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A case report and review of the literature

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Non-anastomotic aneurysmal degeneration of great saphenous vein graft. A case report and review of the literature

INTRODUCTION: *True aneurysmal degeneration of autogenous vein grafts is unusual, despite their widespread use as arterial substitutes. We report a case of acute lower leg ischemia due to thrombosis of a non-anastomotic venous aneurysm.*
CASE REPORT: *A 71-year old man presented at Emergency Unit at our Institution with left lower limb acute ischemia. The patient had 9 years before undergone left below the knee femoro-popliteal bypass with in situ autologous great saphenous vein (GSV) graft. Doppler ultrasound exam revealed patency of the entire conduit, and subtotal acute thrombosis of a saccular aneurysm of the vein 2 cm before the distal anastomosis of the graft. Aneurysmectomy and distal thrombo-embolectomy with Fogarty catheter of below the knee popliteal artery and tibio-peroneal trunk was performed. Vein graft continuity was restored by a termino-terminal anastomosis.*
DISCUSSION: *Arterialized autologous veins are at risk of degenerative changes because of histological differences with arteries, but the cause of true aneurysmal degeneration of these grafts is still unknown. The mean time from graft implantation to clinical manifestation of the aneurysm is 7 years and the management of venous graft aneurysms should be subjected to the same criteria as other aneurysms. The first choice in detecting vein graft aneurysms is Duplex ultrasonography and the type of surgical intervention depends on the cause, type and extension of aneurysmal dilatation.*
CONCLUSIONS: *Aneurysmal degeneration of deep lower extremity vein conduits implanted for vascular reconstruction has been rarely reported, but when detected they can lead to graft thrombosis, distal embolization, acute rupture, or skin ulceration. Therefore, Doppler ultrasound guided surveillance of GSV grafts should be mandatory and long time from vein graft creation to onset of aneurysms makes long-term graft surveillance even more imperative.*

KEY WORDS: Acute Leg Ischemia, Great Saphenous Vein, Graft; Aneurysm, Thrombosis

Introduction

True aneurysmal degeneration of autogenous vein grafts is described in literature as an unusual event, despite their widespread use as arterial substitutes. Various studies have underlined the superiority of autogenous veins over synthetic prosthetic grafts in below knee infra-inguinal bypass surgery, with excellent long-term patency, making the autologous great saphenous vein (GSV) the preferred graft of use in peripheral bypass surgery whenever possible [1-4].

True aneurysm formation in infra-inguinal autologous vein grafts is regarded as an uncommon complication, but its true incidence remains unknown [5-7]. Also the etiology of vein graft aneurysms remains uncertain, with proposed etiologies including atherosclerosis, infections, tobacco consumption, generalized predisposition to aneurysmal disease and technical factors. [8]

Aneurysmal degeneration of vein grafts most commonly involves thin-walled pseudoaneurysms at anastomotic sites. [9] True aneurysmal degeneration of autogenous vein grafts has been most often reported in literature after

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great saphenous vein (GSV) coronary artery bypass or after implantation of renal artery grafts, especially in children. [10-12] Even rarer are aneurysms affecting autogenous GSV grafts used for infrainguinal bypasses. [13-15].
We report a case of non-anastomotic vein graft aneurysm formation in a patient who underwent 9 years before left femoro-popliteal bypass using in situ great saphenous vein.

