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Flu pathogenesis proteolytic theory and its role in the improvement of flu's treatment

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Introduction: Interaction of virus and cell in the pathogenesis of viral diseases is insufficiently studied. The main point here is penetration of virus into a healthy cell with an obligatory virus' deproteinization. However the deproteinization of viruses is studied insufficiently. First of all it refers to the mechanisms of introduction of flu virus in the cells of mammals, including humans. In this regard in 1983 we offered the new theory of flu pathogenesis with participation of proteinase-inhibitory system.

Objective: To study the state and role antiproteinase systems of the virus and recipient in the development of an influenza infection for receiving essentially new medical preparations on the basis of inhibitors of trypsin-like proteinases.

Methods: In work presented we used flu viruses, A/PR/8/34(H1N1), AO/32(H1N1) strains, white mice, chicken embryos, white rats, waste of γ-globulin and albumin manufacturing, human interferon and immunoglobulin, herpetic, gonococcus and tularemia vaccines and medicines: Influvac, Fluarix, Vaxigrip-anti-influenzal vaccines, Avaxim-vaccine for hepatitis A and blood preparations-Fraxiparine, Solcoseryl.

Results: It has been established that cleaning and concentration of influenza virus A various by different methods does not exempt virus from cellular enzymes trypsin-like proteinases and their inhibitors. Both domestic (human immunoglobulin and interferon, anti-influenza and herpetic vaccines) and foreign preparations (Influvac, Fluarix, Vaxigrip, Avaxim, Fraxiparine and Solcoseryl) had trypsin-like proteinase and its inhibitor in their structure. In the experiments on the white mice at infection with flu A virus there was a violation of proteinase-inhibitory balance, especially during the first hours after contamination. From the lungs of healthy mice six isoforms of trypsin-like proteinase have been allocated and antiproteinase immune serums were received to them. At the treatment of the animals infected with a lethal dose of flu A virus, only one serum (to the third isoform) has protected white mice from death. From the waste of γ-globulin manufacture of donor blood, inhibitor of trypsin-like proteinases which protected for 80% of white mice from death was emitted.

Conclusions: Endogenous inhibitors of human blood proteinases are perspective preparations in the fight with flu in humans.

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Recombinant HBV vaccine enhances the rate of sustained virological response when initiated early after anti-HCV combination therapy

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The overall SVR rate for chronic hepatitis C genotype 4 using the standard of care is 54.3%. HBV infection can be prevented by the administration of effective and safe vaccine. Evaluation of the vaccination-induced anti-HBs response rates in a cohort of HCV Egyptian patients after being exposed to antiviral combination therapy and the magnitude of its effect on the rate of SVR through its putative role in induction of crossed immunity. 500 HCV patients who had completed the course of antiviral therapy and achieved end of treatment response (ETR) were retrospectively analyzed and received 20 µg of recombinant DNA vaccine for hepatitis B at time intervals (0, 1, 4 months). The first dose of the vaccine was initiated one month post treatment. Laboratory analysis included routine preliminary investigations to anti viral therapy and specific investigations as determination of anti-HBs antibodies 2 months following the third dose of vaccine. 433 patients showed protective response (86.6%), 67 patients were non responders (13.4%) (p=0.003). Adding HBV vaccine 1 month post treatment increased SVR (400 patients, 80%) (Chi-square=40.3, p=0.000). Diabetes affect response to HBV vaccine (p=0.0001). Adding HBV vaccine to the post treatment care of patients with HCV after termination of antiviral therapy gain two benefits; protection from HBV and significant increase in rates of SVR.

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