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Aneurysmal degeneration of vein conduit used for vascular reconstruction—Case report and literature review

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ABSTRACT

INTRODUCTION: Popliteal artery aneurysms (PAA) are the most prevalent form of peripheral arterial aneurysms. Greater saphenous vein grafts and endoaneurysmorrhaphy remains the mainstay therapy for open repair of PAA. True aneurysmal degeneration of lower extremity infrainguinal autologous vein grafts are relatively rare and its etiology is not completely understood.

CASE PRESENTATION: We present a case of a 57-year-old man with recurrent autologous venous graft aneurysmal dilatations following a surgical popliteal artery aneurysm repair.

DISCUSSION: The pathogenesis of true aneurysmal graft dilatation remains speculative with possible pathogenesis including progression of underlying atherosclerosis, systemic dilating diathesis, autologous venous graft varicosities, low-grade infections and post-stenotic dilatations. Management of venous graft aneurysms should be subjected to the same criteria as other aneurysms. Diagnosis requires a high index of suspicion. The initial study of choice is duplex ultrasonography as it can diagnose the aneurysm and distinguish it from other popliteal masses, provide accurately measurements and identify thrombus within the aneurysm. Once diagnosed, surgical repair should be performed as soon as possible as graft dilatation tends to occur overtime and is typically followed by a rapid increase in size over a short period of time.

CONCLUSION: Aneurysmal degeneration of autologous saphenous venous graft following PAA repairs occur infrequently. Its etiology remains largely speculative. Accurate diagnosis and early surgical intervention can prevent progression of aneurysmal dilatation and minimize the potential of complications.

1. Introduction

Popliteal artery aneurysms (PAA) are the most prevalent form of peripheral arterial aneurysms, accounting for more than 70% of all peripheral aneurysms [1]. The standard operative management involves surgical bypass of the aneurysmal segment with ligation of the popliteal artery proximal and distal to the dilatation. Greater saphenous vein grafts and endoaneurysmorrhaphy remains the mainstay therapy for open repair of PAA as it is considered effective and long-lasting [2]. True aneurysmal degeneration of lower extremity infrainguinal autologous vein grafts are relatively rare and its etiology is not completely understood. In this study we describe a case of recurrent true aneurysm formation in the autologous vein graft secondary to an open popliteal aneurysm repair followed by a review of current literature.

2. Case presentation

Six years ago, a 57-year-old man came to our institution with an acute thrombosed right popliteal aneurysm. A reversed saphenous vein graft was implanted. His past medical history was remarkable for longstanding arterial hypertension and heavy cigarette smoking. In addition, he was found to have a 4.5 cm abdominal aortic aneurysm (AAA) on his initial presentation, which was managed with regular surveillance. No known history of diabetes mellitus, hypercholesterolemia or dyslipidemia. Four years post-operatively, he developed sudden rest pain on his right leg which was found to be secondary to graft thrombosis. The patient underwent thrombolysis, which resulted in complete recanalization. In the vein graft, a true aneurysm situated 6 centimeters proximal to the popliteal artery anastomosis measuring 1.6 cm in diameter had developed. This was treated by resection and end to end anastomosis due to laxity in the vein graft. Two years later, he was noted on follow up ultrasound to have developed progressive dilation in the mid right vein graft. Again, a true aneurysm 2.8 cm in diameter had developed (Figs. 1 and 2). The aneurysmatic segment (Fig. 3) was resected and replaced by an interposition basilic vein graft (Fig. 4). By means of histopathologic examination of the explanted aneurysm, the

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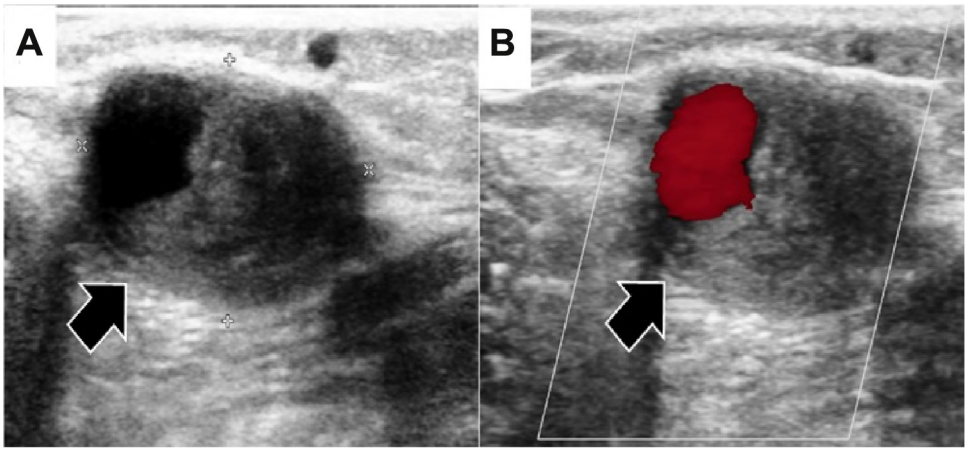

Fig. 1. Aneurysmal degeneration of lower extremity autologous vein graft demonstrated on grayscale (A) and color (B) ultrasound.

