Managing HCV/HIV coinfection

Sir,

Some 20–25% of all human immunodeficiency virus (HIV) cases in the western world are estimated to be coinfected with hepatitis C virus (HCV). There are multiple causes of liver damage in a case of HCV–HIV coinfection. For example, heavy alcohol use, nonalcoholic steatohepatitis, pre-existing viral hepatitis (B or D), HIV-related opportunistic infections (e.g., Mycobacterium avium complex), drug-induced hepatotoxicity and AIDS-related cholangiopathy in patients with CD4 cell counts <100 cells/mm³. It appears that HIV-associated immunosuppression stimulates HCV replication and also impairs spontaneous immune-mediated HCV clearance. Thus, the risk of cirrhosis, end-stage liver disease and hepatocellular carcinoma is higher in coinfected cases.

Because of the high prevalence of coinfection, it is recommended to investigate for HCV infection in all HIV-infected cases and vice versa. The investigative work-up for HCV in known HIV cases should proceed as otherwise, i.e. serology (anti-HCV detection by 3rd generation enzyme immunoassay or enzyme-linked immunosorbent assay) followed by reverse transcriptase polymerase chain reaction (PCR) to detect circulating HCV RNA. Because of immunosuppression, the initial serologic test may be falsely negative. The Canadian Consensus Guidelines, 2007[1] thus recommend performing a qualitative HCV RNA assay in all HIV cases suspected of being coinfected with HCV even if the initial anti-HCV test is negative.

Liver biopsy may be considered in patients who elect not to be treated with antiviral therapy for any reason or those with persistently normal aminotransferase levels.[2] If liver biopsy shows only a minimal fibrosis limited to the portal tract (Metavir score <2 or Ishak score <3), initiation of interferon therapy can be delayed (in genotype 2 and 3 cases), or altogether avoided (in genotype 1 cases).

HIV cases with a relatively high CD4 cell count (>350 cells/mm³) are usually considered appropriate candidates for HCV therapy; highly active antiretroviral therapy (HAART) for HIV infection can be deferred in such cases. Conversely, HAART should be given first in all cases with a low CD4 cell counts (<200 cells/mm³); HCV therapy can be deferred in such cases till HIV suppression is achieved.

In HCV/HIV coinfected cases, peginterferon–ribavirin combination therapy for an extended period of 48 weeks is associated with better sustained virological response (SVR) rates and lower relapse rates in both genotypes 1 and 2 and 3 cases.[3] All HIV/HCV-coinfected cases should be subjected to a repeat quantitative PCR for HCV RNA at week 12 as the likelihood of attainment of SVR is best assessed by attainment of an early virologic response (EVR) at this stage.[4] Higher weight-based doses of ribavirin (1,000–1,200 mg/day) are more efficacious than the lower fixed doses of 800 mg/day for the treatment of genotype 1 hepatitis C cases.

Studies have shown that erythropoietin (EPO) can help avoid dose reductions in cases of ribavirin-induced hemolytic anemia. Possible indications of EPO include a fall in the hemoglobin (Hb) level by >4 g/dl, Hb levels of <8 g/dl and patients developing symptoms and signs of anemia (palpitations, dyspnea, easy fatigability, pallor).[5] Every effort should be made to avoid potentially dose-limiting complications, such as hepatotoxicity, mitochondrial toxicity, anemia, neutropenia and depression.

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Sir,

As known, the liver is a major part of the reticuloendothelial system and is a site of HIV replication and organ for many opportunistic infections. In HIV infected individuals, abnormal biochemical test results can develop as a result of hepatic parenchymal disease. The transaminases (alanine aminotransferase and aspartate aminotransferase) are enzymes made in liver. When liver cells are damaged these enzymes leak into bloodstream. Serum transaminase levels (AST and ALT) were studied for 40 HIV positive and 40 healthy and HIV negative control cases (from Government Medical College and Hospital, Aurangabad, Maharashtra, India) with mean age of approximately 35 years. Hepatomegaly cases were excluded. The mean serum AST and ALT in control group was demonstrated to be 22.15 ± 2.67 IU/L and 17.85 ± 1.84 IU/L, which was found to be increased up to 95.85 ± 26.9 IU/L (p < 0.001) and 85.67 ± 28.56 IU/L (p < 0.001) in HIV positive patients. The increase was found to be statistically highly significant. Increased levels were demonstrated in HIV patients by Vazmediano et al and Von Appen et al. [2,3] Poles et al. stated that up to 90% of patients with AIDS had abnormalities of the liver-associated enzymes. [1] It could be concluded that the liver function tests are deranged in HIV positive patients as compared to control. The deranged serum AST and ALT levels may identify patients requiring further investigations, thus can be used as a diagnostic and prognostic tool.

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