Endovenous ablation of incompetent perforating veins is effective treatment for recalcitrant venous ulcers

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Objectives: **Endovenous closure of incompetent saphenous veins has been reported to facilitate venous ulcer healing; however, there is little information about the effectiveness of perforator ablation (PA) in healing recalcitrant venous ulcers. We report our experience with PA with venous ulcers unresponsive to prolonged compression therapy.**

Methods: **Patients with nonhealing venous ulcers of >3 months' duration underwent duplex ultrasound to assess their lower extremity venous system for incompetence of superficial, perforating, and deep veins. Patients who had either no saphenous incompetence or persistent ulcers after saphenous ablation underwent PA of incompetent perforating veins >3 mm that demonstrated reflux; initial treatment was performed on the perforator vein adjacent to the ulcer with additional incompetent veins treated if ulcer healing failed.**

Results: **Seventy-five ulcers with 86 associated incompetent perforating veins were treated with PA in 45 patients with CEAP 6 recalcitrant venous ulcers. Treated incompetent perforator veins were located in the medial ankle (61%), calf (37%), and lateral ankle (2%). Initial success of PA, assessed by postprocedure duplex ultrasound, was 58%; repeat ablation was 90% successful and 71% had eventual successful perforator closure. No complications (skin necrosis, infection, or nerve injury) occurred. Failure of ulcer healing with successful perforator closure occurred in 10% and was due to intercurrent illness, patient noncompliance, and patient death due to unrelated causes. Of patients who healed their ulcers, the healing occurred at a mean of 138 days; an average PA of 1.5 incompetent veins per ulcer was required for healing. Ninety percent of ulcers healed when at least one perforator was closed; no ulcer healed without at least one perforator being closed.**

Conclusions: **This experience demonstrates both the feasibility and effectiveness of PA for a selected group of patients with venous ulcers who fail conventional therapy with compression. (J Vasc Surg 2011;54:737-42.)**

Incompetent perforating veins have been implicated in the development of venous ulcers since the relationship was first proposed by Homans in 1916.[1] Procedures to eliminate incompetence and reflux in the perforating veins have been advocated using surgical ligation,[2] subfascial surgical approaches,[3] and sclerotherapy.[4] The approach has shifted toward minimally invasive techniques, due to the fragility of the skin around the ulcer and the risk of creating new nonhealing wounds with surgical procedures.

Recently, endovascular ablation has been advocated to close refluxing perforating veins.[5] Endovenous ablation reports have demonstrated good technical success and low complication rates,[6] although most reports have focused primarily on successful ablation of the vein, rather than venous ulcer healing.

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We conducted this study to evaluate patients who had chronic venous ulcers that had received optimal therapy with compression and wound care for at least 3 months and therefore were considered to be refractory to conventional treatment. These patients were optimal candidates for our study to determine the impact of perforator ablation on the healing of recalcitrant venous ulcers.

METHODS

Patients with venous ulcers were evaluated for treatment in the Gonda Wound Care Center at UCLA. Some patients had undergone procedures at outside facilities but none had undergone endovenous ablation of perforating veins before evaluation and treatment in our program.

As part of the initial evaluation at the wound care center, all patients underwent a medical history and physical examination, ankle-brachial index (ABI) when indicated, photography of the ulcer with measurements of area recorded using a dedicated wound software system (Wound Expert), and duplex ultrasound of the superficial, deep, and perforating veins. Arterial disease was corrected by endovascular or bypass procedures. All superficial veins, including saphenous veins and tributaries, that were dilated to >3 mm and showed reflux, were treated with either endovenous ablation or microphlebectomy. Patients had endovenous ablations and procedures on the tributary veins as well as skin grafts. When these procedures failed to heal or improve the venous ulcer over 3 months of closely

observed wound care, the patient was evaluated for perforator incompetence.

