

Portal system thrombosis in an HIV-infected child

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Abstract:

HIV infection can lead to venous thrombosis due to protein C deficiency, protein S deficiency, or antiphospholipid syndrome. Most patients present with deep vein thrombosis or pulmonary embolism. We report a 10-years-old HIV-infected girl who presented with life-threatening hematemesis. Computed tomography scan of the abdomen and angiography showed thrombosis of the portal vein, superior mesenteric vein, and splenic vein. She was advised antiretroviral therapy and prothrombotic workup, however, she took discharge against medical advice.

Key words: HIV, portal vein, thrombosis, venous thrombosis

INTRODUCTION

HIV infection is known to be a prothrombotic state with a 2–10-fold higher than expected prevalence of venous thromboembolism (VTE) as compared to the general population.^[1] The possible etiologies of VTE in HIV-infected individuals are low CD4⁺ cell count, protein S deficiency, protease inhibitor therapy, and antiphospholipid antibodies.^[1-3] The common VTE reported in HIV-infected individuals are deep vein thrombosis (DVT) and pulmonary embolism.^[1] Portal system thrombosis has rarely been reported in HIV-infected adults^[4-7] but not in children. We present a 10-years-old HIV-infected girl presenting with hematemesis and detected to have portal vein, superior mesenteric vein, and splenic vein thrombosis.

CASE REPORT

A 10-year-old girl presented with fever, abdominal pain, hematemesis, and melena for 1 day. She had typhoid at 5 years of age and no other illness in past including jaundice. Father died due to HIV

and tuberculosis 3 years ago and mother was also HIV infected. On examination, weight was 20 kg and she had hepatosplenomegaly with ascites. Investigations showed hemoglobin 8.1 g/dl, white blood cell count of 23,000/cumm (85% polymorphs and 15% lymphocytes), and platelets of 45,000/cumm. Liver function tests were normal. Hepatitis B surface antigen and anti-hepatitis C ELISA were negative. HIV ELISA was also positive by two different kits. Ultrasound (USG) of the abdomen and Doppler showed portal vein thrombosis (PVT) with portal collaterals. Initially, she was treated with intravenous fluids, blood transfusion, and even required invasive ventilation. After stabilization, she was put on diuretics and propranolol. Ascites required recurrent tapping and finally, a peritoneal drain was put. Subsequently, a computed tomography (CT) of the abdomen and angiography was done that showed thrombosis of the portal vein, superior mesenteric vein, splenic vein with splenic infarcts, and abdominal lymphadenopathy. She was advised regarding testing for CD4 count and starting antiretroviral therapy and upper gastrointestinal

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endoscopy along with thrombotic workup but took discharge against medical advice.

DISCUSSION

Portal system thrombosis in children can be secondary to liver transplantation, infections, splenectomy, sickle cell disease, chemotherapy, presence of antiphospholipid syndrome, and hypercoagulability.^[8] Although cases of portal system thrombosis in HIV patients had been previously reported, it is a rare form of venothromboembolism seen in HIV infection.^[4-7] HIV-induced thrombosis commonly involves the deep venous system, pulmonary veins, retinal vein, and cerebral vein.^[1] A wide spectrum of findings such as the presence of lupus anticoagulant and antiphospholipid antibodies; high levels of von Willebrand factor, factor VIII, and homocysteine; low protein C, protein S, antithrombin, heparin cofactor II, and CD4 counts are reported.^[2,3] Some of these findings themselves can be attributed to other underlying causes, such as direct effect of HIV antigen on endothelial cells, the role of opportunistic CMV infection, interference with the metabolism of coagulation pathway proteins, and effects of molecules secreted by AIDS-associated neoplasms.^[1,3] Antiretroviral drugs particularly indinavir (and its associated lipodystrophy) also interferes with coagulation protein's metabolism.^[1,3] Megestrol acetate used as appetite stimulant in HIV patients suffering from cachexia is also associated with hypercoagulability.^[1] Thrombosis in non-HIV infected people is usually explained by Virchow's triad (hypercoagulability, endothelial dysfunction, and hemodynamic changes), these factors may be absent or exacerbated in HIV-infected individuals presenting with thrombosis.^[1]

Acute presentation of PVT is relatively rare. Acute thrombosis may remain clinically silent or presents as abdominal pain particularly in the right upper quadrant, nausea, and variceal bleeding. Fever, chills, and liver tenderness if present suggests septic PVT (acute pylephlebitis). If the pain radiates to back, intestinal infarction should be suspected due to clot extension. If preexisting varices present, hematemesis and melena may be seen. Splenomegaly and ascites are more commonly seen in chronic cases.^[8] In suspected PVT, diagnosis can

be confirmed by Doppler USG or contrast-enhanced abdominal CT. Treatment of PVT in HIV-infected patients is the same as that of non-HIV-infected children. Anticoagulation therapy with low molecular weight heparin and later warfarin should be started provided there is no excessive bleeding risk. If acute variceal bleeding is present, then variceal banding and sclerotherapy through endoscopy are most appropriate.^[8]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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