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Acute Upper Arm Ischemia in a Patient with Human Immunodeficeincy Virus Infection: Underwent Successful Surgical Thromboembolectomy

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1. Abstract

Patients with human immunodeficiency virus (HIV) are in hypercoagulabe state due to various coagulation abnormalities and at increased risk for thromboembolic events. We report acute upper arm ischemia caused by spontaneous thromboembolism with no identified source in a patient with HIV infection. Patient underwent successful surgical thromboembolectomy and had good postoperative recovery. Physicians should be aware of thromboembolic disease as the possible complication of HIV.

2. Key words

HIV; Primary thrombosis; Thromboembolectomy

3. Introduction

Increased risk of venous thromboembolism is well recognized in patient with human immunodeficiency virus (HIV), and the disease has been suggested to represent a pre-thrombotic state. Even so, little exist in literature concerning arterial thromboembolism causing upper arm ischemia in patients with HIV. We report a case report of acute embolic occlusion of non-diseased brachial and axillary artery in a 42 years old man with HIV infection.

4. Case Report

A 42 years man with HIV infection presented to Emergency Department with acute left upper arm painlasting for 6-7 hours. Pain was acute in onset, severe in intensity, continuous and now was associated with numbness of forearm. He was on Highly Active Antiretroviral Therapy (HAART). There was no history of cardiac disease. At physical examination, normal arterial pulses were present in right (unaffected) arm, but no pulses or doppler signals were found at or below the brachial artery in left upper arm [1,2]. The left upper arm was cyanotic and markedly cooler than the right arm. Capillary refill was markedly delayed and there was evidence of sensory loss in finger tips. Patient was accompanying adopplerultrasound showing total occlusion of left axillary and brachial artery with acute thrombus and no visible distal flow. After blood samples were drawn for laboratory studies, intravenous heparin was started. An electrocardiogram showed normal sinus rhythm. Hemoglobin was 13.1 g/dl, WBC 10X 109/l, Platelets count were 288 x 109/l. International normalized ratio (INR) and partial thromboplastin time (PTT) were within normal laboratory limits. The patient underwent tromboembolectomy through lazy ‘S’ incision in left elbow fossa under general anesthesia (Figure. 1). Longitudinal arteriotomy was made just above the bifurcation of brachial artery and thrombus was removed both proximally and distally with the help of Fogarty balloon catheter. After clearing the arterial tree, longitudinal arteriotomy closed with vein patch. Grossly the arterial wall was normal [2,3]. Postoperatively patient has palpable pulses and hand became warmer and pink. He received postoperatively continuous intravenous heparin infusion which was overlapped with oral anticoagulants. Patient was discharged on oral warfarin and advises to maintain INR in therapeutic range. On follow up at 2 months, he had no recurrence of symptoms and having viable, functional limb.

Echocardiogram showed no abnormality. Levels of Protein C, Protein S, Antithrombin III, and Factor V Leiden were within normal laboratory limits.

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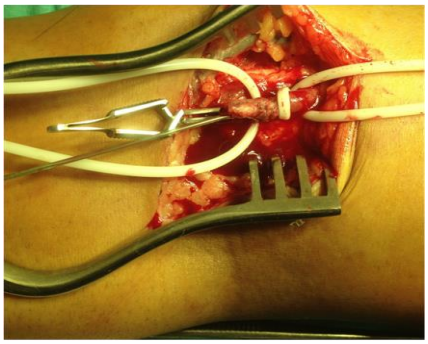

Figure 1: Thrombus extracted from the brachial artery with the help of Fogarty Catheter.

5. Discussion

Patients with human immunodeficiency virus (HIV) have various coagulation abnormalities as well as increased risk for development of clinical thrombosis and subsequent embolic events. Abnormalities predisposing to ahypercoagulablestate that have been detected in HIV patients includeantiphospholipid antibodies , lupus anticoagulants , increased von Will brand factor, deficiency in Protein C and S, antithrombin and heparin cofactor. Factors such as opportunistic infections and neoplasm may also contribute to the hypercoagulable state and hence to thrombotic events.

HIV/ AIDS associated vasculopathy was first recognized as an entity in 1987. Since then there has been several reports on large vessel vasculopathy associated with HIV/AIDS and few of these have dealt with peripheral arterial thrombosis and its management. Pathogenesis of HIV/ AIDS associated vasculopathyis multifactorial. HIV may produce protein –wasting nephropathy or endothelial cell injury, both of which may possibly contribute to alterations in circulating levels of anticoagulant proteins. Furthermore, reduction in CD 4+ cell count to less than 200cellss/ mm3has been observed in patients with thromboembolic complication. Nair et al. [4] first described the pathology in detail, which was regarded as unique to HIV/AIDS vasculopathy. They noted that inflammation was centered on the vasa vasora in the adventitia and was characterized by a vasculitis in which the vessels were surrounded by acuff of neutrophils, monocytes and plasma cells, which ultimately lead to occlusion of vasa vasora with resultant transmural fibrosis and necrosis. This either leads to HIV associated aneurysm formation or localized thrombosis. HIV-related thrombosis, which is segmental, shows a histological picture with inflammatory changes confined to the vasavasora with bland organizing luminal thrombus. The striking feature of this disease is the normality of the arterial tree proximal to the thrombosed arteries by duplex ultrasonography, angiography and macroscopic appearance. The other remarkable feature is the thrombosis of all distal vessels with no demonstrable runoff. Duplex ultrasound also showed hypoechoic ‘spotting’ in the arterial wall, the’ string of pearls sign’, which also has been observed in patients with HIV associated arterial aneurysm [5,6].

Surgical thromboembolectomy is effective is restoring the circulation as was this case. Percutaneous mechanical thrombectomy and thrombolysis has been also found effective in various case reports.

The duration of necessary anticoagulation is unknown, but generally these are candidates for prolong anticoagulation due coagulation abnormalities present in a patient of HIV/AIDS.

6. Conclusion

This case highlight arterial ischemia caused by spontaneous thrombosis is a patient with HIV. The clinical diagnosis of limb threatening ischemia was prompt, as was intervention. Surgical embolectomy restored upper extremity circulation. Physicians should be aware of thromboembolic disease as the possible complication of HIV.

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