HIV-HCV co-infection: epidemiology, pathogenesis and therapeutic implications.

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Abstract

Hepatitis C virus (HCV) is the cause of more than three-quarters of liver-related deaths in HIV-seropositive individuals and it is remarkable that today approximately one-quarter of HIV-infected individuals in Europe and the USA have a HCV coinfection. HIV/HCV coinfected patients were more likely to develop cirrhosis, had an increased risk of developing AIDS, of HIV-related disease and of overall mortality. How HCV may affect the course of HIV infection is not well known even if it was suggested that HCV co-infection is able to increase immune activation and to sensitize CD4+ T-cells towards apoptosis in the absence of HIV therapy. There are many evidences that the simultaneous presence of HIV infection accelerates the liver damage from HCV favouring the evolution to cirrhosis in co-infected patients. HIV increasing of TNF alpha liver production and of HCV replication in peripheral blood lymphomonocytes are the mechanisms at the basis of this phenomenon. HAART had a positive effect on HIV/HCV co-infection, otherwise it does not appear to fully correct the adverse effect of HIV infection on HCV-related outcomes. Traditional treatment with pegilated Interferon plus ribavirin have low rates of sustained virological response in co-infected patients especially if infected with HCV genotype 1, and better results were often obtained in patients in which the use of antiretroviral treatment was avoided to reduce the occurrence of adverse effects. The recent preliminary results on the use of anti-HCV protease inhibitor drugs, boceprevir and telaprevir, in co-infected people seems to demonstrate an enhanced antiviral efficacy in the HIV/HCV co-infected population of triple anti-HCV treatment even is some important limitation as interactions with antiretroviral agents and selection of HCV drug resistance, lead to consider the need for further studies designed to assess the best therapeutic strategies.

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