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Diagnosis of stenosis within the popliteal–femoral venous segment upon clinical presentation with a venous ulcer and subsequent successful treatment with venoplasty

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Abstract

This case study reports the diagnosis and treatment of a lower limb venous ulcer with abnormal underlying venous pathology. One male patient presented with bilateral varicose veins and a right lower limb ulcer. Upon investigation, full-leg duplex ultrasonography revealed total incompetence of the great saphenous vein in the left leg. In the right leg, duplex ultrasonography showed proximal incompetence of the small saphenous vein, and dilation of the anterior accessory saphenous vein, which remained competent. Incidentally, two venous collaterals connected onto the distal region of both these segments, emerging from a scarred, atrophic popliteal–femoral segment. An interventional radiologist performed venoplasty to this popliteal–femoral venous segment. Intervention was successful and 10 weeks post procedure ulceration healed. Popliteal–femoral venous stenosis may be associated with venous ulceration in some cases and may be successfully treated with balloon venoplasty intervention.

Keywords

Venous reflux, venoplasty, stenosis, popliteal–femoral segment

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Introduction

While venous reflux is the predominant pathophysiological feature of chronic venous disease, venous obstruction also contributes towards the possible spectrum of clinical manifestations; these range from telangiectasia to bulging varices, swelling, severe cases of skin damage, and, in some instances, chronic pain syndromes.[1–5] The most commonly seen and recognised symptom of venous disease is lower limb varicose veins, which are bulging superficial venous tributaries arising from an incompetent truncal vein. Varicose vein formation appears to be the best-case scenario in venous disease; it highlights an underlying medical condition and redirects retrograde blood flow, thereby protecting against a spectrum of possible haemodynamic and dermatological changes in the lower limb. Hyperpigmentation, dermatitis, lipodermatosclerosis, swelling, and, in the most severe cases, venous ulceration may develop; whereby the pooling of blood, inflammatory cascades, and microcirculatory alterations manifest.[1–3]

This case report presents the diagnosis and the treatment of a venous ulcer patient with atypical underlying disease pathology. Duplex ultrasound (DUS) outlined a venous collateral pattern indicative of deep venous obstruction, which was confirmed to be in the femoral vein, and was successfully treated with balloon venoplasty.

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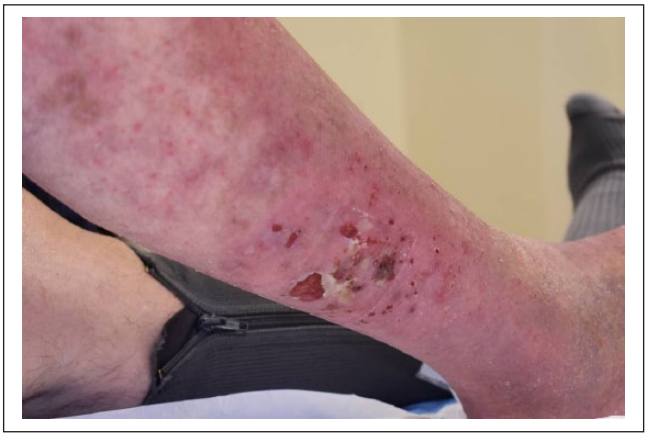

Figure 1. Picture of right leg showing venous ulceration secondary to occlusion in popliteal–femoral venous segment.

Case

One 75-year-old male patient was presented to our specialist vein unit with bilateral skin damage, indicating advanced venous disease. He has no family history of venous disease or of any previous varicose vein surgery. Upon arrival, he received bilateral full-leg DUS.

The left leg venous pattern showed a common manifestation of venous disease; total incompetence of the great saphenous vein (GSV), with some minor emerging tributaries, eligible for endovenous laser ablation (EVLA) treatment. The lower portion of his leg had haemosiderin deposition and was therefore given a Clinical–Etiology–Anatomy–Pathophysiology (CEAP) classification score of 4.

On the right leg, venous disease appeared to have progressed further, presenting as an active ulcer, corresponding to a CEAP classification score of 6 (Figure 1). His right lower leg was swollen with oedema and had considerable haemosiderin deposition. He also had venous claudication with limited mobility. However, DUS showed an abnormal venous pathology, as reflux was only identified in the proximal small saphenous vein (SSV), and, interestingly, the anterior accessory saphenous vein (AASV) was significantly dilated (6 mm), but remained competent. Furthermore, two venous collaterals emerged from the femoral vein, with one connecting onto the distal most region of incompetence on the SSV, and the other directly onto the AASV.

