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Case PRESENTATION

SCLEROTHERAPY OF INCOMPETENT DEEP VEINS

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ABSTRACT

A 43-year-old female presented with chronic venous ulceration of the left lower leg. She was symptomatic, complaining of pain, pruritus and swelling. She had a history of four previous deep vein thromboses and carried a heterozygous Factor V Leiden mutation. She was on indefinite warfarin. Prior to treatment, the ulcer measured 22.3 cm^2. A duplex study demonstrated incompetence of the left great saphenous vein, incompetent posterior tibial veins (PTVs) and an old organised thrombus of the femoral vein. One session of endovenous laser ablation and two sessions of ultrasound guided sclerotherapy (UGS) were performed for superficial venous incompetence which reduced the ulcer size to 2.5 cm^2. Seven months later, UGS was performed to occlude the incompetent PTVs using 1ml of Sodium Tetradecyl Suphate (STS) 3% foam. Follow-up scans revealed sclerosis of both PTVs and patency of the other deep veins. Four layer compression therapy and wound care was applied and the ulcer eventually healed. The closure of the incompetent deep veins shrunk the ulcer to half its size but the ulcer remained open for another 14 months. To our knowledge this is the first report of intentional sclerotherapy of incompetent deep veins.

Introduction

The conservative approach to the management of venous ulcers has included the use of compression bandaging, leg elevation, wound care, and patient education.[1-3] Treatment aimed at correcting the underlying venous insufficiency has been reported to be essential in preventing recurrence.[4] Here we present the case history of a 43-year-old female patient with post-thrombotic syndrome who underwent ultrasound guided sclerotherapy (UGS) of incompetent posterior tibial veins (PTVs) underlying a chronic recalcitrant venous ulcer.

Case History

A 43-year-old female was referred for management of a chronic left lower leg venous ulcer. The ulcer was present for around 2 years and had not responded to conservative treatments which included a multitude of dressings and topical preparations. Subsequently, she developed a number of allergies to a wide range of dressings as well as topical preparations such as chloromycetin ointment. She was reported to be intolerant of compression therapy.

Her presenting symptoms included pain, cramps and pruritus affecting the left lower limb. Her past venous history included four episodes of deep vein thrombosis (DVT). The first two occurred in the late 1970s involving both legs, a third DVT occurred in the right leg in 1981 after the birth of her first child and a right arm DVT was diagnosed in 1995. She was on indefinite warfarin. Genetic analysis had revealed a Factor V Leiden (FVL) mutation. She was gravida 5, para 2, having had 2 miscarriages, 2 healthy children and one child who did not survive. There was also a history of an uneventful varicose vein operation in 1986. She had a strong family history of DVT and thrombophilia. Her father carried a heterozygous FVL mutation and her sister had a history of DVT. She was single and worked as a hospital assistant which required numerous hours of standing on her feet. She lived more than 2 hours away from the location of our clinic. She was on warfarin and the referring specialist had commenced her on oral antibiotics.

On examination, she had bilateral lipodermatosclerosis but worse on the left side. There was a 16.5 cm^2 ulcer located 5 cm above the left medial malleolus (Fig.1a and 1b) and a smaller ulcer present posteriorly. Venous eczema affected the surrounding skin. The base of the ulcer was partially granulating and there was no florid infection.

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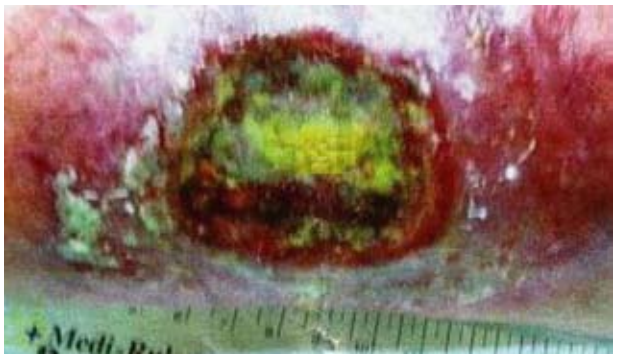

