

in certain patients. Because of this error, an increase in the albumin-to-creatinine ratio was not reported for some patients. Table 3, which has been corrected at NEJM.org, now has slightly smaller sample sizes than those shown in the original version of the table. Patients who had only one follow-up visit were included in the original table. By definition, these patients could not have had progression, since an elevated albumin-to-creatinine ratio for 2 successive years was required to show progression.

Both progression from normal to either microalbuminuria or macroalbuminuria ($P=0.03$) and progression from either normal or microalbuminuria to macroalbuminuria ($P=0.04$) favor intensive treatment. Any progression of albuminuria is now highly statistically significant ($P<0.01$); 13.8% of the patients in the standard-therapy group, as compared with 9.1% of patients in the intensive-therapy group, had worsening albuminuria.

We appreciate the opportunity to update our

results regarding nephropathy, but we regret any confusion this may cause. The rest of Table 3 remains unaffected. As a consequence of these changes in Table 3, parts of the Abstract, Results, and Discussion are affected (see the correction notice in this issue of the *Journal*), but the overall conclusions of the trial are similar. We regret these errors.

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Persistent Carriage of Hepatitis E Virus in Patients with HIV Infection

TO THE EDITOR: Hepatitis E virus is an emerging infection in developed countries, with many clinical manifestations.¹ We describe a 48-year-old bisexual white male who was infected with human immunodeficiency virus type 1 (HIV-1) and who had elevated liver enzymes and infection with hepatitis E virus for at least 24 months. He had received a diagnosis of HIV infection in 2001 and was treated for tuberculosis in 2003. He had a 20-year history of excessive alcohol consumption (30 to 40 units [8 g of pure alcohol] per week) but reported never having injected drugs or received blood products. In January 2007, antiretroviral therapy was begun; the patient had a CD4 cell count of 30 per cubic millimeter and an HIV viral load of 8.3×10^4 copies per milliliter. Other medications started were valganciclovir, co-trimoxazole, oxycodone, and diazepam. The alanine aminotransferase level was elevated (to 51 IU per liter), and the alkaline phosphatase and bilirubin levels were normal. The results of serologic testing for hepatitis A infection were consistent with a past infection; serologic tests for hepatitis B and C were negative, as were polymerase-chain-reaction (PCR) assays for hepatitis B and C and cytomegalovirus.

Liver ultrasonography showed no evidence of parenchymal liver disease.

From March 2007 to January 2009, the patient's CD4 count remained under 200 cells per cubic millimeter, despite suppression of HIV replication starting in June 2007, and the alanine aminotransferase level remained abnormal, though he had reduced his alcohol consumption to 2 to 3 units weekly. Hepatitis E viral RNA was detected in serum and feces specimens, by means of PCR assay, in February 2009. Retrospective testing revealed the presence of hepatitis E virus in serum and feces samples that had been obtained during the preceding 18 months. Tests for hepatitis E virus were performed with the use of hepatitis E virus IgG and IgM enzyme immunoassay kits from two manufacturers: Beijing Wantai Biological Pharmacy Enterprise and Biokit. Wantai and Biokit test results were both positive for serum samples collected later during the 18-month period, whereas for samples collected earlier in the period, the Biokit results were negative but the Wantai tests were positive (Fig. 1). Sequencing showed the hepatitis E virus to be genotype 3, which is predominant in developed countries.¹ Liver biopsy

