



EFFECTIVENESS OF THE DRUG L-MONTUS(MONTELUKAST) IN THE TREATMENT OF BRONCHIAL ASTHMA

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Abstract: Bronchial asthma is a chronic lung disease in which obstruction of the bronchi occurs. This in turn causes the development of symptoms such as coughing, shortness of breath, tightness in the chest and suffocation. In healthy bronchi, breathing and breathing allow air to pass through the respiratory tract unhindered. Due to the development of inflammation, swelling of the walls of the bronchi occurs, which thickens, and at the same time, this leads to a contraction of the muscle fibers of the bronchi with the development of bronchospasm. In the spasmolytic bronchi, mucus accumulates, which disrupts the free passage of air flow, resulting in a feeling of lack of air, shortness of breath and wheezing. Some points can be noted that play an important role in the development of this disease. The main ones are: genetic factor, environmental factors (contaminated air, tobacco smoke, house dust, pollen, viruses and other agents that penetrate the lungs with air flow, affecting the active zones-receptors located in different parts of the bronchi). Bronchial asthma is very common – studies in recent years show that 4-10% of the world's population suffers from this pathology at different levels. The percentage among children reaches 10-15. In addition, according to statistics, over the past 15 years, the number of people with this disease has doubled.

Key words: asthma, obstruction, concentration, allergen, inflammation of the bronchi, immunity, expiratory breathing.

The purpose of the study: in recent decades, the problem of asthma has become one of the most pressing issues in medicine for various reasons: increased incidence and severity of the disease, especially in children and adolescents; early disability; increased mortality. The basis of a suffocation or cough attack in which

bronchial asthma manifests itself is inflammation of the bronchi, often of an immune nature. If the causative agent of an allergen or infectious disease enters the bronchi, a complex immune mechanism is triggered-biologically active substances are released, special cells are activated. This leads

to a violation of the structure and function of the bronchi: swelling of the mucous membrane, spasms of smooth muscles, changes in the nature of bronchial secretion (it becomes thick and viscous, it is difficult to distinguish and close the bronchi secretion). Air entering the pulmonary alveoli during breathing is difficult for the bronchial constricted secretion to flow out. At the same time, a person breathes with difficulty – expiratory shortness of breath, a characteristic sign of asthma appears. Those around them note the whistling sounds that the patient makes. Depending on the factors underlying the development of the disease, two types of bronchial asthma are distinguished: allergic; non-allergic. The first, in turn, is divided into atopic or IgE-conditional and non-atopic (non-IgE). During this period of pathology, periods of lamination and remission are distinguished. There are also 4 levels of morbidity severity: intermittent (episodic), persistent (persistent) lung, medium and severe course. The severity of bronchial asthma is assessed on the basis of the following criteria: the number of nocturnal suffocation attacks per week; the number of suffocation attacks per day and one day per week; the need to apply short-term agonists during the day; the severity of physical activity and sleep disorders; changes in the indicators of external respiratory function during lamination and their absence during remission; part In some patients, the seasonality of the disease may be associated with the flowering period (ripening of seeds) of certain species of plants. Mechanisms of the development of the disease. The basis of the suffocation or coughing bark, in which bronchial asthma manifests itself, is inflammation of the bronchi, often of an immune nature. If the causative agent of an allergen or infectious disease enters the bronchi, a complex immune mechanism is triggered-biologically active substances are released, special cells are activated. This leads to a violation of the structure and function of the bronchi: swelling of the mucous membrane, spasms of smooth muscles, changes in the nature of bronchial secretion (it becomes thick and viscous, it is difficult to distinguish and

close the lumen of the bronchi). It is difficult for air entering the pulmonary alveoli during breathing to flow outward through the bronchial constricted Lumen. At the same time, a person breathes with difficulty – expiratory shortness of breath, a characteristic sign of asthma appears. Those around them note the whistling sounds that the patient makes.