Ulcer management prior to endovenous ablation. Patients underwent treatment in our dedicated wound care center for a minimum of 3 months and a mean of 34 months prior to endovenous ablation of incompetent perforator veins. In addition to twice weekly three and four layer compression therapy, patients had debridement, topical and systemic antibiotic treatment, topical growth factors, and skin substitutes. When patients completed 3 months of compression therapy, those with persistent ulcers that did not show evidence of healing by measurement using the software system underwent repeat duplex ultrasound to re-evaluate for any remaining reflux in the superficial system, and if none were found, to specifically evaluate the limb for incompetent perforating veins. Incompetent perforators in the medial ankle and calf, high calf, paratibial region, thigh, and lateral leg were investigated in a sitting position, using a reflux time of >1 second and a diameter of >3 mm as criteria for perforator incompetence. Patients with incompetent perforating veins were offered endovenous ablation to treat their perforator reflux and potentially heal their venous ulcer.

Endovenous ablation technique. The technique for ablation was based on publications describing the endovenous radio-frequency perforator ablation technique[7] and the IFU for the VNUS Closure RFS device (VNUS Medical Technologies, San Jose, Calif), specifically designed for ablation of perforating veins. The procedures were performed by four vascular surgeons, although one performed >90% of the procedures. The patient was placed in reversed Trendelenburg position on an electronic tilt table and a GE Logiq e portable duplex scanner (GE Healthcare Technologies, Waukesha, Wisc) was used for duplex imaging by the operating surgeon. The incompetent veins had been previously identified in a vascular laboratory and were confirmed by the surgeon prior to the procedure. Once the incompetent veins were marked, the leg was then prepped sterilely for the procedure. Using an ultrasound transducer of 12 MHz, covered with a sterile sheath, the incompetent perforating veins immediately above the ulcer were selected for initial treatment.

We used the stylet from the RFS catheter to puncture the skin, if it was malleable. When it was hard and/or calcified, we used a no. 11 blade to puncture the skin, followed by the RFS catheter. The stylet was advanced at a 45 degree angle, and the transducer was rotated 90 degrees to confirm position in each plane as the stylet was advanced to the junction of the perforator vein and the fascia. An attempt was made to puncture the wall of the vein at the level of or immediately below the fascia. Although aspiration of blood was the goal, it was not always achieved. Once the location of the catheter was confirmed, the stylet was removed, the catheter position again confirmed, and the catheter was then surrounded with lidocaine, which was injected along the catheter with a 25 g needle to the level of the fascia. The patient was then placed in Trendelenburg position, the position of the catheter was again confirmed, and the vein was then treated with radio-frequency energy, using 1 minute of treatment in each quadrant, while impedance and temperature were kept within the therapeutic range (<400 Ω and at 85°C, respectively). A second level of ablation, using a similar technique, was often performed in the vein above the fascia. Although an attempt was made to confirm successful ablation postprocedure, the lidocaine in the region of the perforator frequently caused an appearance of occlusion, even when the compression was from fluid extrinsic to the vein. After removing the catheter, the leg was dressed with a three or four layer compression dressing.

Confirmation of closure of the perforating vein was obtained at the next wound care visit by a vascular laboratory technician who was not involved in the procedure and provided an independent assessment of successful ablation. Perforator closure was defined as no blood flow in a previously incompetent perforator vein, demonstrated in a sitting or standing position, by duplex ultrasound.

Table I. Demographic variables in patients undergoing endovenous perforator ablation

Variable	
Mean age (years)	74
Mean BMI (kg/m^2)	31
Sex (male: female)	26:19
ABI (<0.9)	29%
Anticoagulation therapy	33%
Previous DVT	31%
Deep venous reflux	46%
Diabetes mellitus	18%

ABI, Ankle-brachial index; *BMI,* body mass index; *DVT,* deep vein thrombosis.

