Surgery for long-term hemodialysis access is one of the most common procedures performed by vascular and transplantation surgeons. Long-term durability is best achieved through primary arteriovenous fistulas and autogenous arteriovenous grafts. Many patients, however, do not have a suitable conduit for autogenous construction, or adequate maturation of the fistula fails to develop. These patients require prosthetic or xenograft conduits with a variety of arteriovenous anastomoses in a loop or straight bridge-type configuration. A number of complications have been described for both fistulas and grafts, namely, early thrombosis, pseudoaneurysms, aneurysmal degeneration, distal steal, and infection.1 Although thrombosis of prosthetic conduits occurs frequently, surgeons have not typically been concerned about them as a source of late distal embolization. In this paper, we report a rare and never previously described complication of distal thromboemboli from a chronically occluded arteriovenous graft that was implanted 10 years before and appeared as acute hand ischemia. (J Vasc Surg 2000;32:1229-31.)

Case Report

The patient is a 44-year-old right-handed woman with end-stage renal disease resulting from Alport syndrome who presented with approximately 24 hours of waxing and waning right-hand coolness and rest pain. The pain started on the morning of presentation, and the patient attributed it to sleeping on that arm. Over the course of the day, she began having increasing pain and pallor in her forearm that was exacerbated by physical activity. Her surgical history is notable for multiple arteriovenous grafts in both arms and a successful cadaveric renal transplantation 2 years previously. She no longer requires hemodialysis. In 1991 she underwent placement of a right forearm brachial-axillary loop EPTFE graft, which thrombosed shortly after its construction. This was followed by an upper arm brachial-axillary EPTFE bridge graft in the same arm, which thrombosed after approximately 12 months. The patient was otherwise in good health, without a history of myocardial infarction or arrhythmias. Her only medications included baseline, low-level immunosuppressive therapy.

During physical examination, the patient’s right hand was cooler than the left and had markedly delayed capillary refill. She had a palpable brachial pulse at the elbow crease, but no ulnar pulse, and had a water hammer-type radial pulse just proximal to the wrist. The results from an Allen’s test were positive for both ulnar and radial arteries (ie, no palmar reperfusion after the release of either radial or ulnar arteries). Handheld Doppler scan interrogation confirmed the physical findings and demonstrated a weakly monophasic ulnar signal and an obstructed signal in the radial artery. The patient was admitted for immediate intravenous heparin anticoagulation and arm aortography with a selective right upper extremity arteriogram (Fig 1). This demonstrated two separate thromboembolic lesions, one just distal to the arterial anastomosis of the upper arm arte-
riovenous graft and another one located in the proximal ulnar artery just beyond the bifurcation. No obvious proximal arch lesion was identified. The patient underwent recombinant tissue plasminogen activator (rt-PA) (alteplase; Genentech, South San Francisco, Calif), catheter-directed thrombolysis, but after 4 hours it failed to demonstrate any visible, radiographic, or clinical improvement. Although it is not currently approved by the Food and Drug Administration for use in the peripheral circulation, rt-PA has essentially replaced urokinase, following its withdrawal from the market. Our institutional protocol involves a continuous infusion of 1:100 dilution (0.01 mg/mL) of rt-PA in normal saline at 0.25 to 0.5 mg/h through a multi-sidehole catheter. Concomitant heparin infusion is maintained subtherapeutically at 500 U/h, and follow-up angiography is performed in 4 to 6 hours to determine interval progress. If no improvement is detected, as was in this patient, lysis is terminated.

The patient was taken to the operating room, and while she was under general anesthesia, the distal brachial artery, the radial-ulnar bifurcation, and the arterial anastomoses of the upper arm and forearm arteriovenous grafts were exposed through a single sigmoid incision centered near the elbow crease. Both arterial anastomoses were taken down, and this demonstrated thromboses of the grafts with chronic adherent thrombus protruding into the upper arm brachial anastomosis, almost like a stalactite (Fig 1, A). Embolectomy of the brachial artery and ulnar and radial branches extracted old organized thrombus similar in appearance to the thrombus present at the brachial artery anastomosis. The arteriotomies were repaired with saphenous vein patch angioplasties. After restoration of blood flow, an ulnar pulse was palpable at the wrist, and the water hammer radial pulse was markedly diminished. The hand was pink and warm and had excellent capillary refill. The patient’s postoperative recovery was uneventful, and she has had complete resolution of her ischemic signs and symptoms.

**DISCUSSION**

This case illustrates an unusual complication of late distal embolization from a chronically thrombosed arteriovenous graft. Distal ischemic complications of arteriovenous grafts are often the result of an arterial steal syndrome. This occurs in approximately 1.8% and 4.3% of direct arteriovenous fistulas and arteriovenous grafts, respectively. Typically, symptoms occur within the first year after construction of the shunt and can be severe enough to result in chronic rest pain and tissue loss. Depending on the severity of symptoms, a steal phenomenon can be managed with observation, banding, ligation, or performance of a distal revascularization-interval ligation procedure.
Once a graft becomes thrombosed, either after the first operation or after multiple previous revisions, and is no longer considered salvageable, it is left in place, and as with other nonfunctioning prosthetic grafts, no attempt is made to disconnect it from the arterial circulation. Conventional teaching has regarded these chronically thrombosed grafts to have a benign natural history, and their removal has been considered unnecessary. As this report demonstrates, this is not always the case. Our patient’s arteriovenous grafts were implanted nearly 10 years earlier and had been thrombosed for more than 8 years. Digital subtraction angiography with delayed images demonstrated evidence of recurrent digital emboli without a central aortic/cardiac source, and gross intraoperative findings strongly suggested the origin of the emboli to be the upper arm graft (Fig 2). We suspect that the current events may represent a short-term–on–long-term thromboembolic process exacerbated by extrinsic compression of the arm while the patient was sleeping.

In a review of the medical literature, we have not found any references to this type of complication. There is one report in the French medical literature of distal arterial emboli from a direct arteriovenous fistula that had thrombosed.4 In this instance, the arterialized vein had developed significant aneurysmal degeneration, and the emboli originated from the intramural thrombus within the aneurysmal venous segment. Upper extremity emboli have also been reported from thrombosed axillofemoral arterial bypass grafts. In one study, up to 25% of occluded axillofemoral grafts developed distal upper extremity thromboembolic events.5 On the basis of this, prophylactic detachment of thrombosed axillofemoral grafts has been recommended.

In summary, although we do not recommend routine detachment or removal of thrombosed arteriovenous grafts, distal thromboembolization from the graft may occur. It should be included in the differential diagnosis of acute hand or digital ischemia in patients with existing chronically occluded grafts.

REFERENCES

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