CHRONIC OBSTRUCTIVE PULMONARY DISEASE: THE INFLUENCE OF TOBACCO SMOKING ON SEVERAL LINKS OF PATHOGENESIS AND THE PLACE OF FORMOTEROL IN TREATMENT STRATEGY

Y. V. Prosvetov, S. S. Barinov, I. O. Shehunova, A. Y. Gusarova

Abstract

Tobacco smoking is a major risk factor of chronic obstructive pulmonary disease (COPD), alongside with the other influence of hazardous particles and gases, associated with air pollution and use of biomass fuel. Current review summarizes main influence factors of tobacco smoke on several links of COPD pathogenesis.

Higher level of oxidation stress (OS) due to higher concentration of oxidants contained in tobacco smoke is observed in tobacco smokers. Smoking causes an imbalance in oxidant-antioxidant system, which plays an important role in respiratory tract inflammation — a key characteristic of COPD, leading to remodeling of airways and accelerated lung aging. OS promotes binding of nuclear transcription factor (NF-kB) with DNA and expression of mRNA pro-inflammatory cytokines, such tumor necrosis factor (TNF)-α, TNF-β and interleukin (IL)-6 in type 2 alveolar epithelial cells and IL-8 in airways epithelial cells. Elevated serum IL-1β also found in smokers is considered an important aspect of chronic inflammation of airways. Particularly tobacco smoking causes greater elevation of serum IL-6 and IL-8, which are the indicators of systemic inflammation in COPD.

Tobacco smoke due to increased OS and modulation of epithelial-mesenchymal transition in cancer cells facilitate a progression of cancer.

Second part of the review is dedicated to characteristics of pharmacological effects of long acting beta-2 agonist (LABA) formoterol. More often use of formoterol among other LABA in COPD is determined by unique combination of its pharmacological properties: 1) fast start and long duration of effect; 2) effective for reversible or irreversible obstruction; 3) good safety, allowing its use in cardio-vascular diseases; 4) potentiation of long acting M-antagonist and inhaled corticosteroid effects; 5) lack of negative influence on short beta-2 agonist effect when used simultaneously; 6) no cumulation when used in therapeutical doses.

At the same time the authors present the data regarding the ability of formoterol to reduce intensity of OS, inflammation, level of epithelial-mesenchymal transition markers, caused by tobacco smoke, making it a valuable component of pathogenesis therapy of COPD.

Key words: COPD, тобacco smoking, inflammation, formoterol, therapy.

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Yuri V. Prosvetov
Zaporizhian State medical-pharmaceutical university
Professor of department of phthisiology and pulmonology
Doctor of medicine, professor
Tel.: +38 050 4846 453;
zp.pyuv@gmail.com