

Thrombopenia and/or Splenomegaly in HIV/HCV Coinfected Patients With Mild Liver Fibrosis Alerts for the Risk of Portal Hepatopathy

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Background: Noncirrhotic portal hypertension has recently emerged as a new entity in HIV-infected patients. Histological findings often reveal different hepatic portal peri-vascular abnormalities. In most cases, it was related to previous didanosine exposure. Although its recognition could be more difficult, this drug-induced vascular damage in the liver might also appear in HIV/HCV coinfecting patients.

Methods: Three HIV individuals with chronic hepatitis C presented with complicated portal hypertension as esophageal variceal bleeding. Liver fibrosis measured by transient elastometry did not reveal advanced liver fibrosis. Liver function tests were completely normal. All subjects presented previous clinical signs of portal hypertension, thrombopenia and splenomegaly, lasting on average 4 and 2 years, respectively. They had been exposed to didanosine for long periods in the past.

	Case 1	Case 2	Case 3
Age (yrs)	41	49	42
Gender	Male	Female	Male
Race	Caucasian	Caucasian	Caucasian
Risk group	IDU	IDU	IDU
On HAART	Yes	Yes	Yes
Prior didanosine exposure (months)	64	30	44
CD4 count (cells/mm ³)	351	168	324
Plasma HIV-RNA (copies/mL)	<50	<50	<50
Platelet count /uL	78,000	57,000	81,000
Liver fibrosis stage (Metavir, stiffness kPa)	F2 (9)	F3 (11.5)	F4 (15)
Splenomegaly	Yes	Yes	Yes

Discussion: Primary hepatic vascular damage induced by didanosine might result in noncirrhotic portal hypertension. This condition may appear in HIV patients without any known cause of liver disease, as well as superimposed to other hepatic illnesses, as chronic hepatitis C. The recognition of clinical classical signs of portal hypertension (eg, thrombopenia, splenomegaly) in the absence of a significant liver fibrosis or synthetic function compromise may alert on the possibility of this condition.

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