Case Report

A 71-year old man presented at Emergency Unit at our Institution with left lower limb acute ischemia. His past medical history was remarkable only for hypertension, hypercholesterolemia and tobacco abuse, without any prior history of aneurysmal artery disease. The patient had previously undergone left below the knee femoro-popliteal bypass 9 years before with in situ autologous great saphenous vein (GSV) graft for severe rest pain, performed after several endovascular failed procedures of recanalization of left superficial femoral and popliteal artery.
The patient referred a progressive reduction of his walking distance during the last four weeks and an acute onset of paleness and sudden reduction just to few meters in the last day. Peripheral vascular clinical examination revealed an acute ischemia of the leg, with paleness and pulselessness at the ankle.
The vein along the entire extension of its inter-fascial course was easily detectable with normal pulsatility without any pathological mass. Blood tests were normal as well.
Doppler ultrasound showed good direct biphasic common femoral artery flow measured at the proximal anastomosis of the femoro-popliteal bypass, the patency of the entire GSV graft and the presence of a non-anastomotic saccular aneurysm of the vein (maximum diameter of 30 mm), approximately 2 cm before the distal anastomosis of the graft. The aneurism showed an almost complete thrombosis, with post-occlusive demodulated flow pattern in the posterior tibial and peroneal artery (chronic occlusion of anterior tibial artery).
He firstly underwent surgical exploration of the distal anastomosis of the graft with aneurysmectomy of the recent thrombosed vein graft aneurysm (gaining optimal pulsed blood inflow from the proximal part of the saphenous graft) (Fig. 1). Subsequent thrombo-embolectomy with Fogarty catheter of below the knee popliteal artery and tibio-peroneal trunk was made, with removal of small quantity of recent thrombus gaining good backflow (Fig. 2). Finally vein graft continuity was restored by performing a termino-terminal anastomosis between two stumps of the vein maintaining previous distal anastomosis (no mio-intimal hyperplasia was found at this level) (Fig. 3). At the end of the procedure, intra-operative Doppler Ultrasound showed a direct triphasic flow in posterior tibial artery at ankle with the presence of a good palpable pulse. The patient was discharged in satisfactory conditions 4 days later on a regimen of single oral antiplatelet therapy (75 mg of Clopidogrel once a day).

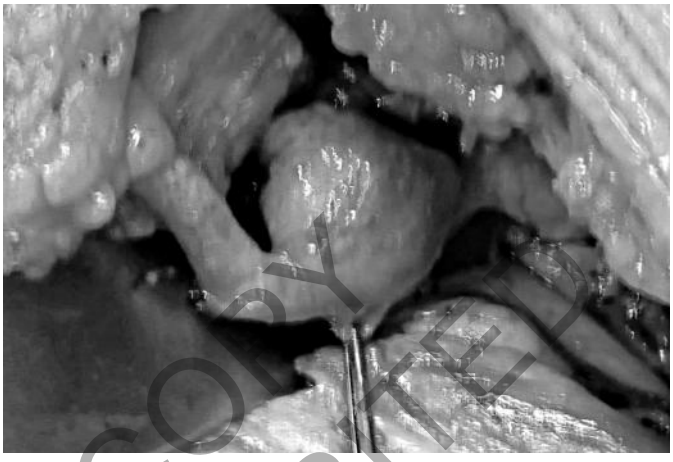

Fig. 1: The surgical exploration of the distal anastomosis demonstrated the presence of a big preanastomotic aneurysm.

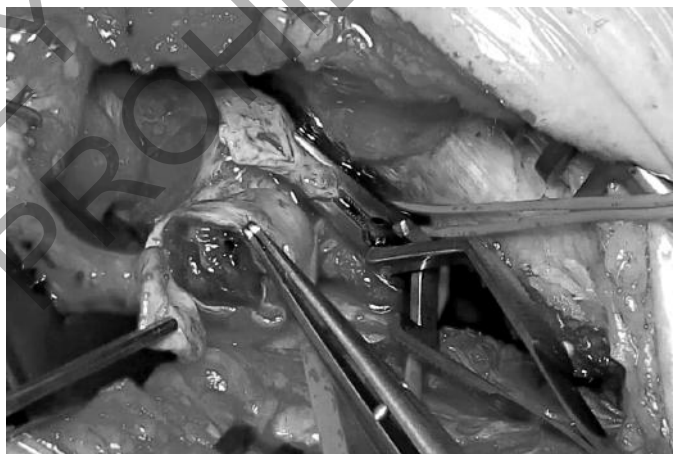

Fig. 2: The aperture of the aneurysm showed a stump thrombus.

