PATHOGENETIC MECHANISM OF REALIZATION OF HARMFUL INFLUENCE OF VISCERAL OBESITY DURING BRONCHIAL ASTHMA

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The goal of the research was the comparative study of the level of inflammation markers during the isolated cases of bronchial asthma (BA) and when accompanied by visceral obesity (VO).

Materials and methods of investigation. The diagnostics of VO was conducted according to the recommendations of WHO. The investigation of inflammation markers was conducted for 71 patients with BA, among which: Group 1 included 35 BA patients with normal body weight, and Group2 had 36 VO patients. The Control Group consisted of 22 practically healthy people. The level of interleikins (IL) IL-6, IL-8 and the tumor necrosis factors-α (TNF-α) in blood serum was determined using immunofermented tverdofaznym method, the antigenic composition of the membrane of mononuclears in peripheral blood was determined using indirect immunoflorescent method, the level of α1-inhibitor proteinase (α1-IP) and α2-macroglobullin (α2-MG) was determined using spectrofotometric method. The achieved results were analyzed using the methods of variance statistics. The correctness of the difference in the average measurements was evaluated using Student's t-criterion.

Results of investigation. Group 1 patients had 3x and Group 2 patients 8x the level of FNP-α, and IL-6 level was elevated by 2,5 and 5,6, respectively. The level of IL-6 in blood serum was abnormal for both patient groups compared to the Controls, while its level for VO patients was evidently higher compared to Group 1 patients ($p<0,05$).

Group 2 patients had levels of CD3, CD4, CD8 and CD16 evidently lower compared to Group 1 patients ($p<0,05$). The levels of adhesive molecules (CD11b, CD54), expression of receptor to IgE (CD23), activation markers of lymphocytes (receptors to IL-2, HLA-DR) were evidently higher for patients of both groups compared to the Control, and higher for Group 2 patients compared to Group 1. It was observed that α1-IP and α2-MG were elevated by 2x and 2,6x respectively compared to healthy individuals.

Results of investigation convey that the presence of VO in BA patients leads to: hyperproduction of proinflammation cytokines (IL-6, IL-8, TNF-α); increase of T-lymphopenia, deficiency of cytotoxic cells, increased expression of activation markers, adhesive molecules, receptors to IgE, HLA-DR; increased level of α1-IP and α2-MG proteins.

These changes, along with known negative impacts of obesity on the process of BA (bronchial hyperactivity, increased frequency of hastroesophageal reflux, aggravation of pulmonary deficiency), represent one more pathogenetic mechanism – the exacerbation of inflammatory process.