Nigerian Medical Journal
Vol. 52 Issue 2
April - June 2011

CASE REPORTS

HIV Associated Deep Vein Thrombosis: Case Reports from Jos, Nigeria

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ABSTRACT

Deep vein thrombosis (DVT) has been reported to be 2-10 times commoner in HIV infected patients than in the general population. We report two cases of extensive unilateral deep vein thrombosis involving the lower limb in HIV infected patients on highly active antiretroviral therapy (HAART). Doppler ultrasound in the two patients revealed evidence of venous thrombosis from the femoral vein down to the posterior tibial veins. None of the patients had a history of acquired risk factors for DVT. Both patients responded well to anti-coagulants. A high index of suspicion may therefore be required to make the diagnosis and institute adequate management for this condition which has potentially life-threatening consequences.

Key Words: Acquired immune deficiency syndrome, Human immunodeficiency viral infection, deep vein thrombosis,

INTRODUCTION

The Human Immunodeficiency Virus (HIV) infection/ the Acquired Immunodeficiency Syndrome (AIDS) has been recognized as an independent risk factor for venous thrombo-embolic events (VTE). Several factors involved in thrombogenesis are influenced by HIV infection including coagulation factors, endothelial function, platelet activation and other pro-inflammatory factors[1, 2]. The current incidence of VTE among HIV-infected persons is estimated to be 2.6/1000 person-years[3]. VTE has been reported to be 2-10 times commoner in HIV infected patients than in the general population[4, 5].

Factors associated with VTE in HIV/AIDS include age = 45 years, presence of AIDS-defining opportunistic conditions, hospitalization and the use of protease inhibitors among others[3]. HIV-associated deep vein thrombosis (HIV-DVT) often occurs in the absence of recognized thrombogenic risk factors such as immobility, advanced age, cigarette smoking, pelvic surgeries, pregnancy, oestrogen therapy and personal or family history of DVT[1].

Despite the fact that sub-Saharan Africa bears the greatest burden of HIV-infection, most reports of HIV- DVT are from the western world. We report DVT in two patients with HIV/AIDS who had no apparent thrombogenic risk factors.

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CASE REPORTS

CASE 1:

IB is a 32 year old man on follow up for HIV/AIDS at the Jos University Teaching Hospital (JUTH). He was diagnosed to have HIV infection in 2007 and had been on highly active antiretroviral therapy (Zidovudine, Lamivudine and Efavirenz) for 13 months with good virological and immunological response. He was admitted with a two week history of progressive right leg swelling and pain around the calf. There was no history of antecedent trauma. Review of systems was not remarkable. History of prolonged immobility, smoking, recent surgery and recent long distance travel were absent. He had no previous history of DVT and no family history of DVT. He was not a known hypertensive or diabetic patient.

Physical examination revealed an otherwise healthy looking man with a pulse rate of 88b/min and a blood pressure of 126/80mmHg. He had a right lower limb oedema with prominent superficial veins and diminished peripheral pulses. Other pulses were normal. The limb was neither warm nor tender. A systemic examination was normal.

A Doppler ultrasonographic scan of the right lower limb showed that all the veins were non-compressible with absent colour flow velocity.

The arteries of the right lower limb were normal. His CD4 cell count was 309cells/μL and HIV viral load was undetectable. His complete blood count, lipid profile and liver biochemistry were all within normal limits.

Following a diagnosis of right lower limb DVT, he was commenced on subcutaneous Enoxaparin 40mg OD and Tabs Warfarin 5mg OD concomitantly. The Enoxaparin was discontinued after five days while he was maintained on Warfarin. He was also placed on Tabs Aspirin 75mg daily. After 11 days on admission the leg swelling resolved, the pains subsided and he was discharged home. He was maintained on low dose Warfarin and dose titrated to achieve a target INR of 2.5 for three months before discontinuation. He had remained symptom free for six months and has continued antiretroviral therapy.

CASE 2:

AP, a 45 year old female teacher was diagnosed to have HIV infection in 2004 and has since been on HAART (current regimen: Tenofovir, Emtricitabine and Efavirenz). She presented with a two months history of left leg swelling with associated pain that was made worse by walking and relieved at rest. She had slight reduction in leg swelling before presentation. There was no history of antecedent trauma. Review of systems was unremarkable. History of acquired risk factors for DVT including recent pregnancy and hormonal contraceptive use was absent. She was not known to have hypertension or diabetes mellitus.

Physical findings included left lower limb oedema with distended superficial veins and differential warmth compared to the right lower limb. There was no tenderness. She had a pulse rate of 92b/min and a BP of 130/80 mmHg. All peripheral pulses were of normal volume. Other cardiovascular and systemic examination revealed normal findings.

Doppler Ultrasound of the left lower limb showed non-compressibility and loss of colour flow velocity from the femoral vein down to the posterior tibial vein. The complete blood count and liver biochemistry were all normal. Her CD4 cell count was 232/μL and HIV viral load was undetectable.

A diagnosis of left lower limb DVT was made and she was commenced on subcutaneous Enoxaparin 40mg OD and Tabs Warfarin 5mg OD; the Enoxaparin was discontinued after five days and she was maintained on Warfarin Tabs. The leg swelling and pain subsided after a week on admission. She was discharged home to be followed up on out-patient basis on oral Warfarin after target INR was achieved.

DISCUSSION

The observation of our report, as well as those of other studies argues for VTE occurring in HIV infected individuals independent of the classical risk factors for DVT[1,2,4]. However, we acknowledge the fact that coagulation factor derangements (protein C and S deficiency, low anti-thrombin III levels, elevated factor VIII levels, elevated homocysteine, presence of antiphospholipid antibodies), which we did not have the facilities to assay have been found in some of the cases of VTE associated with HIV[8]. While these coagulopathies could be found in HIV negative individuals as independent risk factors for thrombosis, they are recognized HIV-mediated acquired mechanisms that partly explain the increased risk of thrombosis seen in HIV-infected populations[9-11].

Certain clinical variables have been associated with VTE in HIV. Increasing age (= 45 years) has been associated with HIV-DVT. Our second patient falls within this age group; however, the first patient was aged 32 years. Besides, VTE has been reported in HIV infected patients aged below 45 years[12]. The protease inhibitors (especially Indinavir) have been associated with venous thrombosis though inconsistently[8,13,14]. None of our patients was on a protease inhibitor-based regimen. Another variable associated with VTE in HIV is the presence opportunistic conditions although none of our patients had any. Reports associating low immune status with VTE in HIV are conflicting [1, 6, 15]. Our patients had CD4 count above 200 and undetectable viral load.

Our patients presented with DVT of the lower extremity. This is in keeping with previous reports that the commonest anatomical location for VTE in HIV patients is the lower extremity[6]. Our patients responded adequately to standard therapy.

Our report supports the fact that DVT occurring in HIV-infected individuals without apparent risk factors for thrombosis is also found in sub-Saharan Africa. A high index of suspicion

may therefore be required to make the diagnosis and institute adequate management for this condition which has potentially life-threatening consequences.

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