

unimportant level soon after the primary HCV infection period has waned.” This statement is not correct. We showed that the calculated FPR using a linear model most likely overestimates fibrosis progression in the setting of acute HCV infection, for which only short follow-up times are available. We are happy to see that Fierer et al agree with this interpretation and no longer calculate FPRs when reporting the outcome of liver biopsies of acute HCV infections. Indeed, concordant to the current consensus statement on acute HCV infections in HIV-infected individuals, we recommended that every HIV-infected patient should be advised to consider early treatment of acute HCV infection if spontaneous clearance does not occur [2]. We fully agree that further follow-up in patients withholding treatment is warranted to detect advancing liver fibrosis early and avoid the development of significant fibrosis and/or cirrhosis and ultimately end-stage liver disease [3].

We also agree with Fierer et al that the use of transient elastography for the assessment of liver fibrosis may be confounded by liver inflammation (as high levels of aminotransferases and of bilirubin are both associated with increased results of liver stiffness); however, this was already discussed in our manuscript. Of note, this confounder was addressed accordingly by multivariate analysis; in addition, the determination of liver fibrosis by liver biopsy may also be confounded by acute liver inflammation, as has been described previously [4]. More important, however, liver biopsy as an invasive procedure cannot rule out a certain degree of confounding by indication; although overall complication rates are low, there still remains a small but not negligible risk of mortality. This may lead to a selection of high-risk patients and overestimate liver fibrosis in patients with acute HCV infection.

Current data are not definitive, and future studies are warranted. Although it is important to raise awareness of possible

complications of acute HCV infection in HIV-infected individuals, we feel it is timely to inform patients and caregivers that fibrosis progression may indeed be lower than previously suggested and patients will not necessarily suffer from liver cirrhosis within a year’s time. In the meantime, noninvasive assessment of liver fibrosis by transient elastography—with consideration of the limitations of the procedure in the setting of acute hepatitis—may provide healthcare providers and patients with helpful information to monitor liver fibrosis after acute HCV infection on an individual patient basis. After all, the repeatability of the elastography measurements in 6-month intervals remains one of the obvious advantages of this noninvasive method over liver biopsy.

Note

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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References

1. Fierer DS, Mullen MP, Dieterich DT, et al. Early-onset liver fibrosis due to primary hepatitis C virus infection is higher over time in HIV-infected men. *Clin Infect Dis* 2012; 55:887–8.
2. European AIDS Treatment Network (NEAT) Acute Hepatitis C Infection Consensus Panel. Acute hepatitis C in HIV-infected individuals: recommendations from the European AIDS Treatment Network (NEAT) consensus conference. *AIDS* 2011; 25:399–409.
3. EACS European AIDS clinical society. Guidelines version 6, October 2011. Available at

Reply to Fierer et al

TO THE EDITOR—We are thankful for the comments made by Fierer et al, which enrich the current discussion on the natural course of acute hepatitis C virus (HCV) infection in human immunodeficiency virus (HIV)–infected individuals [1]. However, we do feel that the authors may have over-interpreted our data when stating that we demonstrated a “sharp decrease in the fibrosis progression rate (FPR) to a clinically

www.europeanaidsclinicalociety.org. Accessed 15 May 2012.

4. Scheuer PJ, Maggi G. Hepatic fibrosis and collapse: histological distinction by orcein staining. *Histopathology* **1980**; 4:487–90.

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