RESULTS

Between April 4, 2007 and September 9, 2010, 208 patients had venous ulcers treated in our wound care center; 86 were managed with compression therapy, 77 patients were treated with a superficial procedure alone, and 45 had perforator ablation. The 45 patients had 75 nonhealing venous ulcers for >3 months in 51 limbs; all patients had CEAP 6 clinical classification on entry into the study. Patients ranged in age from 35 to 93, with a mean age of 74 (SEM ± 1.85) and had a mean body mass index (BMI) of 31 (SEM ± 1.21 range 19-45) (Table I). The location of the ulcers was medial ankle in 61%, calf in 37%, and lateral ankle in 2%. The etiology of the venous ulcer was postphlebitic in 31% and of unknown etiology in 69%; one patient had venous obstruction as well as reflux. The number of incompetent perforating veins ranged from one to five in each leg with a venous ulcer. Patients had their ulcer for a mean of 93 months (range 1-300 months). The average diameter of incompetent perforator was 4.3 mm. Twenty-one patients had deep venous reflux; 10 were post-thrombotic; 12 patients with 13 lower limb ulcers had peripheral arterial disease, and eight limbs underwent arterial revascularization. Patients had undergone procedures

Table II. Procedures performed before referral to UCLA

Procedure	*Number*
Vein stripping (4 GSV & 2 SSV)	6
Perforator procedure	1
Vein ligation/phlebectomy	0

GSV, Great saphenous vein; *SSV,* small saphenous vein.

Table III. Procedures performed at UCLA prior to perforator ablation

Procedure	*Number*
Tributary vein ligation	13
Vein stripping (6 GSV & 5 SSV)	11
SEPS	7
Great saphenous vein ablation	16
Small saphenous vein ablation	5
Skin graft	14

GSV, Great saphenous vein; *SEPS,* subfascial endoscopic perforator surgery; *SSV,* small saphenous vein.

at other institutions to treat the venous ulcers, including four great saphenous vein strippings, two small saphenous vein strippings, and one surgical procedure on a perforating vein (Table II). Patients had concomitant diseases that might interfere with wound healing, including three patients with chronic liver disease, two undergoing chemotherapy for a neoplasm, one immunosuppressed for systemic lupus erythematosus, one with leg edema due to congestive heart failure, one with renal insufficiency that was dialysis dependent, and one with sickle cell disease. Prior to consideration of endovenous perforator ablation, patients underwent multiple procedures at UCLA to heal their wounds, and many had been performed years prior to the procedure (Table III). Patients did not undergo an endovenous perforator ablation unless they had at least a 3-month period of intensive wound care without ulcer healing after one of these procedures and had eliminated all sources of superficial veins reflux, which was confirmed by duplex ultrasound. Ten patients had perforator reflux documented, yet did not undergo a procedure due to insurance company denial.

Endovenous ablation results. Ablation was initially successful in 58% of incompetent perforator veins and 12% additional ablation procedures were performed at a mean of 193 days (range 27-746 days) (Fig 1); overall, the final closure rate was 71%. There was a learning curve with the procedure, and the success improved over time from 56% to 79% (Fig 2). The closure success was independent of the site of the perforator, BMI of the patient, or age of the ulcer. Average of ulcer size before the first successful radiofrequency ablation was 12 cm with SEM ± 2.53 cm. Ulcers healed in patients with successful ablation of at least one perforator in 90%, at a mean of 138 days (range 60-365); six ulcers never healed. The nonhealing ulcers had a mean diameter of 6.5 cm and a mean age of 127 months.

All patients who healed ulcers had at least one incompetent perforator closed by ablation; no patient had wound healing without at least one successful perforator ablation. The average follow-up was 12.85 months with (SEM ± 1.48 months) 4% of healed ulcers recurred (two ulcers in one patient) probably because of common iliac vein obstruction. There were no complications associated with the perforator ablation, such as wound infection, skin necrosis, or nerve injury.