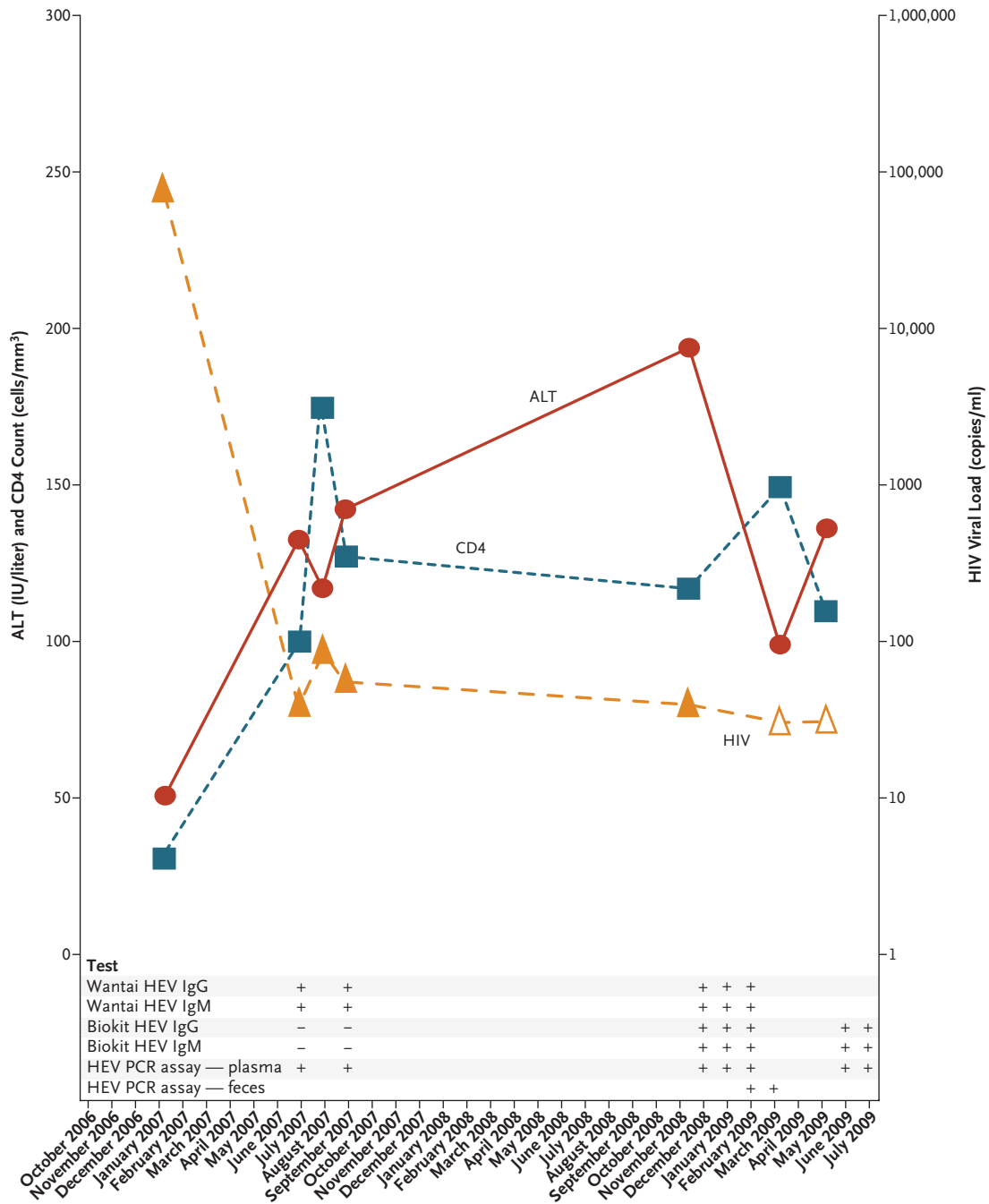


Figure 1. Laboratory Data for a Patient with Coinfection with Human Immunodeficiency Virus (HIV) and Hepatitis E Virus (HEV). For the HIV viral load, the final two data points (shown as open triangles) represent plasma samples with undetectable levels (<40 copies per milliliter). For the serologic and polymerase-chain-reaction (PCR) tests, the plus signs indicate positive results, and the minus signs negative results. ALT denotes alanine aminotransferase.

performed in March 2009 revealed established cirrhosis with active inflammation.

Until recently, hepatitis E virus was considered to cause acute infection only.¹ Chronic infection with hepatitis E virus, along with progressive liver disease, has now been described in recipients of solid-organ transplants² and a patient with lymphoma³ who is receiving immunosuppressive therapy. Data for our patient show that chronic infection with hepatitis E virus may occur in patients with HIV infection and is associated with active hepatitis.

Coinfection with hepatitis E virus and HIV may be overlooked because drug-induced liver injury is common in patients receiving antiretroviral therapy.⁴ Therefore, infection with hepatitis E virus may be misdiagnosed as drug-induced liver injury.⁵ Serologic testing may be unreliable in this context; PCR-based detection of hepatitis E viral RNA is essential to make the diagnosis. The prevalence of coinfection with hepatitis E virus and HIV, and the effects of such coinfection on chronic liver disease and prolonged carriage and the excretion of the hepatitis E virus, should be investigated further.

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CORRECTIONS

Peginterferon Alfa-2b or Alfa-2a with Ribavirin for Treatment of Hepatitis C Infection (August 6, 2009;361:580-93). In the first sentence of the Study Design subsection in Methods (page 581), the two levels of stratification according to hepatitis C virus (HCV) RNA levels given parenthetically should have been expressed as IU per milliliter, not as IU per cubic millimeter. In the first sentence of the Efficacy Assessments subsection in Methods (page 582), the lower limit of quantitation should have been 27 IU per milliliter, not 27 IU per cubic millimeter. In the Efficacy subsection of Results, the two mentions of HCV RNA levels (pages 583 and 584) should have been expressed as IU per milliliter rather than as IU per cubic millimeter. In the footnotes under Table 1 (page 585), the footnote designated by a double dagger should have expressed HCV RNA as IU per milliliter rather than as IU per cubic millimeter. In the footnotes under Table 2 (page 587), the footnote designated by an asterisk should have expressed HCV RNA as IU per milliliter rather than as IU per cubic millimeter. In the footnotes under Table 3 (page 589), the footnote designated by an asterisk should have expressed the lower limit of detection as 27 IU per milliliter, not 27 IU per cubic millimeter. We regret the errors. The article has been corrected at NEJM.org.

Subthalamic Nucleus Stimulation in Severe Obsessive-Compulsive Disorder (November 13, 2008;359:2121-34). In the Appendix (page 2133), the list of members of the French STOC Study Group from Nice was incorrect: E. Michel was listed twice, and M.N. Magnie-Mauro was omitted. The first instance of "E. Michel" should be replaced by "M.N. Magnie-Mauro." The article has been corrected at NEJM.org.