Figure 1a: *Scanned photograph showing the left medial ankle ulcer before treatment. Note the surrounding erythema.*

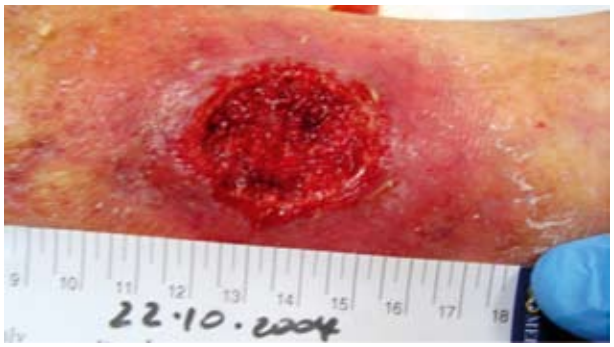

Figure 1b: *Left medial ankle ulcer after some compression therapy. (Courtesy of Dr Duncan Stanford, FACD)*

Given the history of multiple miscarriages, repeat thrombophilia screening was conducted which demonstrated a very low positive IgG anti-cardiolipin antibody, however the IgM was negative. Repeat genetic analysis confirmed a heterozygous FVL mutation.

A lower limb venous duplex incompetence study of the left leg demonstrated an incompetent perforator deep to the ulcer just above the medial malleolus and also incompetence of the distal great saphenous vein (GSV) underlying the ulcer. The proximal GSV was competent but the anterior accessory great saphenous vein was incompetent. There were two incompetent posterior tibial (Cockett) perforators situated above the ulcer and an incompetent tributary with an associated incompetent posterior tibial perforator running directly underneath the ulcer. There was an incompetent distal intersaphenous vein extending below the ulcer. The study also showed the PTVs to be incompetent (Fig. 2a). The Femoral veins (FV) were paired, one of which seemed to have a non-occlusive thrombus.

On a subsequent visit, the ulcer was noted to have enlarged and was now measuring 22.3 cm^2. The ulcer appeared infected and the swabs found a heavy growth of *Staphylococcus aureus*. The patient was commenced on oral cephalexin, venous eczema was treated with topical steroids and 4-layer compression therapy was commenced.

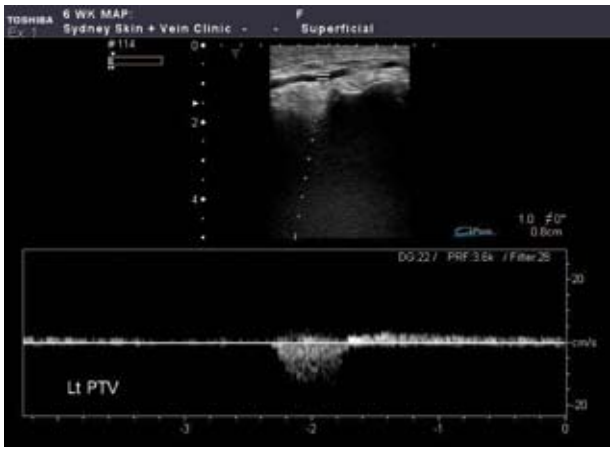

Figure 2a: *Ultrasound image and waveform of the incompetent left posterior tibial vein.*

One session of endovenous laser ablation (EVLA) and two sessions of ultrasound guided sclerotherapy (UGS) were performed to occlude the incompetent superficial pathway. Special attention was given to the incompetent perforators underlying the ulcer which were identified and sclerosed carefully. There was a dramatic improvement after these treatments and the ulcer shrunk to approximately 2.5 cm^2 in size. 4-layer compression therapy (PROFORE) was recommenced however a plateau was reached spanning 2 months where the ulcer remained at the same size. There were compliance issues with the compression and another 5 months of 4-layer compression therapy did not improve the ulcer and its size fluctuated to 4.5 cm^2.