The key pathological feature identified under ultrasound was the presence of thickened vein wall tissue of the deep venous system, located on the proximal popliteal, and the distal femoral vein. The patient has a history of two previous deep vein thrombosis (DVT), one in the 1980's after a laminectomy for a central disc prolapse and the other in 2008 after a flight. The patient has been on anti-coagulation since the second DVT and at the time of presentation was on rivaroxaban. Thickened tissue of the deep venous system is a typical post-thrombotic indication of deep vein obstruction.

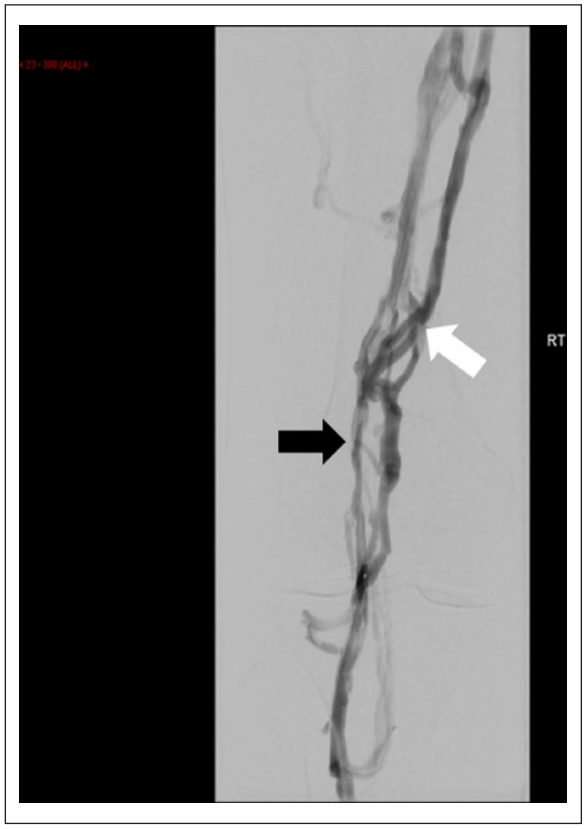

Figure 2. Pre-procedure scan showing the femoral vein and the collateral (black arrow: femoral vein; white arrow: collateral).

Considering the pattern of venous collateral formation, a consultant vascular surgeon together with an interventional radiologist identified this as a possible venous obstruction disorder, with blood flow diversion through the SSV and AASV. DUS ruled out deep vein reflux. Air plethysmography was performed using the venous drainage index; an air cuff was placed on the leg, the leg was then elevated, and the volume difference measured. This ruled out proximal obstruction. This indicated that the popliteal–femoral vein stenosis was obstructing venous return and was therefore responsible for the collateral formation and ulceration. Deep vein stenosis was indicated by the thickened tissue with higher echogenicity of the popliteal–femoral vein segment on DUS. This was confirmed with venography performed by an interventional radiologist. A right common femoral vein puncture was performed and a Tumero wire and support catheter were passed across the scarred stenotic segment (Figure 2). There were no functional valves in the diseased segment. At the level of the emerging collaterals through the atrophic scarred segment, high-pressure balloon angioplasty was performed using a 8 mm × 40 mm and 10 mm × 40 mm Mustang. This was undertaken at 24 atmospheres with no residual wasting of the balloon (Figure 3).

Approximately 6 weeks post procedure, the patient returned for a follow-up examination. DUS identified the

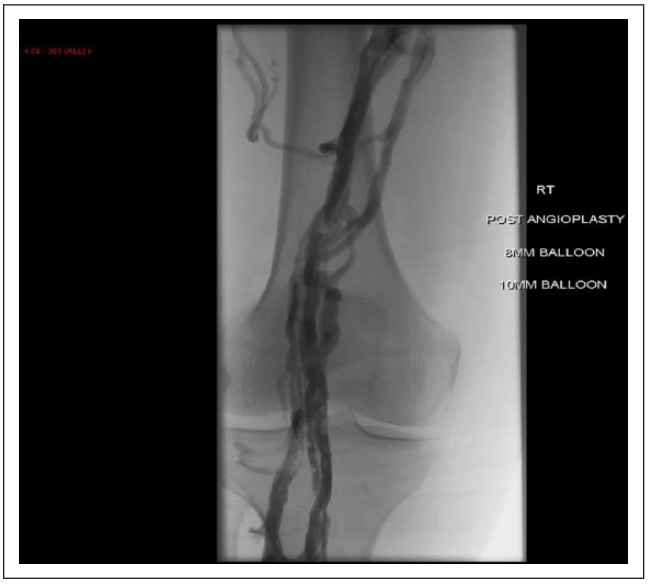

Figure 3. Post-venoplasty venogram following balloon dilatation of popliteal–femoral venous segment.