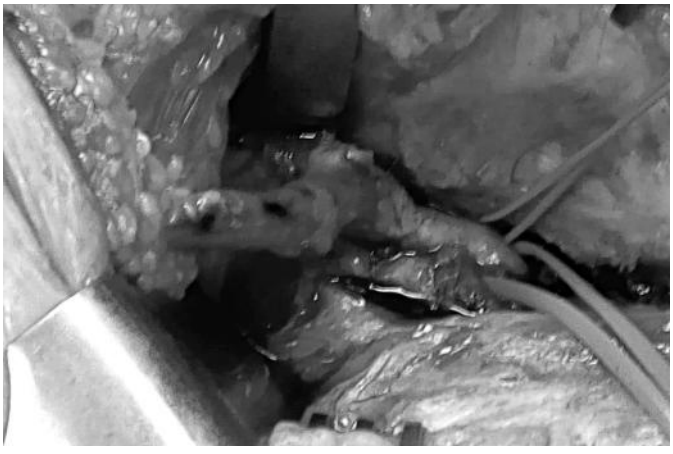

Fig. 3: The vein graft continuity was restored by performing a termino-terminal anastomosis between two stumps of the vein maintaining previous distal anastomosis.

Discussion

True aneurysmal degeneration of autogenous vein grafts is unusual, despite their widespread use as arterial substitutes. In general terms, arterialized autologous veins are at risk of degenerative changes because of histological differences with arteries. The thickness of the three layers (tunica intima, media and adventitia) varies greatly depending on the type of vessel. The tunica media is the layer of concentrically arranged smooth muscle, the autonomic control of which can alter the diameter of the vessel and affect blood pressure. The tunica media of arteries is larger than that of veins of similar size and makes the veins more susceptible to aneurysmal degeneration. Moreover, the tunica adventitia is made of longitudinally arranged collagen fibers and tends to be much larger in veins than arteries.

In a 10-year review of 295 saphenous vein grafts published in 1967, Darling et al. did not report any cases of vein graft aneurysms [16]. Davidson and De Palma reported this complication for the first time in 1972 [17]. In 1973, Szilagyi et al. reported the fate of saphenous vein grafts in 260 patients and found 10 instances of aneurysms, most of them attributed to trauma during vein preparation [18]. In 1975, Vanttinen and Inberg [19] also noted only 1 aneurysmal formation (0.9%) in 109 femoro-popliteal bypass procedures with autologous saphenous vein grafts and in 2004 Majeski reported his personal series of 207 in situ saphenous femoro-popliteal bypass operations, identifying only 3 true vein graft aneurysms (1.4%) [20].

As described before, true aneurysmal degeneration of autogenous vein grafts implanted in peripheral arteries has been reported only rarely, most often after great saphenous vein (GSV) coronary artery bypass or after implantation of renal artery grafts. Renal artery vein graft aneurysmal degeneration was documented by Stanley et al. including their experience with 72 re-operations among 425 primary reno-vascular reconstructions [21]. Citing vein graft ectasia in 20% of vein grafts, [22] vein graft aneurysms in 6% of adult bypass recipients, and vein graft aneurysms in 20% of pediatric bypass recipients, [23] they considered operative repair of vein graft aneurysms primarily to avoid distal thrombus embolization. In 1999 Lavigne et al. reported a 42-year-old female patient with a ruptured vein graft aneurysm 19 years following bypass [24]. One year later, Travis et al. reported a 75-year-old female patient with a ruptured aneurysmal vein graft 22 years following bypass grafting [25]. Both groups recommended long-term follow-up ultrasonography to surveil well established renal artery vein graft bypasses.

Given its relative rarity, the cause of true aneurysmal degeneration of autogenous vein grafts is unknown. Very rarely, an unusual underlying diagnosis, such as a connective tissue disorder has been reached after pathologic examination [26]. Some investigators have suggested that loss of elastic tissue in vein grafts may predispose to the development of aneurismal changes [27]. Manipulation of the vein during harvest may cause loss of endothelial integrity, with subsequent increase in cellular proliferation and lipid infiltration [28]. It was postulated that the use of in situ grafting could reduce atherosclerotic degeneration of the vein[29], but follow-up studies showed that long-term patency rates are similar for both reversed and in situ saphenous grafts when used in peripheral arteries for below knee infra-inguinal bypass surgery [30,31].