A decision was made to sclerose the patient's incompetent left PTVs. Having obtained informed consent, the procedure was performed. Sodium Tetradecyl Sulphate (STS) 3% was obtained as foam using the Tessari technique[5]. One PTV was injected taking care not to inject the posterior tibial artery. Foam entered the other PTV within seconds through the communications between the pair. The injections were limited to the distal PTV and care was taken for foam not to enter the popliteal vein. A total of 1 ml of foam was used. The patient was given a stat 40 mg subcutaneous injection of enoxaparin to complement her oral warfarin. Compression was applied and the patient was ambulated immediately.

Routine venous thrombosis ultrasound screening at 1 week following the procedure revealed the PTVs to be sclerosed along their entire lengths. The FV displayed an old organised occlusive thrombus of the mid segment but otherwise patent. The popliteal vein was patent. On six week follow-up venous incompetence scanning, the PTVs were found to remain sclerosed (Fig. 2b). All other deep veins were patent.

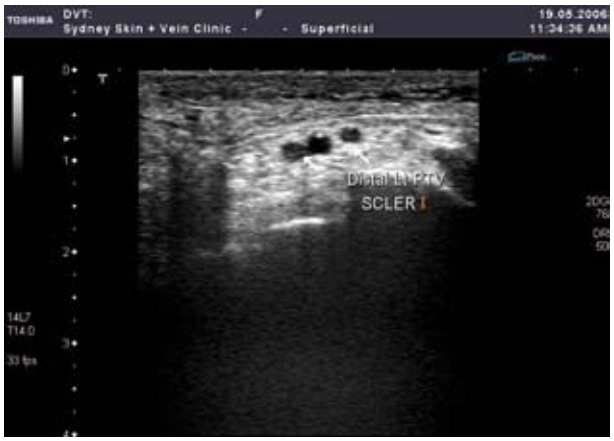

Figure 2b: *Ultrasound image of the left posterior tibial veins post-sclerotherapy.*

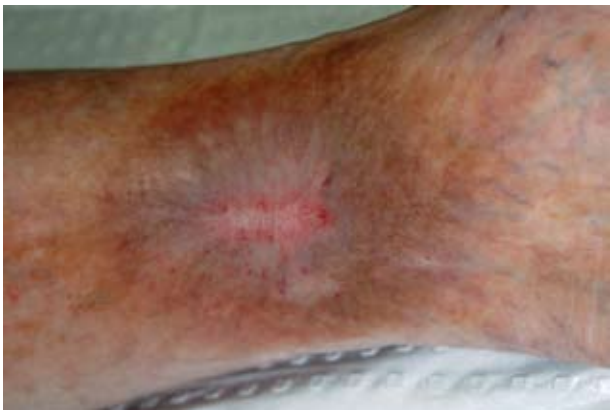

Figure 1c: *Healed left medial ankle ulcer after endovenous laser ablation and ultrasound guided sclerotherapy. Note the atrophie blanche type of scarring.*

4-layer compression therapy was continually maintained and the patient was seen twice a week. Following UGS of the PTVs, the ulcer shrunk further to approximately 1.5 cm² in size. Compliance issues with the compression therapy were noted again and the patient was instructed to strictly adhere to the compression regime. Further scanning revealed PTVs to remain sclerosed but the anterior tibial veins and one peroneal vein were found to be incompetent. The ulcer finally completely healed 14 months after UGS of the PTVs (Fig. 1c). Long term follow-up venous incompetence study performed 18 months after the treatment showed the PTVs to be atrophic with a small incompetent lumen just below the ulcer. The proximal PTVs were patent and incompetent.

Discussion

This paper presents the first report of UGS being performed on incompetent deep veins. To our knowledge no previous publications have described this procedure to be used for this purpose. Deep vein incompetence is a difficult management problem and treatment options are limited. Apart from compression therapy and other conservative measures, the other treatment options are surgical, invasive and limited in their application. The surgical options are usually used in treatment of deep venous obstruction or axial reflux. These can be classified as those involving valve repair and those involving segment substitution. Valvuloplasty techniques include internal (inside the vein) or external (outside the vein) methods, while substitution techniques include transplantation, transposition, or valve substitution.[6-7-8-9] These methods have been elegantly reviewed by Eklof et al.[9] One technique requiring a special mention is external stenting pioneered by the Australian Phlebologist Rod Lane, used to repair incompetent saphenofemoral junctions as well as incompetent deep veins.[6] This procedure involves external placement of a small cuff to bring the valve cusps together to restore normal function. It requires the presence of normal valves and hence has limited application in post-thrombotic veins where the valves are damaged.

