There is a steady increase of bronchial asthma (BA) in the world, including for child population. Today, according to WHO 300 million people worldwide suffer from BA, and by 2025 the WHO has predicted 600 million patients. In addition to inadequate treatment, the causes of inadequate asthma control include: environmental issues, the using of large amounts of chemicals in the home, often uncontrollable drug intake, severe asthma, which is often associated with severe inflammation; chronic exposure to sensitizing unaccounted factors and comorbidities. Particularly noteworthy are comorbidities, which are having common or similar etiological and pathogenetic factors, such as combination of BA and obesity.

Child obesity is a new global health challenge through its tendency to growth in this age group and health outcomes in later life. Both processes BA and obesity are chronic, complex and multifactorial in nature. Being overweight increases 2 times (and obesity increases 2.7 times) the risk of airway hyperresponsiveness, regardless of age or gender. The combination of BA and obesity creates a "vicious circle" in the course of these diseases. As noted Gina, 2015 materials, overweight status appears to increase asthma risk.

The combination of these two pathologies is determined by a number of scientists as a single phenotype or endofenotype of BA. The current of this phenotype of BA is more severe, it has more frequent exacerbations and reduced control of the disease. Some mechanisms by which obesity affects on BA are formulated and described now: systemic inflammation which is associated with obesity (increased levels of circulating cytokines such as IL-6, TNF; the impact of overweight on the mechanics of respiration, oxidative stress, hormones of obesity (leptin, Adiponectin, resistin), common genetic factors, the presence of comorbid conditions such as gastroesophageal reflux disease and sleep apnea.

Excess weight has a negative effect on the function of the respiratory system. The mechanical influence of overweight on the function of the lungs realized by direct pressure of a large mass of subcutaneous fat on the chest, anterior abdominal wall, excess fat deposition on the diaphragm.
Adipose tissue is now considered as an important endocrine organ, which produces number of adipokines with pro- and anti-inflammatory effect. In particular, adipose tissue is a source of proinflammatory mediators such as C-reactive protein, tumor necrosis factor-alpha, eotaxyn, leptin, a protein, transforming growth factor, interleukins 4, 5, 6, 13; plasminogen activator inhibitor-1; protein that stimulates acetylation; free fatty acids; angiotensinogen and others. The balance between pro-inflammatory (leptin, resistin) and antiinflammatory (adiponectin) adipokines plays an important role in combination of BA and obesity, as the imbalance of cytokine production towards proinflammatory may be a factor supporting systemic inflammation. Leptin has a systemic inflammatory effect, which may promote the development of BA. Increased blood leptin may be a marker of more severe obstruction and inflammation at all levels of respiratory tract in patients with BA and overweight. Adiponectin is a fat hormone, which has a positive influence on the metabolism of lipids and carbohydrates. It is an antiinflammatory cytokine. Adiponectin production level decreases in people with obesity and this decreasing causes endothelial damage and the development of systemic chronic inflammation, which complicates the course of BA in people with obesity, increasing its development.

The presence of both episodic bronchial obstruction in BA and constant violation of the external breathing caused by obesity, contribute to the deepening of hypoxia and increasing of the oxidative stress. There is a hypothesis that oxidative stress in obesity can be caused by hyperglycemia, hiperleptynemia, hyperlipidemia and the presence of chronic inflammation. This process is accompanied by increased production of reactive oxygen and it promotes the activity of lipid peroxidation and it causes reduced activity of anti-oxidant protection.

Obesity is also associated with underlying medical conditions such as gastroesophageal reflux disease syndrome and obstructive sleep apnea/hypopnea, each of them aggravates BA.
Another of pathogenic components of this combined states is genetic factor. Identified regions in the genome that may contain genes that lead to the formation of both pathological conditions: 5q23-32, 6p21-23, 11q13, and 12q13-24.

It is possible that one or more of the above simultaneously (or not yet reveal) mechanisms of pathogenesis common to BA and obesity can influence to increase the risk of BA.

The influence of weight loss on the course of asthma among adults was determined by a significant improvement in the respiratory tract on lung volumes, asthma symptoms, exercise tolerance due to weight reduction; and although the question of the impact of weight loss in obese children with BA has not been studied enough, we should expect similar improvements to quality of life.

Thus, the features of BA in children with overweight and obesity require a general understanding of the mechanisms underlying the pathogenesis of obesity and BA, and of course, there are major elements in the development of new therapeutic strategies.

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