Methods of examination: in the treatment of asthma, the drug L-montus is an R-enantiomer of Levocetirizine cetirizine, a group of competitive and selective antagonists of peripheral H1 receptors. Studies have shown that Levocetirizine's association with human H1 receptors is 2 times higher than that of cetirizine ($K_i \approx 3.2 \text{ nmol/L}$, $K_i \approx 6.3 \text{ nmol/L}$). Levocetirizine is released from H1 receptors with a Half-Life of 115 ± 38 minutes. Pharmacodynamic studies have shown that $\frac{1}{2}$ dose of Levocetirizine affected the skin and nose comparable to cetirizine. In vitro studies have shown that Levocetirizine slows the trans endothelial passage of eosinophils by exposure to endotoxine through the skin and lungs *rsatdi*. In Vivo pharmacodynamic experimental studies have identified three main characteristics-Levocetirizine 5 mg compared to placebo, VCAM-1 release in 14 adult patients in the first 6 hours of the pollen reaction, modulation of vascular permeability, and reduction of Eosinophilic accumulation. Montelukast is an antagonist of leukotriene receptors. Montelukast inhibits cysteinyl leukotriene receptors of the airway epithelium while exhibiting the ability to visualize bronchospasm as a result of cysteinyl leukotriene LTD_4 ingestion in patients with bronchial asthma. A dose of 5 mg is sufficient to stop LTD_4 -induced bronchospasm. The use of Montelukast 1 time per day in doses exceeding 10 mg does not increase the effectiveness of the drug. Montelukast induces bronchodilation Within 2 hours of oral administration and may complement bronchodilation caused by beta 2-adrenomimetics. Pharmacokinetics Levocetirizine is quickly and completely absorbed after oral administration. The peak plasma concentration is reached 0.9 hours after

dosing. The constant level of concentration is reached after 2 days. Concentration peaks are 270 ng/ml and 308 ng/ml, with a single and repeated dose of 5 mg once a day. Absorption rates are dose-dependent and do not change during feeding, but the peak concentration decreases. The distribution of levosetirizine is limited, with a distribution size of 0.4 l/kg. the half-life in adults is 7.9 ± 1.9 hours. the total clearance is 0.63 ml/min/kg. about 85.4 percent of the Taken dose of Levocetirizine and metabolites is excreted in the urine, and 12.9 percent in the feces. Levocetirizine is excreted using glomerular filtration and active tubule secretion. After oral administration, montelukast is quickly and almost completely absorbed from the gastrointestinal tract. The consumption of normal food does not affect the maximum concentration in blood plasma (C_{max}) and the bioavailability of the drug. In adults, when taking the drug on an empty stomach at a dose of 10 mg, C_{max} in blood plasma is achieved after 3 hours. Bioavailability is 64% when taken orally. Blood plasma protein binding is more than 99%. Distribution volume (V_d) average 8-11L. when taking the drug at a dose of 10 mg 1 time per day, an average (about 14%) accumulation of the active substance in the plasma is observed. Montelukast is actively metabolized in the liver. When administered at therapeutic doses, plasma concentrations of montelukast metabolites are not detected in equilibrium in adults and children. Cytochrome Z450 (3A4 and 2c9) isoenzymes are predicted to be involved in Montelukast metabolism, while at therapeutic concentrations montelukast does not inhibit cytochrome p4503a4, 2c9, 1A2, 2A6, 2c19 and 2d6 isoenzymes.

Half-Life (T_{1/2}) in young healthy adults is 2.7 to 5.5 hours, with montelukast clearance averaging 45 ml/min in healthy adults. after oral administration of montelukast, 86% is excreted in feces within 5 days and less than 0.2% is excreted in urine, which is almost exclusively excreted from 2.5 ml of montelukast and its metabolites. with bile.

Summary. indicators, the drug for nausea is prescribed to adults and children over 2 years of age for the prevention and treatment of chronic asthma, including the Prevention of day and night symptoms of the disease; prevention of bronchospasm caused by exercise. The drug is indicated for allergic rhinitis (seasonal allergic rhinitis in adults and children over 2 years of age, long-term allergic rhinitis in adults and children over 6 months of age), as well as for the relief of symptoms of Chronic Idiopathic Urticaria. The drug should be taken once a day. With asthma, the drug should be taken in the evening. With allergic rhinitis, the time of taking the drug is selected individually, depending on the favorable time of day for a particular patient. Patients with asthma and allergic rhinitis should take 10 mg of the drug once a night. Adults are prescribed 1 tablet a day. Special instructions it is not recommended to prescribe a drug for the treatment of acute attacks of bronchial asthma. In the acute period of bronchial asthma, patients should be prescribed medications for therapy to stop and prevent attacks of the disease. The dose of inhaled GCS (glucocorticosteroids) used at the same time as the drug can be gradually reduced under the supervision of a doctor. Inhaled or taken orally, GCS therapy should not be drastically replaced with the drug, while in patients treated with anti-asthma agents, including leukotriene receptor antagonists, the decrease in the systemic dose of gcs is rarely accompanied by eosinophilia, vascular rash, exacerbation of lung symptoms, cardiac complications and/or the appearance of neuropathy, sometimes diagnosed. Ostrich-systemic eosinophilic vasculitis. Although no causal relationship of these adverse events with leukotriene receptor antagonist therapy has been established, care must be taken to reduce the systemic dose of GCS in patients taking the drug and appropriate clinical follow-up.

Information indicating that it is an intake affects the ability of the drug to drive, or no moving mechanisms have been identified.

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