Case Report

Pulmonary thromboembolismin an AIDS patient with chronic venous insufficiency: A case report

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Abstract

Pulmonary embolism is a potentially fatal disorder. Data regarding the outcome of clinically recognised cases of pulmonary embolism is sparse. Due to the increasing incidence of detection of Human Immunodeficiency Virus (HIV) and Acquired Immune Deficiency Syndrome (AIDS) along with better antiretroviral therapy, the life expectancy of these patients is increased with early diagnosis and treatment. Literature supports the increased incidence of thrombosis with embolic episodes in immunocompromised cases. We report a 43 year oldimmunocompromised male suffering from pulmonary thromboembolism due to chronic venous insufficiency on antiretroviral therapy.

Keywords: Venous Insufficiency, Pulmonary Thromboembolism, HIV Infection and AIDS.

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CASE REPORT

A 43 year old gentleman was brought to the hospital with history of shortness of breath since 5 days. He gave a history of chronic venous insufficiency since last one year and was hospitalised for same. He had been diagnosed with HIV 4 years ago and was on HAART (Highly Active Anti-Retroviral Therapy)[1] combination of Lamivudine, Nevirapine and Zidovudine since then, none of which are known to cause thromboembolic phenomenon. Physical and systemic examination was normal. He was subjected to an ECG which revealed a sinus rhythm with normal axis and right ventricular strain pattern, 2D ECHO was suggestive of mild dilatation of RA/RV with severe pulmonary hypertension (PASP=75mm of Hg by TR Jet) and the IVC was normal in size and collapsing with respiration. Bilateral venous doppler revealed a right popliteal deep venous thrombosis. CT pulmonary angiogram showed thromboembolism involving bilateral pulmonary arteries, their lobar and segmental branches.

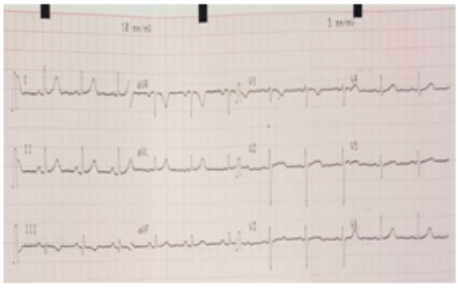

Figure 1

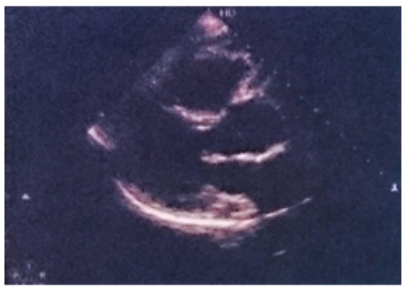

Figure 2

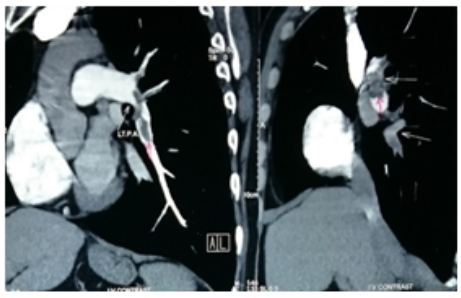

Figure 3

Legend

Figure 1: ECG showing a right ventricular strain pattern and normal axis.

Figure 2: 2D ECHO; PLAX view showing RV dilatation.

Figure 3: CT Pulmonary Angiogram showing luminal narrowing of left pulmonary artery following intravenous contrast

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Procoagulant workup in form of Protein C and S, Anti-Phospholipid Antibodies, Anti-Thrombin III levels, Lupus Anticoagulant, and Anti-Cardiolipin Antibodies were within normal limits. Homocysteine levels were 20.2 mmol/L (<15 mmol/L). He was admitted in the ward with a bridging therapy of Low Molecular Weight Heparin and Warfarin with alternate day monitoring of INR, and oral supplementation of Vitamin B6, B9, B12. There was relief of symptoms after 2 days of therapy. He was discharged on day 7 with an INR of 2.7. 2D ECHO at discharge suggested normal RA/RV size with mild Pulmonary Hypertension (PASP=38 mm of Hg).

REVIEW

Multiple risk factors are involved in the development of Pulmonary Thromboembolism (PTE) which can be grouped into venous stasis, injury to the vessel wall and hypercoaguable states described as Virchow's triad[2]. In patients with HIV apart from the mentioned aetiology there is increased predisposition to disseminated intravascular coagulopathy, interference with anticoagulation factors due to the primary disease and its therapy in the form of protease inhibitors[9]. These abnormalities are also related to the degree of immunosuppression i.e. low CD4+T cell count.

DISCUSSION

Retrospective cohort studies of patients infected with HIV suggested an incidence of venous thromboembolism (VTE) to be 1% to 2%, and is 10 times that expected among people without HIV. In patients with HIV infection, several abnormalities were detected that predispose to the hypercoagulability state, including endothelial cell injury[5], low-grade disseminated intravascular coagulopathy[4], interference with the anticoagulation factors[3,4,6,8] and the use of the drugs such as protease inhibitors[7]

TAKE HOME MESSAGE

Though the diagnosis and treatment of PTE in HIV/AIDS patients is similar to those of Non-HIV/AIDS patients, in young patients with unprovoked thromboembolic events, testing for HIV should be considered in absence of any other identifiable risk factors.

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