DISCUSSION

Ambulatory superficial venous hypertension secondary to reflux in the deep, superficial, and perforating veins of the leg is the accepted pathogenesis of most venous ulcers,[1,8,9] although venous hypertension may also occur due to venous obstruction.[10] Local cellular changes in the tissues occur secondary to the venous hypertension, and include white cell mediated inflammation and fibrin cuffs.[10] The most common identifying etiology of venous incompetence results from changes in the vein valves and walls following deep vein thrombosis (DVT) that renders the valves incompetent. Once ambulatory venous hypertension occurs, lipodermatosclerosis may develop and eventually venous ulceration occurs, although this process can be delayed or prevented by leg compression. Once venous ulceration occurs, healing of the tissue can best be accomplished by reducing leg edema and superficial venous hypertension. The initial approach is typically compression of the skin and subcutaneous tissue with multilayered dressings, which is effective for most patients. However, some continue to have progressive lipodermatosclerosis and are unable to heal venous ulcers when they occur. If refractory ulceration occurs, imaging with duplex ultrasound can identify the location of refluxing or obstructed veins, and physiologic studies can quantify the degree of reflux. When compression fails to heal venous ulcers, therapeutic options focus on preventing superficial vein and perforator reflux and occasionally correcting deep valvular reflux. Most investigators have favored correction of the superficial reflux initially by removing or ablating the refluxing saphenous veins and tributaries, since these procedures are relatively simple and effective and will heal many venous ulcers.[2,11,12] When superficial ablation does not result in venous ulcer healing, perforator interruption is usually the next step. Whether it is done with direct surgery, sclerotherapy, subfascial endoscopic perforator surgery (SEPS), or endovenous ablation, the elimination of perforator reflux reduces ambulatory venous hypertension. Minimally invasive techniques are being used more frequently, since they are associated with fewer wound complications from the procedure. There are few papers that address success rates for ablation; we had a lower success rate, but also a much lower complication rate.[3,13]

In our study, patients underwent not only optimal wound care and compression for their ulcers, but also had refluxing superficial veins treated with ablation and ligation, and yet they were unable to heal their ulcers. The average period of wound care was ~3 years in our center,

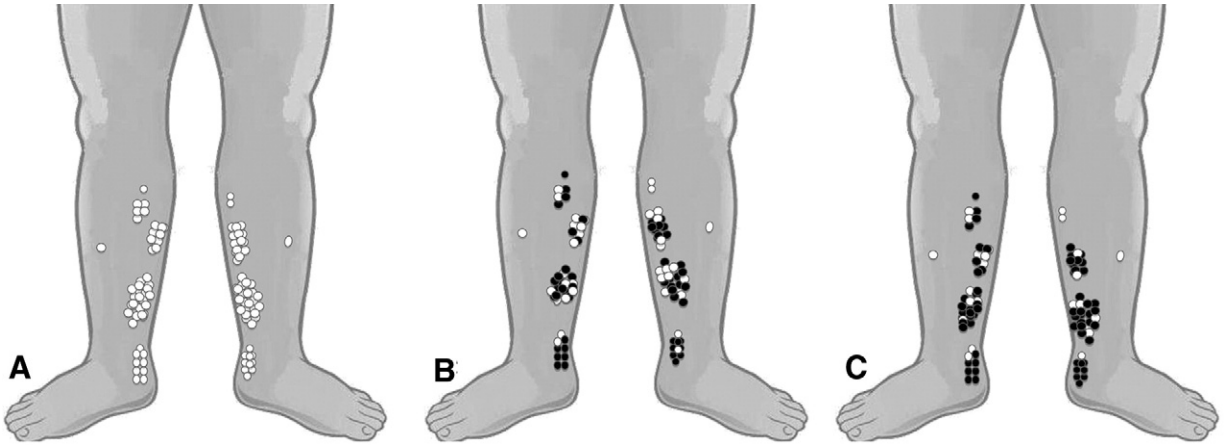

Fig 1. A, The location of incompetent perforating veins in patients with recalcitrant venous ulcers. Each *open circle* represents a refluxing perforating vein. **B,** After the initial ablation procedure, the *black circles* represent ablated veins. **C,** After the second ablation procedure, additional veins were closed, as noted in this diagram.

