Pseudocirrhosis in HIV: Unmasking Non-cirrhotic Portal Hypertension Induced by Long-Term HAART

Abstract No: 105

Author: Intan Aaroni, MI[1]

[1] Gastroenterology and Hepatology Unit, Department of Internal Medicine, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Malaysia

Introduction

Non-cirrhotic portal hypertension (NCPH) is a rare but recognized complication in patients with HIV. While antiretroviral therapy—particularly certain agents within HAART—has been implicated in its development, other contributing factors such as pre-existing liver disease and immune reconstitution inflammatory syndrome (IRIS) should also be carefully evaluated. We present the case of a young woman with vertically acquired HIV on long-term HAART who developed persistent elevated alkaline phosphatase (ALP). Abdominal imaging revealed an irregular liver surface, features of portal hypertension, and splenomegaly, raising suspicion for chronic liver disease. However, liver biopsy showed no fibrosis but demonstrated a ground-glass appearance suggestive of HAART-induced liver injury. Liver stiffness measurements were within normal limits, supporting a diagnosis of non-cirrhotic portal hypertension.

Case report

24-year-old woman with vertically transmitted HIV, on HAART since age 11, was switched to tenofovir disoproxil, emtricitabine, and efavirenz in 2018. In 2021, she developed persistent jaundice and elevated alkaline phosphatase (ALP). Abdominal ultrasound revealed features of liver parenchymal disease with portal hypertension. Initial workup for hepatitis B and C, autoimmune hepatitis, and Wilson's disease was unremarkable. She was empirically treated for early cirrhosis.

Magnetic resonance cholangiopancreatography (MRCP) was performed due to persistently elevated ALP, incidentally revealing multiple liver lesions. Subsequent CT confirmed hepatocellular carcinoma (HCC), for which she underwent successful microwave ablation. After extensive counselling, a liver biopsy was performed.

Histology showed features consistent with non-cirrhotic portal hypertension (NCPH): hepatic arteriolar and portal vein stenosis, absent bridging fibrosis (confirmed by Masson trichrome and Victoria blue stains), and pseudo ground-glass hepatocytes; suggestive of HAART-induced liver injury, likely due to tenofovir disoproxil and efavirenz. Fibroscan readings remained stable and low over two years (4.9 and 4.5 kPa), supporting the absence of fibrosis.

Following multidisciplinary discussion, her antiretroviral regimen was changed to tenofovir alafenamide, lamivudine, and dolutegravir.

Discussion

This case underscores non-cirrhotic portal hypertension (NCPH) as an important but often overlooked differential in HIV-positive patients with signs of portal hypertension. Although imaging may mimic cirrhosis; with features like splenomegaly, varices, and ascites; normal liver stiffness measurements and the absence of fibrosis on liver biopsy point toward a non-cirrhotic etiology. Long-term HAART, particularly tenofovir disoproxil, has been implicated in NCPH. Histological findings such as ground-glass hepatocytes and obliterative portal venopathy support drug-induced liver injury. The underlying pathophysiology involves injury to hepatic sinusoids and portal veins, leading to obliterative venopathy and nodular regenerative hyperplasia (NRH), which increase portal pressure without fibrosis. Mitochondrial toxicity and immune reconstitution may also play contributory roles. Early recognition of NCPH is essential to avoid misdiagnosis and unnecessary treatment, and to allow timely adjustment of antiretroviral therapy.

Conclusion

Non-cirrhotic portal hypertension should be considered in HIV patients on long-term HAART presenting with portal hypertension signs. Liver biopsy and elastography are key distinguishing it from true cirrhosis, allowing for appropriate management and therapy adjustment.

Next step

Long-term management, monitoring, and prevention of complications of non-cirrhotic portal hypertension (NCPH). Change of HAART hopefully halt the progression of NCPH and its complications.

Acknowledgement

We thank the Infectious Disease and Hepatology teams for their valuable clinical support and guidance in managing this case. We also acknowledge the Radiology and Pathology departments for their assistance with imaging and histopathological analysis. Finally, we express our gratitude to the patient and her family for their cooperation and consent to share this case for educational purposes

Contact Email

intanaaroni@gmail.com