COPD AS METABOLIC PATHOLOGY: PROBLEM OF ENDOTOXICITIES AND NEUROTOXICITY

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Chronic obstructive pulmonary disease (COPD) is an often severely disabling chronic lung disease with a high prevalence of over 250 million cases worldwide. Currently, according to a global study by the World Health Organization, chronic obstructive pulmonary disease is the third leading cause of death and deteriorating health in the world [1 – 3].

Therefore, the aim of the presented work was to study the relationship between the development of oxidative stress, endotoxicities and neurotoxicity as a manifestation of a violation of the general metabolic state in patients with COPD.

This work is based on the results of examination and treatment of 269 patients due to bronchopulmonary diseases hospitalized in the Dnipro’ Municipal Hospital during the period 2018–2019. The COPD cohort included patients (men and women) older than 40 years who had been diagnosed with COPD (ICD-10 codes J42, J43, J44) (n = 126), asthma (J45, J46) (n = 96) and pneumonia (J12–J17) (n = 47). The control group consisted of 50 healthy volunteers.

The was evaluated the neutral cell adhesion molecule (N-CAM) as a convenient neurospecific marker for the study of neurotoxic effects. N-CAM level in serum was determined by competitive enzyme-linked immunosorbent assay ELISA using monospecific rabbit antisera against all three N-CAM polypeptides to study the possible neurotoxic effects in bronchopulmonary pathology development [4].

The statistic analysis was performed by the Statistica 10.0 program. Differences between groups were analyzed using Student’s t-test and P-values <0.05 were considered statistically significant. The results are presented as the median and interquartile range in the form of the 25th and 75th percentiles.

The pathogenesis of COPD involves several pathogenetic processes including oxidative stress, inflammation, protease/antiprotease imbalance, apoptosis, cellular senescence, endogenous intoxication, neurotoxic affects, however, the relative contribution of each of these pathologies to COPD varies among patients.

Studies of neurospecific marker proteins of cellular and subcellular elements of nervous tissue in pathologies associated with disorders of the central nervous system (CNS) are quite relevant, because in many cases they are due to changes in permeability of the blood-brain barrier (HEB) and yield neurospecific proteins in the
bloodstream. N-CAM is an integral membrane glycoprotein localized on the outer side of the neuronal membrane. Bronchopulmonary pathology development leads to the accumulation of N-CAM in the serum, which depended on the disease nosology. Thus, the maximum concentration of N-CAM was detected in the serum of patients with COPD (mean level was 8.96 μg / g of total protein to 3.22 μg / g of total protein in the control group), then patients with non obstructive bronchitis (8.03 μg / g of total protein), in patients with acute pneumonia, the average level of N-CAM was 6.40 μg / g of total protein, bronchial asthma, 5.675 μg / g of total protein, respectively.

N-CAM is expressed by neurons and involved in controlling the processes of neurite outgrowth and synaptogenesis, plasticity of the nervous system, the formation of a neural network during development [5]. The presence of N-CAM in the serum of COPD patients indicates a violation of the resistance of the blood-brain barrier and the release of neurospecific proteins into the bloodstream, which under certain conditions can cause autoimmune aggression, changes in the signaling system and distortion of the cellular response to certain extreme stimuli.

References: