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



M.J. Tao, W. Al-Jundi, G. Roche-Nagle*

Department of Vascular Surgery, Toronto General Hospital, Toronto, ON, M5G 2C4, Canada

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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.

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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

* Corresponding author at: Department of Vascular Surgery, Toronto General Hospital, 200 Elizabeth St. Toronto, ON, M5G 2C4, Canada.

E-mail address: graham.roche-nagle@uhn.ca (G. Roche-Nagle).

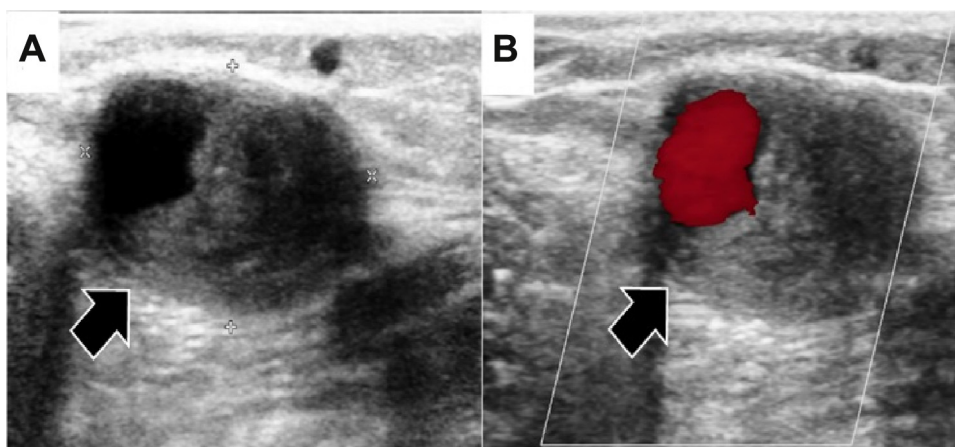


Fig. 1. Aneurysmal degeneration of lower extremity autologous vein graft demonstrated on grayscale (A) and color (B) ultrasound.

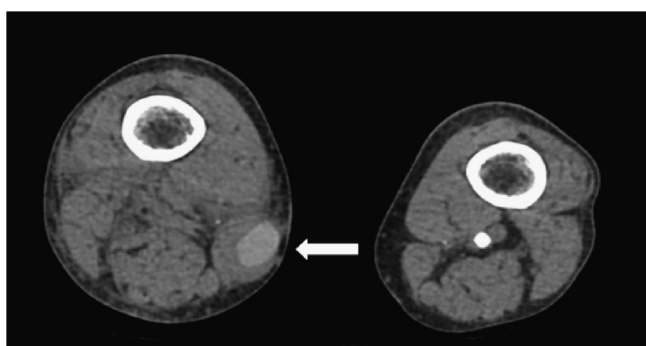


Fig. 2. Aneurysmal degeneration of lower extremity autologous vein graft (arrow).

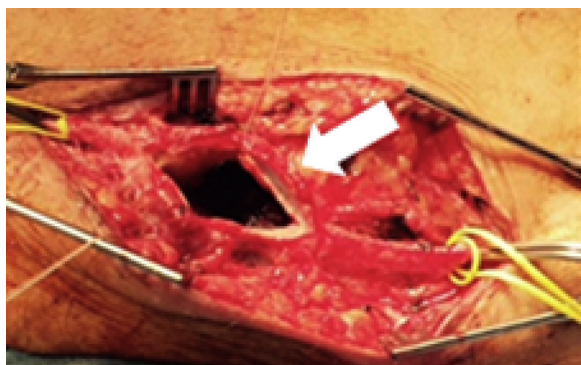


Fig. 3. Aneurysmal degeneration of lower extremity autologous vein graft at surgery. The aneurysm sac has been opened (arrow).



