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Poster presentation

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Unexplained severe portal hypertension in HIV-infected patients: a new clinical entity?

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Background

Cases of non-cirrhotic portal hypertension have been reported in HIV-negative patients as a result from exposure to adenosine analogues (e.g. azathioprine), bacterial infections, trace metals and chemicals, genetic coagulation disorders and/or autoimmune diseases. More recently, reports of similar cases in HIV-positive individuals have attracted much attention.

Methods

Description of clinical and histological findings of all consecutive cases of SPH of unknown etiology seen in HIV patients in three outpatient clinics in Spain.

Summary of results

A total of 13 (31.7%) out of 41 patients with unknown liver disease were diagnosed with SPH. All had elevated ALT for longer than 12 months and were followed for at least 4 years in three HIV outpatient clinics in Spain and in Italy.

Summary of results

The majority (85%) were males and homosexuals (77%), with a median age of 50 years. The median time since HIV diagnosis was over 7.5 years. All but one were on antiretroviral therapy and had undetectable plasma HIV-RNA. None had current CD4 counts >200/mmc, although four of them had CD4 nadir <cells/mmc any time in the past. Median time of exposure to antiretroviral drugs in the 12 individuals on HAART was 50.5 months for didanosine, 21 months for stavudine and 18 months for nevirapine.

During follow-up, eight patients (61%) experienced complications of SPH, including six upper gastrointestinal bleeding from oesophageal varices, and five portal thrombosis. A liver biopsy was performed in all subjects; none showed advanced hepatic fibrosis. Main features were as follows: nodular regenerative hyperplasia (31%), perisinusoidal fibrosis (8%), drug-induced hepatitis (8%), NASH (31%), and unspecific lesions (22%).

Conclusion

A small subset of HIV patients may develop SPH in the absence of known predisposing conditions and advanced hepatic fibrosis. These patients may eventually experience potentially fatal GI bleeding. Exposure to didanosine seems to be involved in most cases. A primary injury of the portal vessels by this adenosine analog may play a central pathogenic role in this condition.