ted to an exhaustive prothrombotic screen [2]. In all patients, the results of the screening were negative, thus excluding the role of prothrombotic alterations in the development of HIV-associated IPH. Demonstration of the absence of prothrombotic disorders would further support the causative role of antiretroviral treatment (eg, didanosine), as shown by Kovari et al [1], in the pathogenesis of HIV-related IPH. In addition, we would like to point out that there is a high probability of HIV-infected patients with IPH developing portal vein thrombosis during follow-up, and this may further worsen the existing portal hypertension.

In summary, there are several important features of HIV-related IPH which may help the physician differentiate between cirrhotic portal hypertension and IPH in an HIV-infected patient who is receiving highly active antiretroviral therapy. First, despite clinical evidence of significant portal hypertension, the HVPG in most patients with HIV-related IPH is normal or only mildly elevated (<10 mHg). Second, liver elastography may help to raise the suspicion of IPH by giving false-negative results in the assessment of complications of portal hypertension. Third, these patients are prone to developing portal vein thrombosis during follow-up, which calls for regular screening of portal vein patency and consideration of anticoagulation. Clinicians should thus be aware of this emerging phenomenon and institute the appropriate screening and therapeutic measures.

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Pik-Eu Jason Chang and Juan-Carlos Garcia-Pagan

[1]Department of Gastroenterology and Hepatology, Singapore General Hospital, Singapore; and [2]Hepatic Hemodynamic Laboratory, Liver Unit, Institut de Malalties Digestives i Metabòliques, Hospital Clinic, Institut d'Investigacions Biomèdiques August Pi i. Sunyer, Barcelona, Spain

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Reprints or correspondence: Dr Jason Chang, Singapore General Hospital, Dept of Gastroenterology, Outram Rd, Singapore 169608, Singapore (jason.jan@pacific.net.sg).

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Reply to Chang and Garcia-Pagan

To the Editor—We thank Chang and Garcia-Pagan [1] for their interest in our study [2], and we offer the following reply. First, in our case-control study of human immunodeficiency virus (HIV)–infected patients with noncirrhotic portal hypertension (NCPH), inclusion criteria were the presence of endoscopically documented esophageal varices or hepatic venous pressure gradient (HVPG) ⩾10 mmHg, absence of hepatic cirrhosis on liver biopsy, and no common cause of liver disease. All of our case patients had endoscopically documented esophageal varices, and no patient was excluded because of a HVPG <10 mmHg. Eight of 15 case patients underwent hepatic hemodynamic evaluation; the median HVPG was 24.5 mmHg (range, 7–54 mmHg). Except in 1 case patient, all HVPG values were ⩾10 mmHg. Second, because most case patients received a diagnosis of NCPH at a time before liver elastography was regularly conducted, we did not evaluate liver stiffness values systematically. Third, we were not able to search for prothrombotic disorders, because cases were included retrospectively. However, in contrast to Chang et al [3], who did not find coagulopathies in their 8 patients, other reports have noted thrombophilic abnormality in affected patients [4–6]. This supports a multifactorial pathogenesis of NCPH in HIV infection, with antiretroviral therapy and a prothrombotic state leading to microthrombosis and vascular obstruction.

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Helen Kovari, Bruno Ledergerber, and Rainer Weber

Division of Infectious Diseases and Hospital Epidemiology, University Hospital, University of Zurich, Zurich, Switzerland

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Reprints or correspondence: Dr Helen Kovari, Div of Infectious Diseases and Hospital Epidemiology, University Hospital, CH-8091 Zurich, Switzerland (helen.kovari@usz.ch).

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