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A 4-year follow-up of a cohort of HIV-positive cirrhotic patients

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Background

As mortality of HIV+ patients has dramatically declined following the introduction of HAART, chronic liver disease has become one of the leading causes of death in this population. While HIV infection worsens the progression of liver fibrosis in HIV/HCV co-infected persons, the balance between the benefits of antiretrovirals and the deleterious consequences of antiretroviral-associated liver toxicity are still controversial.

Methods

A cohort of HIV cirrhotic patients was followed-up from October 2004 to June 2008 at one large HIV clinic. Asymptomatic cirrhotic patients were identified using transient elastometry (FibroScan). Median elastometric values >14 Kpascals were considered for liver cirrhosis, as previously showing 97% correlation with liver biopsy.

Summary of results

A total of 178 HIV+ cirrhotic patients were identified. Main baseline characteristics: mean age 45 years, 72% male, IDUs 81%, 80% under HAART. Etiology of liver cirrhosis: 87% chronic hepatitis C, 23% alcohol abuse, 10% chronic hepatitis B, 4% cryptogenic hepatitis, 3% hepatitis delta, and 7% multiple viral hepatitis. During a median follow-up of 4 years, 26 (15%) patients were lost to follow-up, 11 (7%) died (eight from liver decompensation, two from liver cancer, and three from other causes). Moderate to severe hepatic dysfunction (Child B or C) was diagnosed in 13% of patients at the end of follow-up. Mean liver stiffness was 25 kPa at baseline and 28 kPa at the end of follow-up. Mean aminotransferase levels (AST/ALT) decreased from 76/74 to 64/62 IU/L. After successful therapy, six patients cleared HCV.

Conclusion

Liver cirrhosis in HIV+ patients under HAART does not seem to progress as fast as expected without antiretroviral therapy. This observation may be due to multiple factors, such as appropriate prevention of variceal bleeding and liver cancer following diagnosis of compensated cirrhosis using FibroScan, successful treatment of chronic hepatitis C, and selection of less hepatotoxic drugs in this population.