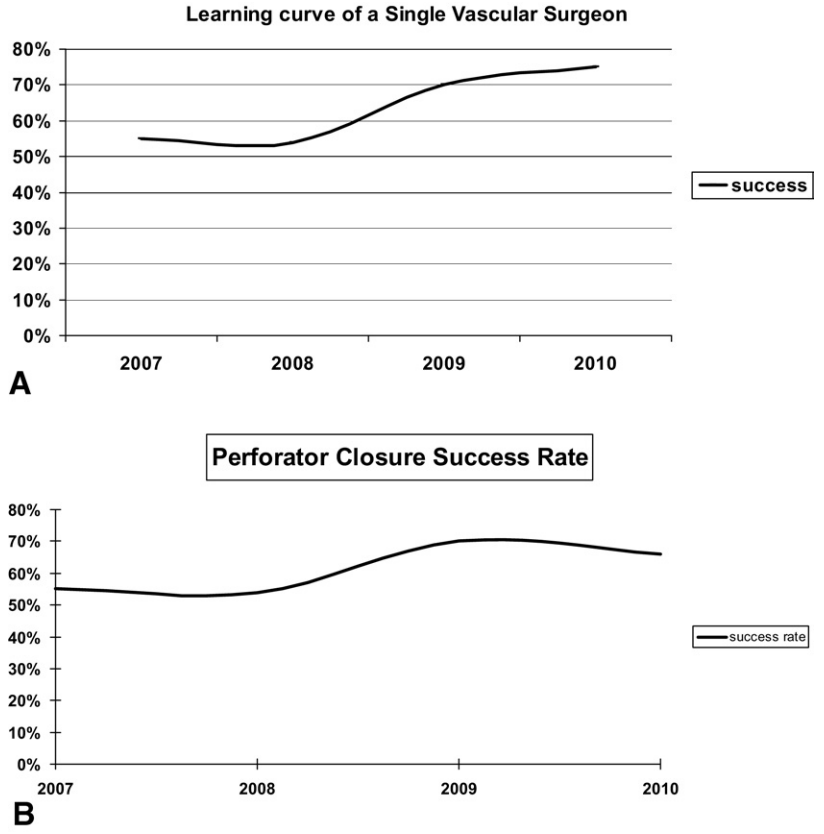

Fig 2. A, Learning curve of a single vascular surgeon. **B,** Perforator closure success rate. The successful ablation rate for our center is shown here over a 4-year period.

indicating that aggressive attempts at wound healing were made before perforator ablation was offered to the patients. Consequently, the only remaining option is perforator ablation; there are two minimally invasive approaches to perforator ablation, sclerotherapy, and endovenous thermal ablation. We have had little experience with perforator sclerotherapy, but are concerned about the risk of reflux of the solution into the deep system and development of

DVT.[14] The endovenous ablation of perforators seems to have a more predictable outcome with a lower risk.

We learned several important principles from this study. First, the selection of appropriate patients is critical to maximize the impact of perforator ablation. There are patients who are unable to heal their venous ulcers after many years of compression, in spite of excellence compliance with recommended therapy. It is this group of patients who are most likely to benefit from ablation of incompetent perforator veins. When we encounter a patient with venous ulcers who demonstrates compliance with recommended compression therapy over a period of more than 3 months under direct supervision, we have found that the likelihood of later healing is low. Consequently, we try to identify all incompetent superficial and perforating veins that might be contributing to ambulatory venous hypertension and attempt to correct the incompetence with ablation of refluxing veins. Due to the high success of ablation of saphenous veins and other axial veins, which in our experience is >99%,[15] we correct them first. We also ligate or perform microphlebectomy on incompetent tributary veins that reflux into the area of the ulcer, although we are reluctant to make incisions into skin with lipodermatosclerosis, since wound healing can be a problem. Once all other causes of venous reflux are dealt with, if the ulcer remains nonhealing, we investigate for incompetent perforating veins by repeating the duplex ultrasound. For medial ankle venous ulcers, we focus on the region of the posterior tibial upper, mid, and lower groups. For lateral ankle ulcers, we focus on the lateral calf and ankle perforators. If no incompetent perforating veins are found in these regions, we then look for paratibial veins, the 24 cm perforating vein, and even thigh perforating veins that are grossly dilated, incompetent, and reflux through tributary veins into the area of the venous ulcer.

