Portal vein thrombosis in an HIV-positive man: a case report

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ABSTRACT

Background: HIV is known as a risk factor for venous thromboembolic events. However, portal vein thrombosis is rare among HIV-infected patients. Few cases have been described in the literature.

Case Presentation: We report a case of abdominal pain in a 25-year-old HIV-positive man with a low CD4 count and co-infection with hepatitis B. The pain existed for 4 days. Computed tomography showed portal vein thrombosis, and the anticoagulation therapy was initiated.

Conclusion: This case provokes questions about the etiology of portal vein thrombosis in HIV-positive patients and might determine risk factors. It emphasizes the importance of considering portal vein thrombosis as a cause of abdominal pain in HIV-infected patients, in particular when they have low CD4 counts or co-infection with hepatitis B. In addition, HIV and viral hepatitis should be considered in patients who present with thrombotic events.

Keywords: Venous thromboembolism, portal vein thrombosis, HIV, viral hepatitis, thrombosis, hepatitis B, case report.

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Background

HIV-infection is an independent risk factor for venous thromboembolic events [1]. Incidence rates of venous thromboembolic events (VTE) among HIV-infected patients range from 0.19% to 7.63% per year [1]. The risk of VTE is about 2- to 10-fold higher than expected in the general population. Portal vein thrombosis (PVT) is rare among HIV-infected patients [2]. However, the risk increases if a patient is co-infected with viral hepatitis. Our patient suffered from a co-infection with chronic hepatitis B.

Case Presentation

We report a case of a 25-year-old homosexual man, who developed a portal vein thrombosis after 3 weeks of HIV diagnosis and the initiation of combination antiretroviral therapy (cART).

He was admitted to our hospital because of abdominal pain. His complaints started 4 days before presentation and the pain persisted continuously in the upper abdomen. He also reported nausea and vomiting for 4 days. His stools were normal. He did not smoke and was not known with alcohol or substance abuse. Three months earlier, he was diagnosed with an HIV-1 infection by his general practitioner who performed the test because of fever.

On physical examination, the abdomen was diffusely tender, and the liver was palpable 2 cm under the costal arch.

Screening for the hepatitis B virus (HBV) was positive but there were no signs of an acute HBV infection. Three months before presentation, cART [emtricitabine, tenofovirdisoproxil, elvitegravir, cobicistat (Stribild®)] had been initiated. At the time of his HIV-1 diagnosis, the viral load was 180,000 copies/ml and his CD4 count was 125 cells/mm^3. Laboratory results on presentation are shown in Table 1.

An abdominal ultrasound showed a portal vein thrombosis, extending to the splenic vein. A computed tomography scan confirmed the diagnosis. There was no ascites.

Anticoagulation therapy was initiated. In a few days, the pain decreased and the patient could be discharged after 5 days.

Discussion

HIV infection is a well-known prothrombotic condition. A potential pathophysiological pathway is the increased inflammatory and hypercoagulable state and the endothelial dysfunction in HIV-infected patients [1]. Furthermore, deficiencies of protein S and protein C—common in HIV-positive patients—are contributable factors due to a decreased synthesis in endothelial cells, hepatocytes and megakaryocytes, and high consumption in disseminated intravascular coagulation [1,3]. Although the risk of thrombosis in HIV-positive patients impressively

Table 1. Laboratory results at day of presentation.

LABORATORY MEASUREMENT	RESULT	NORMAL RANGE	UNITS OF MEASUREMENT
Hemoglobin	12.7	13.0–17.0	g%
Leukocytes	5.1	3.5–10.5	10^9/l
Thrombocytes	170	150–390	10^9/l
Creatinine	9.6	7.0–13.0	mg/l
C-reactive protein	27	<0.80	mg%
Aspartate aminotransferase	29	10–40	U/l
Alanine aminotransferase	22	9–44	U/l
Gamma-glutamyltransferase	23	12–64	U/l
Alkaline phosphatase	74	30–110	U/l
Bilirubin total	0.6	<1.2	mg%
Lactate dehydrogenase	311	125–226	U/l

decreased since the era of cART, the chance of developing thrombosis is still 2- to 10-fold higher than in the general population [1]. The risk increases if the CD4 count decreases below 200/mm^3 and when the immune suppression is accompanied by an opportunistic infection [1,2,4]. This patient had a CD_4 count of 125/mm^3. We did not test for protein S or C deficiency because there were no indications to assume the patient suffered from these coagulation disorders. Furthermore, false positive results in the acute phase are common and there would not be clinical consequences.

Considering the chronic inactive HBV infection, viral hepatitis leads to an increased incidence of portal vein thrombosis, even without liver cirrhosis [5]. HBV can induce inflammatory changes in the endothelium of the portal vein, leading to activation of the coagulation system by inflammation.

Both portal hypertension and hepatic cirrhosis are major risk factors for PVT. Although portal hypertension is often a complication of hepatic cirrhosis, noncirrhotic portal hypertension (NCPH) is a known entity. One of the causes of NCPH is acute or chronic viral hepatitis, due to an inflammatory effect leading to fibrosis of the space of Disse. In this case, no signs of hepatic cirrhosis nor portal hypertension were noted but the chronic liver disease itself can cause a prothrombotic state due to the release of both proinflammatory and procoagulant factors, such as plasminogen activator inhibitor-1, C-reactive protein, interleukin-6, and fibrinogen [6].

Antiretroviral therapy has been associated with developing VTE. The patient started on Stribild® 3 weeks before the diagnosis of portal vein thrombosis had been confirmed, but the risk of VTE is, in particular, associated with protease inhibitors, which is not included in Stribild® [7]. Thus far, no association between the use of Stribild® and VTE has been reported.

This patient most likely developed VTE because of his combination of HIV with hepatitis B. Chronic viral infection (HIV as well as viral hepatitis) is a thrombotic risk factor because of the infection-mediated continuous inflammatory state and hypercoagulability. The location of his VTE—the portal and splenic vein—is very likely due to his co-infection with the HBV. Some studies suggest that HBV is the major risk factor for PVT but those studies included South Asian populations where geographically HBV is the most common cause of liver cirrhosis [8]. Probably, the cirrhosis itself is more important than the cause of the cirrhosis (HBV). However, more research is needed to clarify the impact of the causes of cirrhosis on the incidence of PVT.

Conclusion

In conclusion, the occurrence of PVT in HIV-positive patients may be of multifactorial origin. Both disease risk factors as well as co-infections (e.g., HBV infection) and medication may play a role. Portal vein thrombosis must be included in the differential diagnosis of HIV-positive patients with abdominal pain, in particular when they have low CD_4 counts or are co-infected with hepatitis B. In addition, HIV and viral hepatitis should be considered in patients who present with thrombotic events.

Acknowledgment

None.

List of Abbreviations

cART	Combination antiretroviral therapy
HBV	Hepatitis B virus
HIV	Human immunodeficiency virus
NCPH	Noncirrhotic portal hypertension
PVT	Portal vein thrombosis
VTE	Venous thromboembolic event

Consent for publication

Informed consent was obtained from the patient to publish this case in a medical journal.

Ethical approval

Ethical approval is not required at our institution for publishing a case report in a medical journal.

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Summary of the case

Patient (gender, age)	1	Male, 25 years old
Final diagnosis	2	Portal vein thrombosis
Symptoms	3	Abdominal pain, nausea, and vomiting
Medications	4	Fraxiparine and acenocoumarol
Clinical Procedure	5	Anticoagulation therapy; first with both fraxiparine and acenocoumarol, later with acenocoumarol only
Specialty	6	Internal medicine