Proteolytic enzymes in the pathogenesis of the influenza virus

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Introduction:

The interaction of the virus and the cell is a single whole, which is a combination of the internal relations of two opposite principles.The final result of this interaction is genetically determined by both the host and the pathogen and depends on the regulation of both the process of viral reproduction and the body's defenses.The balance between these processes determines the outcome of the interaction, the result of which may be the death of the owner, his full recovery or the formation of a chronic form of infection Deproteinization of a flu virus is necessary for its penetration into a cell and this occurs for the account of trypsin-like proteinases of the host's cell (1,3,7,8). We assumed that this enzyme has an important role in morphogenesis of flu virus and considerably defines its pathogenic and virulent properties. (5,6)

Keywords :*virus grippa, tripsino-Like proteinase, ingibitor*

Objective:

To examine changes in proteinase and inhibitory behavior in the development of white mice influenza infection previously infected with flu A virus.

Methods:

We worked with A / PR/8/34 (H1N1) flu virus and measuring 7-8 g and 16-17 g white mice. For animal infection we took a dose of 2,5-2 LD 50 virus. Such a dosage caused the animals to die 100 per cent for the 6th day after infection. The animals were slaughtered and took lungs. Blood was taken in 15, 30 min., 1, 6 hours and further in 1, 2, 3, 4, 5, 6 days after infection. Animals who were not infected were slaughtered in turn and their lungs were removed according to the same terms.We defined infectious, hemagglutinating, proteinase and activity inhibiting and total protein in the homogeneous and blood serum of the lungs.Protease activity was determined by the method of K.M. Veremeenko (2). The method is based on the reaction to arginine, formed during the hydrolysis of protamine and histones and not precipitated with 20% trichloroacetic acid.**The determination of a proteinase inhibitor in the homogenate of the lungs and in blood serum was carried out by the casein method (4Results**. It has been is established that the level of trypsin-like proteinase and its inhibitor in the lungs and blood serum of not infected white mice were in balance at rather high level and did not change considerably during the whole period of supervision (6 days).Proteinase activity depended on the age and weight of the animals. In 16-17 gram mice, the amount of proteinase was higher than in 7-9 gram mice, especially high proteinase activity was determined in lactating females.At infection of white mice with flu virus A/PR/8/34 (H1N1) there was a breach of proteinase-inhibitory balance In the infectious cycle, three cycles can be distinguished, which are characterized by differing degrees of virus replication, proteinase activity level and inhibitory activity level. The most profound changes happened during the first hours after infection.. 6 hours after infection, the amount of proteinase in both the lungs and in the blood serum of infected animals decreases and the proteinase inhibitory activity. Thus, it can be assumed that a decrease in proteolytic activity is associated with a corresponding accumulation of a proteinase inhibitor. Apparently, infected cells of the influenza virus induce the accumulation of an inhibitor in both lung tissue and serum.Can be assumed that a decrease in proteolytic activity is associated with a corresponding accumulation

of a proteinase inhibitor.During the period of the greatest accumulation of the infectious virus (2 days after infection), proteolytic activity also decreased, but this decrease was not accompanied by an increase in inhibitory activity. This suggested that a second decrease was not accompanied by an increase in inhibitory activity. This suggests that the second decline is due to other reasons. Perhaps one of the reasons is the use of trypsin-like proteinases for proteolytic activation of the virus in the lungs of infected mice.The third period of the accumulation of protease activity coincides with an increase in the infectious offspring of the virus and, apparently, is associated with the consequences of the viral infection and the layering of the bacterial infection. So, it was shown that staphylococcal protease causes proteolytic activation of the influenza virus (9).

Conclusions:

Increasing activity of proteinase during the first hours of infection has contributed to increased activity of infectious and hemagglutinating.
Increasing inhibitory activity within 5-6 days of infection results in some arrest of influenzal infection.

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