Several recent studies have emphasized the role of venous obstruction in the development of venous ulcers.[16] Currently, we limit our evaluation of patients with nonhealing venous ulcers to duplex ultrasound of the leg and thigh, up to the common femoral vein and distal external iliac vein, and do not extend the evaluation to the entire iliac system, using either contrast venography, computed tomography (CT), or magnetic resonance (MR) venography, although we do look for obstruction of the deep venous system in the femoral vein and the popliteal vein, using duplex ultrasound. In this series of venous ulcers, one patient had obstruction of the proximal deep venous system, and he failed to heal his ulcer, even after successful perforator ablation. We had decided to investigate for proximal obstruction, as recommended by Raju and Hingorani, since their reports indicate that correction of proximal obstruction can heal venous ulcers, even when they have reflux.[13,16]

Patients who have either healed ulcers (CEAP 5) or lipodermatosclerosis (CEAP 4) were not included in this study, although we have encountered many CEAP 4/5 patients who have reflux in the perforating veins. We currently are looking at outcomes in these patients to determine if ablation of incompetent perforating veins will slow the progression of lipodermatosclerosis or prevent recurrent ulceration. Until this issue is resolved, we do not recommend ablation of perforating veins for CEAP 4 or 5 disease but do recommend lifetime compression with quality support hose.

Second, we learned that this is a technically difficult procedure with the current ablation devices and has a significant learning curve. In our study, the learning went on over 4 years and benefited from the relatively large number of patients with venous ulcers. For an individual surgeon who does not have a large venous practice, the learning curve may be longer than ours. It takes patience and a significant number of procedures to be sure that the stylet and catheter are in the perforating vein at the level of the fascia. There is a very short-distance between the skin and the tibial nerves and arteries, so placement must be more precise than with ablation of the saphenous system. We believe that a goal of 80% procedural success is achievable for most surgeons, although many will start out at 50% to 60%. However, even when ablation fails to close the perforator vein, the procedure can be repeated with good success, and the complications of failure are rare. We believe that a second and even third procedure is justified in these patients.

We have debated how many incompetent perforators should be closed at a single sitting and currently believe that the best approach is to pick the incompetent perforator veins immediately adjacent to and above the ulcer—ones that are likely to reflux into the ulcer when the patient is sitting or standing as the initial veins for closure. Once these veins are closed, then a search for other large incompetent perforating veins in the region should be made. Frequently, other nearby perforators will be difficult to visualize once the first perforator has been ablated due to the infiltration of lidocaine throughout the adjacent tissue. If refluxing and close to the ulcer, a second incompetent vein should also be considered for ablation.

Since this was not a prospective, randomized study, it does not provide unequivocal evidence of the benefit of perforator ablation, although it is highly suggestive. Using patients as their own controls and subjecting them to a lead-in phase with a mean time of 3 years, where we attempted to heal their ulcers under direct observation in a very experienced wound care setting, indicates that these ulcers were very unlikely to heal without some other modification, such as weight loss, better nutrition, or elimination of the dependent position by strict bed rest. For most patients, these options are not achievable, so the patients we treated were presented with either prolonged wound care or procedures to ablate refluxing veins. The only other option of excision of the ulcer with ligation of the refluxing veins in the ulcer bed, followed by a skin graft, has been reported by other authors[17] but this option was not offered to our patients.

The most compelling argument that perforator ablation facilitates ulcer healing is that we had no patient in our series who healed an ulcer following failed ablation, so

simply improving wound care intensity and patient compliance was only successful when at least one perforator was closed. In addition, when at least one perforator was closed, a very high percentage of patients (>90%) had complete healing of their ulcer.