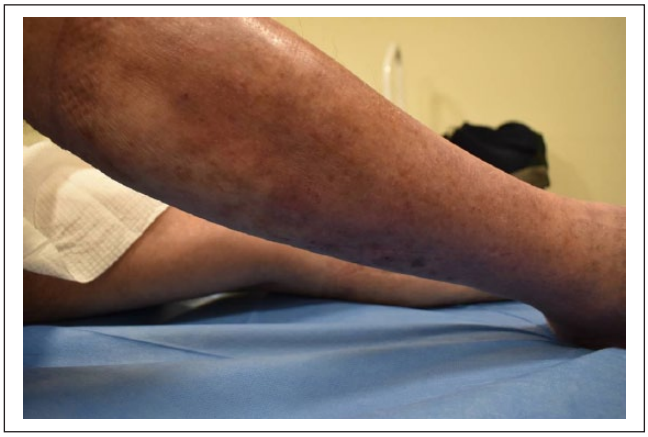

Figure 4. Approximately 10 weeks post procedure, leg shows that the venous leg ulcer has healed and the surrounding skin is less inflamed.

common and superficial femoral veins, and the popliteal vein as patent, while some residual scarring prevailed. This confirmed that no significant deep venous stenosis remained. Despite this, some reflux was again noted within the SSV, and the AASV remained highly dilated. The venous ulcer was in the process of healing, but had failed to heal completely. However, the patient's mobility had significantly improved since the venoplasty intervention and frequent walking was recommended.

Less than a month after this follow-up, the patient returned, and upon additional examination, the ulcer had completely healed (Figure 4). Approximately 10 weeks post procedure, venoplasty with concomitant compression (with class II compression stockings) and exercise resulted in ulcer healing, whereby compression alone had previously failed.

Despite a good result being initially achieved after venoplasty, approximately 7 months after the procedure, the patient developed an open lesion on his right calf. DUS investigations identified a large incompetent perforator vein in the right calf underlying the new ulcer. This was treated with the TRansLuminal Occlusion of Perforator technique (TRLOP); an endovenous laser was passed into the perforator vein under ultrasound control and the vein was treated with a power of 10 W. DUS confirmed closure and the patient received compression to the local area after treatment. A month post procedure, the patient returned for follow-up, and the ulcer had once again healed.

Discussion

Within the last two decades, DUS has enabled significant progression within the field of venous surgery. This investigation permits adequate clinical evaluation of anatomical and physiological aspects of venous disease, predominantly within the superficial saphenous system, but, as outlined here, it may also indicate deep venous pathology. As venous reflux is the predominant pathophysiology of venous disease, surgical intervention within the field typically targets these haemodynamic alterations, and, in the case of ulceration, with the addition of compression therapy to reduce inflammation, and subsequently reverse the microcirculatory changes and tissue breakdown characteristic of a venous ulcer.[1,5]

Inadequate recanalization following DVT has been regarded to be the most common cause of obstruction of venous outflow, manifesting as post-thrombotic syndrome. Even following treatment and subsequent recanalization, it may contribute towards lower limb clinical symptoms.[2,6–12] It has been estimated that over 50% of individuals who have had a previous DVT develop post-thrombotic syndrome,[12] with ulceration developing in around 5%–10% of this cohort.[13] Following DVT, intraluminal fibrotic strands develop, thickening the vein wall, constricting the lumen, and limiting adequate venous drainage.[12,14] Partial recanalization and venous collateral formation are characteristics of a post-thrombotic limb, but provides inadequate blood diversion, as evidenced by the development of skin changes over time, manifesting as swelling, venous eczema, claudication, and venous ulceration.[12]

Research has suggested that the speed of chronic venous disease progression, together with the severity of symptoms, is enhanced in post-thrombotic limbs.[3,6] Enhanced risk of skin damage of a four-fold prevalence has been reported in post-thrombotic limbs relative to primary disease in one study, with other research highlighting the enhanced clinical severity coinciding with the combined impact of reflux and obstruction.[1–3,5–9]

