Spontaneous arterial thrombosis in a patient with human immunodeficiency virus infection: Successful treatment with pharmacomechanical thrombectomy

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Patients with human immunodeficiency virus (HIV) have various coagulation abnormalities as well as increased risk for development of clinical thrombosis and subsequent embolic events. We report acute lower leg ischemia caused by spontaneous atheroembolism with no identifiable source in a young patient with HIV infection. Treatment included percutaneous mechanical thrombectomy and thrombolysis, which reversed the arterial ischemia. Physicians should be aware of thromboembolic disease as a possible complication of HIV. (J Vasc Surg 2003;38:392-5.)

Spontaneous atheroembolism to the lower extremities most commonly occurs in older patients, with approximately 85% of peripheral emboli being of cardiac origin. In younger patients, who may not have an apparent identifiable cause, evaluation for hematologic abnormalities and hypercoaguability is often pursued. Increased risk for venous thromboembolism is well recognized in patients with human immunodeficiency virus (HIV), and the disease has been suggested to represent a pre-thrombotic state. Even so, little exists in the literature concerning arterial thromboembolism causing acute ischemia in patients with HIV. We present a case report of acute embolic occlusion of a non-diseased popliteal artery in a 37-year-old man with HIV infection.

CASE REPORT

A 37-year-old African American man with known HIV infection had acute left lower extremity pain. He was a cigarette smoker, and had no history of cardiac disease; he was receiving chronic antiretroviral therapy. After blood samples were drawn for laboratory studies, intravenous heparin was started. At physical examination, normal arterial pulses were present in the right (unaffected) leg, but no pulses or Doppler signals were found below the popliteal artery in the left leg. The left foot was cyanotic and markedly cooler than the right foot. An electrocardiogram showed normal sinus rhythm. Hemoglobin was 16.8 g/dL; white blood cell count, 6900/μL; and platelet count, 204,000/μL. International normalized ratio (INR) and partial thromboplastin time (PTT) were within normal laboratory limits.

An arteriogram demonstrated complete occlusion of the distal popliteal artery, with delayed reconstitution of the anterior tibial artery (Figs 1 and 2). The patient underwent percutaneous mechanical thrombectomy (Angiojet; Possis Medical, Minneapolis, Minn) with an 0.018-inch system. Two units of retaplase (Centecor, Alvern, PA) were added to the saline solution infusion bag. Intravenous abciximab (Eli Lilly, Indianapolis, Ind) was begun during the procedure, in addition to continuous heparin infusion. Subsequently the foot was warm with strong Doppler signals, although no pulses were palpable. The abciximab was continued for 12 hours, and the patient was subsequently discharged with warfarin sodium maintenance therapy.

An echocardiogram showed no thrombus, normal ventricular function, normal valves, and no patent foramen ovale. Protein C, protein S, lupuslike anticoagulant, antiphospholipid antibody, antithrombin, activated protein C resistance, homocysteine, and PT G202010A levels were all within normal laboratory limits. At outpatient evaluation 1 month post-procedure, the patient had persistent hypersensitivity of the dorsum of the foot and pain with weight bearing. Ankle-brachial index in the affected limb was 0.91, compared with more than 1.0 in the contralateral extremity, consistent with residual thrombus. Despite slightly decreased perfusion, the left foot had returned to its normal color and warmth.

DISCUSSION

This unusual case of acute limb-threatening ischemia secondary to spontaneous arterial thrombosis in a patient with HIV infection is distinctive in both cause and treatment with percutaneous mechanical methods for rapid and successful revascularization.

Arterial macroembolism in the lower extremities is most commonly due to a cardiac source of embolic debris. Patients often have a history of cardiac disease, with a precipitating event such as atrial fibrillation, or have limb-threatening ischemia as the initial sign of atrial myxomatous disease. Acute thrombosis as the initial manifestation of

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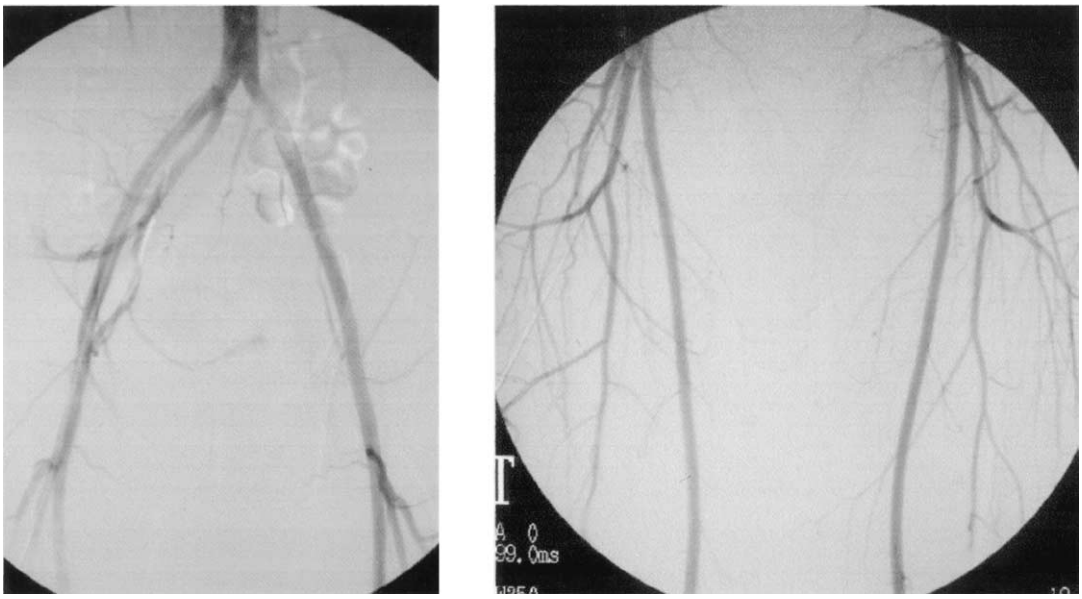