In 1973, De La Rocha et al. reported a case of aneurismal dilatation in a saphenous vein graft 5 years after implantation for lower extremity occlusive disease in the British Journal of Surgery's section entitled "Short notes of rare or obscure cases [14]." Histologic examination revealed extensive atherosclerosis with subintimal fibrosis, giant cell formation, and cholesterol deposition [32,33]. In other reports, pathologic findings have also included lipid-laden macrophages, medial degeneration, and inflammation as well as other atherosclerotic changes in the vein wall [32,33]; however, atherosclerosis affects approximately only 30 to 50% of arterialized veins and vein graft aneurysms represent an unusual complication, as described above. Cigarette smoking, hypercholesterolemia, and hypertriglyceridemia have been also linked to aneurysm development [33]. However, some investigators have reported aneurysms in patients without any such risk factors [34]. Most likely, a combination of factors leads to aneurysm formation, including vessel wall lipid metabolism, hemodynamic factors, and as yet-to-be-determined systemic factors.

Interestingly, vein graft aneurysms are most common when the vein graft was originally implanted for lower extremity aneurysmal disease, raising the possibility of a systemic dilatory process, involving proteolytic degradation of vessel wall connective tissue, inflammation and immune responses, biomechanical wall stress and molecular genetics [23,35,36]. Circumstantial evidence suggests that arterial aneurysms are not due to atherosclerosis but are most likely part of a generalized dilating diathesis. If so, the rate of vein graft aneurysms after leg bypass should be higher in patients with popliteal aneurysms than for patients with lower limb atherosclerosis. Loftus et al. reported their 5-year experience with vein grafts involving 221 revascularization procedures for occlusive disease and 24 for popliteal aneurysms [37]. Graft surveillance revealed spontaneous aneurysm formation in 10 (42%) of the 24 bypass grafts for popliteal aneurysms; however, vein graft aneurysms were only detected in 4 (2%) of the 221 bypass procedures performed for occlusive disease. A long latency period from graft implantation to aneurysmal degeneration was seen in the reported cases, ranging from 3 to 22 years [32-34].

Vein graft aneurysms can also lead to thrombosis [38]. Their management is subject to the same criteria applied to other aneurysms; surgical repair is needed to prevent catastrophic complications. In lower extremities, main

complications are thrombosis and distal thromboembolism. When reconstruction is required, aneurysm excision with an end-to end anastomosis, if feasible (as in our experience), or replacement with the use of autogenous vein or prosthetic graft seems appropriate.
According to the literature, the mean time from graft implantation to clinical manifestation of the aneurysm is 7 years, but graft dilatation was observed as early as 2 years postoperatively [39]. DeWeese reported aneurysm formation 1 year after graft implantation with a postoperative recurrence 5 months later, but infection rather than atherosclerosis was suspected to be the cause of early aneurysms [7].
Management of venous graft aneurysms should be subjected to the same criteria as other aneurysms. Diagnosis requires high index of suspicion. Physical examination may reveal an expansive popliteal pulse at the popliteal fossa or along the course of the vein if it has been used in an "in situ" configuration. Clinical manifestation is mostly characterized by a pulsatile mass, which may lead to skin ulceration, acute rupture, graft thrombosis or distal embolization. The firts choice in detecting vein graft aneurysms 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. Angio-CT scan could be a second level useful exam as well as arteriography, which 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 [5]. 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 [40]. Endovascular placement of expandable polytetraflurorethylene-covered nitinol endoprosthesis has been also described and could represent an alternative to open surgery in selected cases [41].

Conclusions

Aneurysmal degeneration of deep lower extremity vein conduits implanted for vascular reconstruction has been rarely reported and could be considered a rare complication. Ultrasound guided surveillance of GSV grafts should be mandatory; it may not only improve patency by detecting vein graft stenosis, but it will also help to detect graft-related problems, such as aneurysmal dilatation, which could result in limb loss if rupture or thrombosis was to occur. The long lag period from vein graft creation to diagnosis of aneurysms affecting vein grafts makes long-term graft surveillance even more imperative.

Riassunto

Riportiamo il caso assolutamente inusuale di una degenerazione aneurismatica preanastomotica distale di un innesto autologo in vena safena in situ per by-pass femoro-popliteo sottogenicolare, esordito con ischemia acuta d'arto inferiore per trombosi dell'aneurisma. Nonostante la trombosi sub occludente della camera aneurismatica con embolizzazione al tronco tibio-peroniero, l'intero innesto venoso rimase pervio. Venne effettuata una aneurismectomia con embolectomia distale e successivo ripristino della continuità del by-pass mediante anastomosi termino-terminale.
Viene descritto il caso e viene fornita una revisione della Letteratura sullo specifico argomento.

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