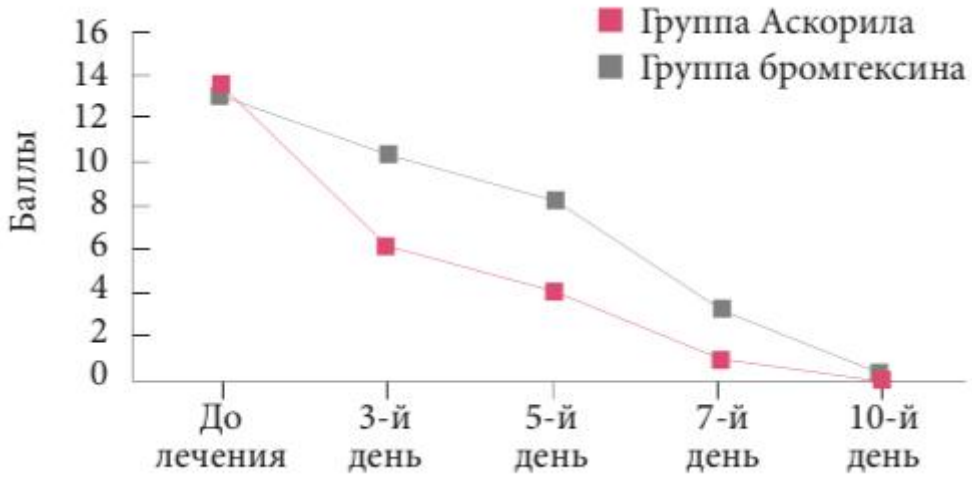

Clinical improvement correlated with bronchophony and spirometry normalization, indicating restored bronchial patency. One child experienced an allergic rash; no other adverse reactions were reported.

Thus, Ascoril Expectorant, through combined bronchodilator and mucolytic action, enhances mucociliary clearance, accelerates symptom regression, prevents severe prolonged disease, and reduces subsequent antibiotic use. Its clinical efficacy and high safety profile make it a drug of choice for children over 2 with mild to moderate bronchial obstruction during ARVI.

In emergencies with pronounced bronchial obstruction, short-acting bronchodilators form the pharmacological basis. They are essential for obstructive bronchitis, bronchiolitis, or asthma exacerbation. Pediatric use includes beta-2 agonists, anticholinergics, and methylxanthines.

Beta-2 agonists act via adrenergic receptor stimulation, activating adenylate cyclase, increasing cAMP, and stimulating the calcium pump, resulting in bronchial dilation. Inhalation provides rapid bronchodilation (3–5 minutes) and improves mucociliary transport. Delivery depends on age and severity: metered-dose inhalers (MDIs), MDIs with spacers, nebulizer solutions, or enteral administration.

Combination therapy with short-acting beta-2 agonists and anticholinergics is used for pediatric obstruction. According to GINA (2010) and the Russian National Program "Bronchial Asthma in Children" (2008), a fixed combination of fenoterol and ipratropium bromide (Berodual®) is first-line therapy for exacerbations in children from early age. Synergy allows lower beta-2 agonist dosing, reducing side effects. Nebulized Berodual dosing: neonates and infants – 1 drop/kg; 1–6 years – 10 drops; >6 years – 10–20 drops, 3–4 times/day. Observe the child 30–40 min post-inhalation.

For obstruction with severe respiratory distress, inhaled corticosteroids (budesonide) are indicated. Budesonide suspension (Pulmicort®) via nebulizer is well-studied. Studies show efficacy in children aged 3 months–18 years with varying obstruction severity. Pulmicort® has been used in acute care in Yekaterinburg and Nizhny Novgorod, normalizing conditions and avoiding hospitalization. Dose: 0.25–0.5 mg at home; 0.125–0.25 mg twice daily in

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exacerbations. Early combination therapy (Pulmicort + Berodual + ambroxol) provides faster improvement than separate inhalations.

Initial Pulmicort® dose for exacerbations or obstruction: 0.5–1 mg twice daily (≥6 months). Once-daily dosing also effective. Pulmicort® is the only nebulizer-registered inhaled corticosteroid for treating croup, dosed 1 mg twice 30 minutes apart.

If inhaled steroids are ineffective or not feasible, systemic steroids are used (oral/parenteral) for insufficient bronchodilator response, severe/life-threatening cases, or history of steroid use for exacerbation. Therapeutic effect lasts 8–12 hours, onset delayed 4–6 hours, dose: 1 mg/kg/day orally, course 3–5 days; abrupt cessation is allowed.

Since ARVIs can trigger asthma exacerbations, clinicians must know management algorithms. For mild exacerbation, begin short-acting beta-2 agonist inhalation with spacer, repeating every 20–30 minutes for an hour. Early-age children may use combined bronchodilator (beta-2 agonist + ipratropium) via nebulizer. If no improvement, reassess severity.

Moderate exacerbations require inhaled steroids (Pulmicort® suspension) with bronchodilators via nebulizer. Severe exacerbation warrants hospitalization; outpatient emergency care follows the above principles, with simultaneous bronchodilators, systemic steroids, and oxygen therapy.

In early childhood obstruction during ARVI, combining inhaled bronchodilator, mucolytic, and corticosteroid is effective. Mucokinetic agents may be added from day 2–3; ambroxol is preferred. Mineral water should not be used in nebulizers.

A comprehensive approach to ARVI-related obstruction, considering clinical situation and age, ensures high therapeutic effectiveness and improves patient quality of life. For mild obstruction with tenacious mucus, combined mucolytic + bronchodilator preparations like Ascoril provide bronchodilation, expectoration, shorter cough duration, productive cough, and enhanced mucociliary transport.

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