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Title: The role of epithelial-mesenchymal transition in the possibility of chronic process in the lungs during influenza pneumonia, caused by the virus AH1N1

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Body: Despite numerous influenza pandemics and morphological studies of influenza pneumonia, the question of the possible mechanisms of chronisity of the process has not been studied. It is known that in the process of chronic inflammation and sclerosis of various organs plays an important role of epithelial-mesenchymal transition. The aim of our study - to test this hypothesis in the lungs. Studied 30 sectional cases of influenza pneumonia using immunohistochemistry studies: evaluated the expression of cytokeratin and AE1/AE3 pantcytokeratina 18, vimentin, alpha-smooth muscle actin (monoclonal antibody, imaging system En Vision, Daco). The material was divided in 2 groups based on dominant lokalization. First group included 20 cases with significant damage of alveolar epithelium (necrosis, apoptosis, desquamation) and intaalveolar exudate (fibrinous, hemorrhagic, suppurative and often mixed). The second group included 10 cases with predominant changes of interalveolar septae (thickening due to edema, infiltration by inflammatory cells, stromal proliferation with transition fibrosis into myofibroblasts, accumulation of intercelular matrix. In only half of the cases in first group showed features of proliferation of alveolar epithelium in the form of small groups and sometimes numerous type 2 pneumocytes, which are more resistent to damaging factors, capable to division and differentiation toward type 1 pneumocytes and producing surfactant. All cases of the second group were found significant hyperplasia of type 2 pneumocytes. The results of our study allow to suggest that this mechanism may underlie the chronic influenza pneumonia.