Journal of the International AIDS Society

Oral presentation

Open Access

O231 Cryptogenetic liver disease, steatosis, portal hypertension, transaminitis and antiretrovirals

S Pol

Address: Hôpital Cochin, Paris, France
from Ninth International Congress on Drug Therapy in HIV Infection
Glasgow, UK. 9–13 November 2008

Published: 10 November 2008

Journal of the International AIDS Society 2008, **11**(Suppl 1):O24 doi:10.1186/1758-2652-11-S1-O24

This abstract is available from: http://www.jiasociety.org/content/11/S1/O24

The decline in the mortality specifically related to the Human Immunodeficiency Virus (HIV) has revealed a growing number of liver diseases, which are now one of the leading causes of death among HIV-infected patients treated with antiretroviral combinations. Liver diseases in HIV-infected patients are usually secondary to hepatotoxicity of antiretroviral treatments, metabolic syndrome and lipodystrophy which may be associated with antiretroviral treatments, excessive alcohol consumption and co-infection by one or several hepatotropic viruses. One of the difficulties in the understanding of the liver enzymes abnormalities is the potential intricacy of all the causes: steatosis may be associated with hepatitis C infection, especially genotype 3; alcohol chronic consumption is frequently associated with HCV/HIV co-infection after an history of intravenous drug use; hepatotoxicity of antiretroviral drugs is clearly increased by underlying liver disease whatever its cause, enhancing the direct toxicity of a given drug or its metabolites. Recently, several cases of unexplained liver disease in patients with a long history of HIV infection, undetectable viral load and adequate immune restoration have been reported. Nodular regenerative hyperplasia, defined by a diffuse transformation of normal hepatic parenchyma into small, regenerative nodules with little to no fibrosis, seems to account for an unknown proportion of these cases. The mechanism of HIV-associated nodular regenerative hyperplasia is, to date, not known but does not seem to be related to HIV-infection and acquired immunodepression per se since the majority of patients are adequately immune-restored under combined antiretroviral therapy. The obliteration of the small portal veins results in ischemia of the supplied acini and regenerative hyperplasia of the remainders in order to maintain liver cell mass. We have reported that the occurrence of anti-Protein S IgG antibodies in HIV-infected patients leads to protein S deficiency that results with time in a progressive obliteration of the small portal venules of the liver, leading to obliterative venopathy and compensatory nodular regenerative hyperplasia. The causative role of antiretroviral drugs and especially didanosine, is difficult to assess. In summary, there are several causes of liver abnormalities in HIV-infected patients which require first to be recognised, second to be clearly analyzed and third treated when possible. The initial liver evaluation of any HIV-infected patient appears to be necessary to better understand the future occurrence of liver abnormalities. The place of non-invasive markers of fibrosis in this setting remains debatable.