Traditionally, venous stenosis or obstruction was treated invasively through a bypass surgery. This has since largely been replaced by non-invasive percutaneous endovenous stenting, which has been shown to provide relief from the

symptoms associated with both deep venous obstruction and deep venous reflux.[7] This is largely used to overcome iliofemoral venous outflow obstruction,[2,5,7] either for post-thrombotic syndrome or for compression caused by angiectopia, such as in May-Thurner syndrome. Post-thrombotic syndrome more commonly develops within and appears to elicit more severe symptoms with iliac vein and proximal femoral venous involvement.[12,14] The safety and efficacy of percutaneous iliac vein stenting has been reported for the correction of pelvic venous outflow obstruction; one study reported 88% stent patency at 5 years and an active ulcer healing rate of 54% within that same time.[7]

Although it is significantly less common, there are reports of the development of stenosis within the popliteal–femoral venous segments,[12] but treatment of this condition with balloon venoplasty does not appear to be widely reported. Considering the complete healing of the venous ulcer 10 weeks post intervention, this demonstrates two things; first, that DVT may cause femoral obstruction, and second, that this may induce venous ulceration. These preliminary results appear to suggest that lower leg patency appears to be the objective, possibly mimicking arterial ulceration. This study appears to imply that popliteal–femoral stenosis can be corrected by balloon venoplasty; regardless, this case study presents with sample size limitations, and therefore, more research should be conducted into the development and treatment of ulceration from lower leg venous stenosis.

Declaration of conflicting interests

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

References

1. Chi YW and Raffetto JD. Venous leg ulceration pathophysiology and evidence based treatment. *Vasc Med* 2015; 20(2): 168–181.
2. Neglén P, Hollis KC, Olivier J, et al. Stenting of the venous outflow in chronic venous disease: long-term stent-related outcome, clinical, and hemodynamic result. *J Vasc Surg* 2007; 46(5): 979–990.
3. Labropoulos N, Patel PJ, Tiongson JE, et al. Patterns of venous reflux and obstruction in patients with skin damage due to chronic venous disease. *Vasc Endovascular Surg* 2007; 41(1): 33–40.
4. Hobbs JT. The pelvic congestion syndrome. *Br J Hosp Med* 1990; 43(3): 200–206.
5. George R, Verma H, Ram B, et al. The effect of deep venous stenting on healing of lower limb venous ulcers. *Eur J Vasc Endovasc Surg* 2014; 48(3): 330–336.
6. Labropoulos N, Gasparis AP, Pefanis D, et al. Secondary chronic venous disease progresses faster than primary. *J Vasc Surg* 2009; 49(3): 704–710.
7. Raju S, Darcey R and Neglén P. Unexpected major role for venous stenting in deep reflux disease. *J Vasc Surg* 2010; 51(2): 401–408.
8. Labropoulos N, Leon M, Nicolaides AN, et al. Venous reflux in patients with previous deep venous thrombosis: Correlation with ulceration and other symptoms. *J Vasc Surg* 1994; 20(1): 20–26.
9. Neglén P, Thrasher TL and Raju S. Venous outflow obstruction: An underestimated contributor to chronic venous disease. *J Vasc Surg* 2003; 38: 879–885.
10. Johnson BF, Manzo RA, Bergelin RO, et al. Relationship between changes in the deep venous system and the development of the postthrombotic syndrome after an acute episode of lower limb deep vein thrombosis: a one- to six-year follow-up. *J Vasc Surg* 1995; 21: 307–312.
11. Friedrich de Wolf MA, Arnoldussen CW, Grommes J, et al. Minimally invasive treatment of chronic iliofemoral venous occlusive disease. *J Vasc Surg* 2013; 1(2): 146–153.
12. Spencer EB, Stratil P and Mizones H. Novel treatment techniques for recanalization of femoral-popliteal deep venous occlusion from chronic thrombosis. *Tech Vasc Interv Radiol* 2014; 17(2): 114–120.
13. Wahlgren CM, Whalberg E and Olofsson P. Endovascular treatment in postthrombotic syndrome. *Vasc Endovascular Surg* 2010; 44(5): 356–360.
14. De Wolf MAF, de Graaf R, Kurstjens RLM, et al. Short-term clinical experience with a dedicated venous nitinol stent: initial results with the sinus-venous stent. *Eur J Vasc Endovasc Surg* 2015; 50(4): 518–526.