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

that saphenous vein grafts implanted to bypass PAA are of significantly larger diameter than those implanted to treat peripheral arterial occlusive disease [9]. Furthermore, Loftus et al. demonstrated a 42% incidence of vein graft aneurysms for patients with PAA compared to 2% incidence in patients with lower limb arterial occlusive disease [10] which suggests that dilatation of vein grafts may be a manifestation of systemic predisposition towards aneurysm formation. Such systemic dilating process may involve biochemical changes including proteolytic degradation of vessel wall connective tissue, inflammation infiltration, medial degeneration, biomechanical wall stress, and increased levels of matrix metalloproteinase [11,12]. Our patient's primary diagnosis was popliteal artery aneurysm and he developed aneurysmal degeneration of his vein graft on two occasions, which raises the suspicion that an underlying systemic dilating mechanism exists in his case. In addition, our patient had coexisting AAA. A recent prospective study of patients who underwent PAA vein bypass revealed that all patients who developed graft aneurysms had aneurysmal disease elsewhere compared with 45% of patients who did not develop graft aneurysms [13]. Therefore, it may be argued that patients with aneurysms at other sites and those who develop vein graft aneurysm may require life-long postoperative graft surveillance. Other proposed mechanism of graft aneurysm development include weakness at valve sites, 'blow-outs' at side-branches, mycotic aneurysm following low-grade graft infections, post-stenotic dilatation and varicosities in venous grafts [5,7].

Smoking may be linked to aneurysmal dilatation as recent research provides a convincing link between abdominal aortic aneurysm formation and nicotine, the major culprit of cigarette smoke. Using mouse models, Wang et al. found that nicotine stimulates the $\alpha 2$ isoform of AMP-activated protein kinase in vascular smooth muscle cells which in turn phosphorylates a transcription factor that drives the expression of matrix metalloproteinase 2, a matrix-degrading protein that weakens the artery wall [14]. A similar mechanism on vein graft aneurysms has not been investigated yet. Nevertheless, smoking cessation is recommended for all vascular patients.

Management of venous graft aneurysms should be subjected to the same criteria as other aneurysms. Diagnosis requires a high index of suspicion. Aneurysmal formation in venous grafts following PAA repair may be asymptomatic or may lead to localized pain, swelling, pressure necrosis, and occasionally rupture [15]. The differential diagnosis should include anastomotic false aneurysm as it is a more common complication following bypass revascularization compared to true venous graft aneurysmal dilatations. Physical examination may reveal an expansive popliteal pulse at the popliteal fossa. 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. Arteriography is a valuable modality in evaluating inflow and outflow at the lesion.

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 [7]. The type of surgical intervention depends on the extent of aneurysmal dilatation as diffuse disease dictates complete graft replacement whereas aneurysm resection with preservation of graft and autologous or prosthetic interposition may be achieved in isolated aneurysms [16]. Endovascular placement of expandable polytetrafluoroethylene-covered nitinol endoprosthesis has been used as an alternative to open surgery in rare cases, however the long-term patency and efficiency are unknown to date [17].

With accurate diagnosis and proper surgical management, excellent limb salvage can be expected and postoperative function should be achieved following venous graft aneurysm repairs. Life-

long ultrasound surveillance is warranted due to the propensity for recurrence of vascular dilatations.

4. Conclusion

Aneurysmal degeneration of autologous saphenous venous graft following PAA repairs occur infrequently. Its etiology remains largely speculative with possible pathogenesis including progression of underlying atherosclerosis, systemic dilating diathesis, autologous venous graft varicosities, low-grade infections and post-stenotic dilatations. Our case highlights that patients who develop venous graft aneurysms on one occasion are susceptible to develop recurrent graft aneurysms. We suggest that such patients require life-long graft surveillance as early diagnosis and surgical intervention can prevent progression of dilatation and minimize potential complications. Presented case has been reported in line with the SCARE criteria [18].

Conflicts of interest

None.

Sources of funding

None.

Ethical approval

N/A.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution

The idea of the project was conceived by Graham Roche-Nagle. Mary Jiayi Tao carried out the literature review. The paper was written and reviewed by Mary Jiayi Tao and Wissam Al-Jundi. All authors contributed to the refinement of the case report and approved the final manuscript. Graham Roche-Nagle was the senior consultant in charge of this case.

Registration of research studies

N/A.

Guarantor

Graham Roche-Nagle.

Consent

Informed consent was obtained and documented.

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