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INFLAMMATION OF RESPIRATORY SYSTEM AND ACQUIRED PROTEOLYTIC ENZYME INHIBITORS DEFICIENCY DEVELOPMENT

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Background. Proteolysis is one of the most universal mechanisms in the organism taking part in regulation of various processes. Role of proteinases in the lungs pathology begin to study when almost 50 years ago C.Laurell & S.Eriksson (1963) published findings about possible development of inherited genetically determined α-1-antitrypsin (α-1- proteinase inhibitor) deficiency for the first time. It started intensive study of the role of proteinases and their inhibitors in development of lung pathology and many other pathological conditions. In lungs pathology, participation of proteinases in processes of alteration at the level of bronchus and alveoli are most important. The aim of our investigation was to study general mechanisms of changes in nonspecific proteinases and their inhibitors in inflammatory lung pathology and to find out whether acquired proteinase inhibitor deficiency is possible and what typical reactions in the proteinases and their inhibitors may development.

Designe. Systemic and local changes in proteinase-inhibitor system in inflammatory lung pathology were studied experimentally and clinically. Acute (1-14 days) and chronic (1-6 months) pneumonia was modeled on 150 Wistar rats. Bronchoalveolar lavage (BAL) was received through washing the lungs with saline. Clinical investigations were performed on 120 patients with acute and chronic inflammatory processes in the lungs. BAL was received during bronchoscopy. The elastase-like (ELA) and trypsine-like (TLA) activities and level of acid-stable inhibitors (ASI) and acid-nonstable antitrypsine activity (ATA) in BAL were measured.

Results. It is found out that changes in proteinase-inhibitor system are of regular character irrespective of genetic inheritance of the form of proteinase inhibitor. In modeling the inflammatory process in the lungs, at acute stages there was increase in the level of proteinases and inhibitors both locally (in bronchoalveolar lavage) and systemically (in blood). In development of chronic inflammation in the lungs, elevated level of proteinases and their inhibitors in blood was preserved. Locally, inhibitor deficiency parallel with higher activity of proteinases, first of all, elastase in BAL it was determined. It was proved by both experimental and clinical investigations. Furthermore, locally, in BAL we defined four types of acquired proteinase-inhibitor imbalance. In all the types there was increase in proteinase activity of various degree but reaction in inhibitor condition was different. Compensated type characterized by moderate increase in TLA и ELA and marked increase in ATA и ASI; potentiated type - diminished ATA and growing ASI; destructive type - prevailing growth in ELA and decrease in ASI; decompensated type - marked growth in proteinases and diminished ATA и ASI.

Conclusion. The investigations made allowed to conclude that development of chronic inflammation in the lungs causes local acquired proteinase inhibitor deficiency. We described four types of reactions in proteinase-inhibitor system on the level of bronchus and alveoli (compensated, potentiated, destructive and decompensated). Using of this types allow more exact diagnosis and pathogenic correction of the conditions accompanying by proteolytic aggression.

Key words: proteinases, proteinase inhibitors, deficiency, lungs, inflammation