CONCLUSIONS

This experience with ablation of CEAP 6 nonhealing venous ulcers demonstrates both the feasibility and effectiveness of perforator ablation in patients who fail a 3-month trial of compression therapy. After superficial refluxing saphenous and tributary veins have been treated, those patients who are still unable to heal their venous ulcers with compression and local wound care benefit from perforator ablation. The technique of ablation has a significant learning curve, but complications are uncommon and repeated ablation until at least one incompetent perforator has been closed results in a healing rate of >90% and no recurrent ulcers over the short-term in patients who are compliant with compression therapy. We recommend perforator ablation after all other forms of therapy have failed for patients with recalcitrant venous ulcers.

AUTHOR CONTRIBUTIONS

Conception and design: PL
Analysis and interpretation: PL
Data collection: AA
Writing the article: PL, AA
Critical revision of the article: PL, AA, DR, BD, HG, JJ
Final approval of the article: PL
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Overall responsibility: PL

REFERENCES

1. Homans J. The operative treatment of varicose veins and ulcers, based upon a classification of these lesions. Surg Gynecol Obstet 1916;22: 143-59.
2. Linton RR. The communicating veins of the lower leg and the operative technic for their ligation. Ann Surg 1938;107:582-93.
3. Gloviczki P, Cambria RA, Rhee RY, Canton LG, McKusick MA. Surgical technique and preliminary results of endoscopic subfascial division of perforating veins. J Vasc Surg 1996;23:517-23.
4. Masuda EM, Kessler DM, Lurie F, Puggioni A, Kistner RL, Eklof B, et al. The effect of ultrasound-guided sclerotherapy of incompetent perforator veins on venous clinical severity and disability scores. J Vasc Surg 2006;43:551-7.
5. Peden E, Lumsden A. Radiofrequency ablation of incompetent perforator veins. Persp Vasc Surg Endovasc Ther 2007;19:73-7.
6. Proebstle TM, Herdemann S. Early results and feasibility of incompetent perforator vein ablation by endovenous laser treatment. Dermatol Surg 2007;33:162-8.
7. Elias S, Peden E. Ultrasound-guided percutaneous ablation for the treatment of perforating vein incompetence. Vascular 2007;15:281-9.
8. Cockett FB. The pathology and treatment of venous ulcers of the leg. Br J Surg 1953;43:260-78.
9. O'Donnell TF Jr., The present status of surgery of the superficial venous system in the management of venous ulcer and the evidence for the role of perforator interruption. J Vasc Surg 2008;48:1044-52.
10. ′Neglén P, Thrasher TL, Raju S. Venous outflow obstruction: an underestimated contributor to chronic venous disease. J Vasc Surg 2003;38:879-85.
11. Bello M, Scriven M, Hartshorne T, Bell PR, Naylor AR, London NJ. Role of superficial venous surgery in the treatment of venous ulceration. Br J Surg 1999;86:1475-6.
12. Marrocco CJ, Atkins MD, Bohannon WT, Warren TR, Buckley CJ, Bush RL, et al. Endovenous ablation for the treatment of chronic venous insufficiency and venous ulcerations. World J Surg 2010;34: 2299-304.
13. Hingorani AP, Ascher E, Marks N, Shiferson A, Patel N, Gopal K, et al. Predictive factors of success following radio-frequency stylet (RFS) ablation of the incompetent perforating veins (IPV). J Vasc Surg 2009;50:844-8. Epub;2009, July 3.
14. Guex JJ, Allaert FA, Gillet JL, Chleir F. Immediate and midterm complications of sclerotherapy: report of a prospective multicenter registry of 12,173 sclerotherapy sessions. Dermatol Surg 2005;31: 123-8; discussion:128.
15. Lawrence PF, Chandra A, Wu M, Rigberg D, DeRubertis B, Gelabert H, et al. Classification of proximal endovenous closure levels and treatment algorithm. J Vasc Surg 2010;52:388-93.
16. Raju S, Darcey R, Neglén P. Unexpected major role for venous stenting in deep reflux disease. J Vasc Surg 2010;51:401-8.
17. Jones JE, Nelson EA. Skin grafting for venous leg ulcers. Cochrane Database Syst Rev 2007;2:CD001737.

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