Fig 1. Normal aortoiliac and superficial femoral arteries in 37-year-old patient with acute left lower limb ischemia.

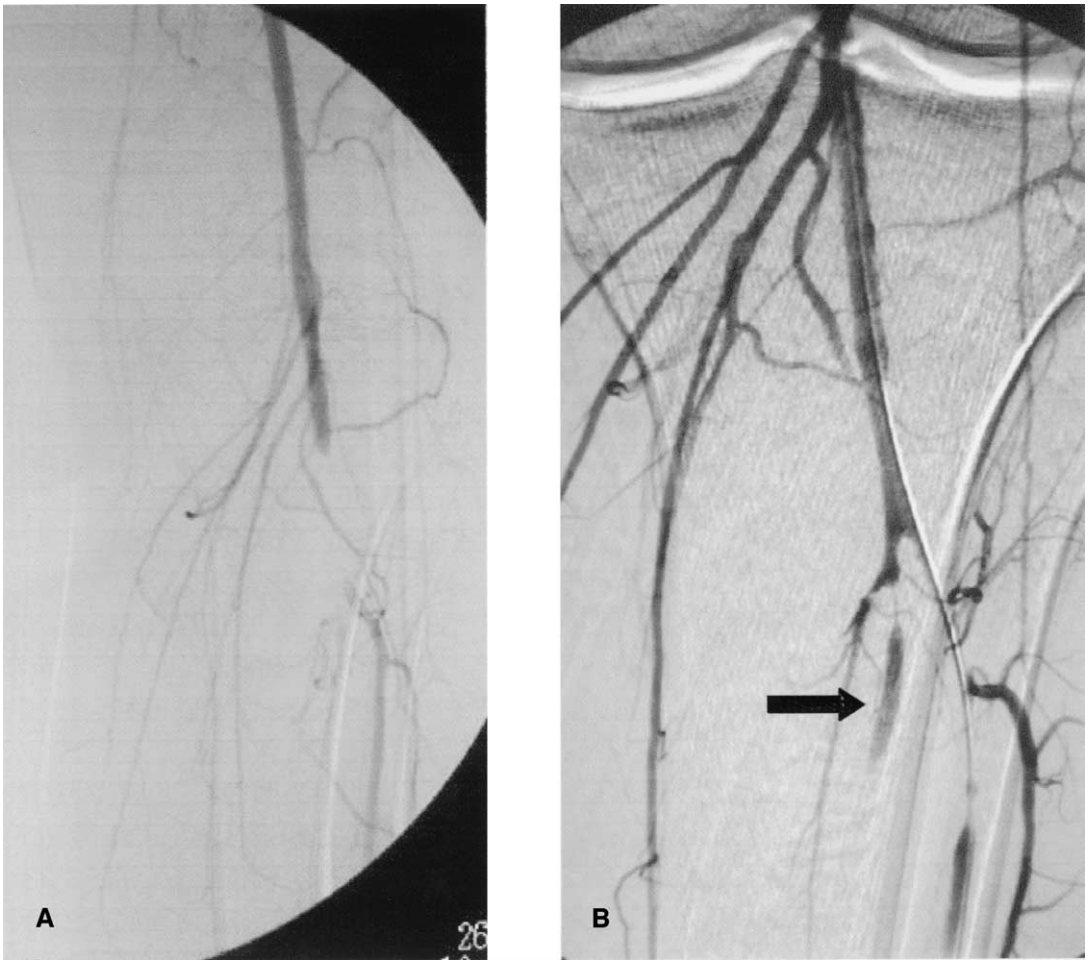

Fig 2. **A**, Abrupt occlusion of distal popliteal artery with reconstitution of anterior tibial vessel. **B**, Tip of percutaneous mechanical thrombectomy device (*arrow*) with improved filling of tibial branches after a few passes with the device.

