HIV-2 (Human-Immunodeficiency Virus) : A Myriad of Myths – Presenting as Multiple Large Vessel Arterial Occlusions

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A 17 year old unmarried girl presented to us with a 1 month history of bilateral lower limb claudication pain and 1 week history of right upper limb rest pain with oliguria. Examination revealed feeble right radial pulse with radio-radial delay and bilateral cold lower limbs. Blood pressure in right upper limb was systolic 60 mmHg and in left upper limb 100/60 mmHg. Cardiac and respiratory systems were normal on examination. An urgent CT angiogram was done which revealed occlusion of right subclavian artery (Figure 1), Superior mesenteric artery occlusion (Figure 2) and near total occlusion of descending aorta at the level of renal arteries (Figures 3 and 4). Her anti-phospholipid antibodies and autoimmune serology were within normal limits. Transesophageal echocardiography excluded any cardiogenic source of emboli. Surprisingly, her ELISA for HIV antibody was positive. In view of her young age and unmarried status, HIV serology of both parents were done which was negative. Her CD4 count was 36 and Western blot revealed HIV-2 infection. She was started on TDF/FTC/ATV(r) (Tenofovir / Emtricitabine / Boosted Atazanavir) with aspirin and statin. However, her renal dysfunction worsened due to near total renal artery occlusion requiring hemodialysis. She succumbed to her illness after four weeks.

HIV infected patients are at increased risk for venous and arterial thromboembolic events. Multiple markers related to inflammation (IL-6, TNFRI, C-reactive protein) and coagulation (tissue factor expression, FVIII, thrombin, fibrinogen and D-dimer levels) are increased in HIV infection, and are predictive of thrombotic risk and mortality. The mechanism may be related to chronic immune activation and inflammatory state in both untreated and treated HIV infection. However, traditional risk factors, including smoking and dyslipidemia must also be considered.¹ One study observed a marked increase in vWF levels as well as a correlation of vWF to first and recurrent venous thromboembolic events.² Few cases have described the presentation of HIV with arterial thrombosis.³⁻⁵ Due to the association of HIV infection and autoimmune disorders such as antiphospholipid antibody syndrome, evaluation of serum level of antiphospholipid-antibodies has been introduced as a fundamental step in the management of both symptomatic and asymptomatic HIV patients. Frequency of thrombophilic abnormalities in HIV infection increases with its progression to AIDS and correlates with the severity of immunosuppression, with the presence of concurrent opportunistic infections or neoplastic processes.³ D-dimer levels have been strongly linked to both venous and arterial thrombosis in HIV. Targeted interventional studies may help to identify the determinants of coagulation risks in treated HIV infection. Although

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aspirin and statins have proven value in reducing cardiovascular risks among HIV-uninfected populations, their clinical utility in treated HIV infection has not yet been demonstrated and merits evaluation. As per a study, HIV infection is an independent risk factor for coagulation abnormalities and this could be a reason to prolong anti-thrombotic treatment in patients with a history of thrombosis.

References