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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References

- Kovari H, Ledergerber B, Peter U, et al. Association of noncirrhotic portal hypertension in HIV-infected persons and antiretroviral therapy with didanosine: a nested case-control study. Clin Infect Dis 2009; 49:626–35.
- Chang PE, Miquel R, Blanco JL, et al. Idiopathic portal hypertension in patients with HIV infection treated with highly active antiretroviral therapy. Am J Gastroenterol 2009; 104:1707–14.
- Alvarez-Larran A, Abraldes JG, Cervantes E, et al. Portal hypertension secondary to myelofibrosis: a study of three cases. Am J Gastroenterol 2005; 100:2355–8.
- Osada Y, Kanazawa H, Narahara Y, Mamiya Y, Nakatsuka K, Sakamoto C. Wedged hepatic venous pressure does not reflect portal pressure in patients with cirrhosis and hepatic veno-venous communications. Dig Dis Sci 2008; 53: 7–13.
- Bosch J, Garcia-Pagan JC, Berzigotti A, Abraldes JG. Measurement of portal pressure and its role in the management of chronic liver disease.
 Semin Liver Dis 2006; 26:348–62.
- Ziol M, Handra-Luca A, Kettaneh A, et al. Noninvasive assessment of liver fibrosis by measurement of stiffness in patients with chronic hepatitis C. Hepatology 2005; 41:48–54.
- Levine AM, Vigen C, Gravink J, Mack W, Watts CH, Liebman HA. Progressive prothrombotic state in women with advancing HIV disease. J Acquir Immune Defic Syndr 2006; 42:572–7.
- 8. Eyal A, Veller M. HIV and venous thrombotic events. S Afr J Surg **2009**; 47:54–6.

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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 var-

ices, 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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References

- Chang P-EJ, Garcia-Pagan J-C. Idiopathic noncirrhotic portal hypertension in HIV-infected patients. Clin Infect Dis 2010; 50:127–8 (in this issue).
- Kovari H, Ledergerber B, Peter U, et al. Association of noncirrhotic portal hypertension in HIV-infected persons and antiretroviral therapy with didanosine: a nested case-control study. Clin Infect Dis 2009; 49:626–35.
- Chang PE, Miquel R, Blanco JL, et al. Idiopathic portal hypertension in patients with HIV infection treated with highly active antiretroviral therapy. Am J Gastroenterol 2009; 104:1707–14.
- Garvey LJ, Thomson EC, Lloyd J, Cooke GS, Goldin RD, Main J. Response to Mallet et al., 'Nodular regenerative hyperplasia is a new cause of chronic liver disease in HIV-infected patients.' AIDS 2007; 21:1494–5.
- Saifee S, Joelson D, Braude J, et al. Noncirrhotic portal hypertension in patients with human immunodeficiency virus-1 infection. Clin Gastroenterol Hepatol 2008; 6:1167–9.
- 6. Mallet VO, Bralet MP, Pol S. Response to Schi-

ano et al. Hepatoportal sclerosis as a cause of noncirrhotic portal hypertension in patients with HIV. Am J Gastroenterol **2008**; 103:808–9.

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