The surgical management of deep venous reflux is usually reserved for symptomatic patients with an axial deep vein obstruction and/or reflux.[9] This usually limits the pathology to iliac, femoral or popliteal veins.[7-8-9] These procedures are not widely available and are technically difficult. Most surgeons would advocate treatment of superficial venous incompetence first, before deep veins are listed for surgery.[9] Our patient presented with post-thrombotic incompetence of small calf veins and hence none of these surgical approaches were indicated or feasible.

We treated the superficial venous incompetence successfully with a combination of EVLA and UGS which significantly reduced the size of the ulcer from 22.3 cm² to 2.5 cm². The ulcer however remained active for another seven months. A decision was made to sclerose the incompetent left PTVs. It was reasoned that these deep veins were contributing to local venous hypertension underlying the ulcer. There was precedence for closing incompetent deep veins and ligation of incompetent deep veins was reported as far back as 1965 by Dodd and later on by others.[10, 11] The closure of the incompetent PTVs reduced the ulcer to half its size but it took another 14 months of 4-layer compression therapy for the ulcer to completely heal. Therefore, it cannot be concluded that this procedure had a dramatic effect on the healing of the ulcer. Others have reported using EVLA to occlude incompetent deep veins with good results (personal communication with Dr John R. Kingsley, USA).

Concern might be raised regarding the possibility of venous thromboembolism (VTE) following UGS of deep veins.

Our patient had a history of four previous DVTs, and was heterozygous for FVL mutation. The heterozygous mutation carries a 5-10 fold increased risk of VTE.[12] Given this, the patient was maintained on warfarin and was given a prophylactic dose of enoxaparin during each treatment to prevent thromboembolic complications. Follow-up scans did not detect DVT post-sclerotherapy. It must be noted however, that this procedure was carefully planned and thoroughly discussed with the patient and it was not undertaken lightly. The author's in vitro research has previously reported the detergent sclerosants and in particular STS to possess anticoagulant properties at high concentrations.[13] Special care was taken to introduce the sclerosant into the target vessel at 1 cm intervals hence avoiding a drop in the effective concentration and its associated pro-coagulant properties.

Apart from VTE, the other potential major complication of this procedure would have been inadvertent intra-arterial injection. B-mode ultrasound was used in conjunction with the colour and spectral Doppler modes to visualize the accompanying artery. Intra-arterial injection of posterior tibial arteries could have lead to a disastrous clinical outcome. Inadvertent injection of the accompanying posterior tibial nerve would have caused severe pain and possibly permanent nerve injury.

The long term follow-up ultrasound scans showed incompetence of both anterior tibial veins and a single peroneal vein. This was not detected in the initial scans and might have contributed to the prolongation of the healing time. These scans also demonstrated recanalisation of the proximal PTVs and atrophy of the distal segment. Deep veins are no different to superficial veins and a single and careful session of foam UGS was not adequate to fully sclerose these veins. Maintaining the sclerosant in set anatomical segments is always a challenge. We know from treating superficial veins that treating a single tributary in isolation from other veins is difficult and the agent may enter the truncal veins inadvertently. The same principle applies to the deep veins as it is difficult to ~~close deep~~ tributary such as PTV without foam entering the popliteal vein. This is why the treatment was done cautiously which might have contributed to the proximal recanalisation.

Treatment of the superficial venous incompetence including the incompetent underlying perforators dramatically reduced the ulcer size from 22.3 cm^2 to 2.5 cm^2. This case further highlights the importance of treating the underlying venous incompetence as an adjunct to compression therapy.

In summary, we present the first report of ultrasound guided sclerotherapy of incompetent deep veins. This procedure was performed to control the venous hypertension underlying a chronic recalcitrant ulcer. The safety and effectiveness of this treatment approach requires further evaluation and more experience is needed for this technique to be used more effectively.

Acknowledgements

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