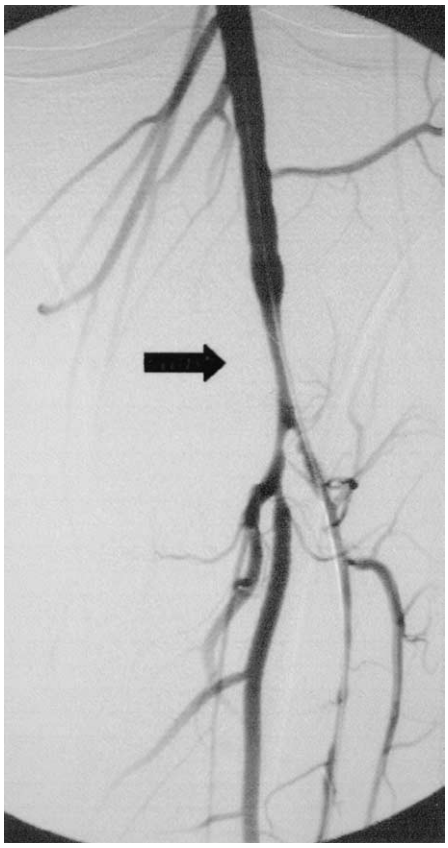

Fig 3. Further improvement in distal circulation with resultant vasospasm (hourglass appearance to vessel, *arrow*) secondary to guide wire and thrombectomy device passage.

HIV infection has been reported.[1] In the absence of any identifiable source, hematologic studies are performed in search of a hypercoagulable state.

Multiple coagulation and hematologic abnormalities have been reported in patients infected with HIV.[2] Various cytopenias and factor deficiencies may be secondary to the direct effects of HIV infection or occur in response to a specific drug therapy. Hypercoagulability, opportunistic infections, and malignancies may increase sensitivity of the patient with HIV to thrombotic events.[3] More specifically, the occurrence of anticardiolipin antibodies, protein S deficiency, antithrombin deficiency, and elevated D-dimer levels have previously been documented in HIV- infected patients, suggesting the presence of a pre-thrombotic state. Documented thromboses have been described in both the venous and arterial systems in patients with HIV, with several series reporting as much as 10 times greater risk for deep venous thrombosis in these patients.[3,4]

There is active research in search of the pathogenesis for the hematologic abnormalities in patients with HIV. In all probability, the mechanism is 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.[5] Furthermore, reduction in CD4 cell count to less than 200 cells/mm^3 has been observed in patients with thromboembolic complications.[5] The CD4 count in our patient was more than 400 cells/mm^3, above the level correlated with a predisposition to thromboembolism in other studies.[5] However, as no antecedent risk factors for arterial thrombosis were identified, we must surmise that either degree of immunosuppression was heightened or there existed a concurrent opportunistic infection, neoplasm, or other inflammatory disease, which was not apparent at presentation.

The evaluation and treatment of acute thrombosis must be prompt and aggressive. Arteriography offers an opportunity to both localize and document the site of thrombosis or embolism; in addition, percutaneous methods are available for rapid clot extraction. We chose to perform thrombus removal with a rheolytic thrombectomy catheter (AngioJet). We consider this to be a rapid and efficient technique for both acutely thrombosed native leg arteries and bypass grafts. The device works via the Venturi effect. Rapidly flowing saline solution jets (8000 psi) are directed backward from the tip of the device, not outward to the vessel wall. The thrombus is drawn into the vacuum created by these rapidly flowing jets, then out the catheter to a collection bag. Any endothelial damage is probably from the thrombus or from basic catheter and wire manipulation. Furthermore, the AngioJet catheter enables declotting of smaller, more distal vessels through one percutaneous access site.

Advantages of percutaneous mechanical thrombectomy for treatment of acute limb ischemia have been demonstrated, with initial thrombus removal rate in excess of 95%.[6] Retaplase (2 units) was added to the infusion solution for the catheter. There are no data as to whether the adjunctive tissue plasminogen activator augments clot extraction. Even so, pharmacologic thrombolysis may aid in resolving occlusion of vessels more distal to the reach of the thrombectomy device. Nonetheless, as a result of using a mechanical thrombectomy device, a low dose of thrombolytic agent without a prolonged infusion period may have assisted in attaining almost complete thrombus resolution.

In conclusion, this case illustrates arterial ischemia caused by spontaneous thrombosis in a patient with HIV infection. Whether the pathogenesis for this event was heightened immunosuppression, an antiretroviral medication complication, or idiopathic thrombosis was not evident. The clinical diagnosis of limb-threatening ischemia was prompt, as was intervention. Percutaneous mechanical thrombectomy with adjunctive thrombolysis restored lower extremity arterial circulation. The duration of necessary anticoagulation is unknown, because HIV infection in this patient may be the foremost predisposing factor to thromboembolism. Furthermore, with increasing numbers of patients surviving over the long term with active HIV infection, thromboembolic complications may become more pronounced in this population (Figs 1-3).

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