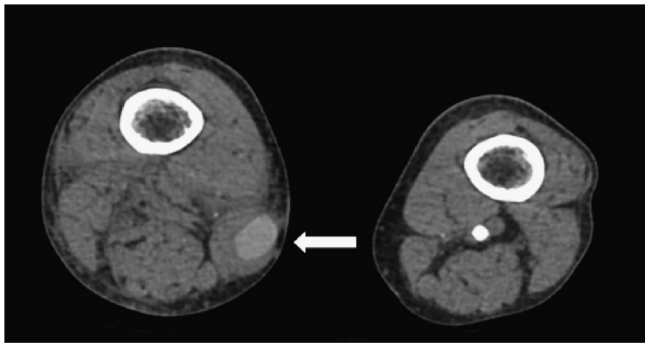

Fig. 2. Aneurysmal degeneration of lower extremity autologous vein graft (arrow).

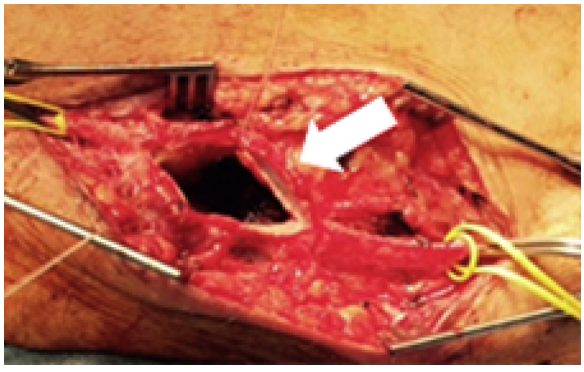

Fig. 3. Aneurysmal degeneration of lower extremity autologous vein graft at surgery. The aneurysm sac has been opened (arrow).

Fig. 4. The aneurysmatic segment was resected and replaced by an interposition basilic vein graft (arrow).

structural architecture of the vessel wall demonstrated fibrocellular intimal thickening. Recovery was uneventful, and the patient was included in a follow-up program with regular graft mapping by means of color duplex Doppler ultrasound scan every 6 months.

3. Discussion

Popliteal artery aneurysms account for most peripheral arterial aneurysms and are potentially dangerous as the five-year cumulative risk of complication is up to 68% [3]. Significant complications include acute thrombosis, aneurysmal occlusion, local pressure effects, aneurysmal rupture and distal embolization. Given its severity, symptomatic PAA should be repaired irrespective of size as the incidence of limb loss increases with the onset of symptomatic disease [1].

Greater saphenous vein grafts and endoaneurysmorrhaphy remain the gold standard for open PAA repairs [2]. Typically a medial or posterior surgical approach is performed. The greater saphenous vein is the most widely used conduit for arterial bypass as most data from literature indicate superior long-term patency of vein grafts compared to prosthetic grafts. In a systemic review of literature that included 2445 PAA, the 5-year patency was 77–100% for vein grafts versus 29% to 74% for prosthetic grafts [1].

Histologically, arterialized autologous veins are subjected to degenerative changes due to its structural differences to arteries. True aneurysmal degeneration of saphenous vein grafts (SVG) however is a rare but hazardous complication following infrainguinal arterial revascularizations [4,5]. The pathogenesis of true aneurysmal graft dilatation remains speculative. It has been suggested that vein graft aneurysms are a direct consequence of advanced atherosclerotic changes in the vein wall as subendothelial cholesterol deposition, foamy macrophages, ulceration and obliteration of elastic lamina, and fibromuscular thickening of the intima have been seen in histopathologic specimens [4–6]. Such micromorphologic changes cause weakening of the vascular wall leading to possible aneurysmal dilatation [7].

Nevertheless, additional etiologic factors should be considered as atherosclerosis is not universally observed given that approximately 30–50% of arterialized veins are affected by atherosclerosis whereas vein graft aneurysms represent a rare entity [7]. In fact, several studies have described non-atherosclerotic aneurysmal formation in autologous saphenous vein grafts suggesting that atherosclerosis may be a facilitating factor for aneurysm development or simply coincidental [6,8].

Systemic arterial aneurysmal disease is a risk factor that has been linked to venous graft aneurysms. In one study, it was found