DOI: 10.14260/jemds/2015/1542

ORIGINAL ARTICLE

TO STUDY THE ROLE OF COMPRESSIVE THERAPY AND SURGICAL INTERVENTION IN TREATMENT OF VENOUS ULCERS

Sashi Walling T[1], K. Vinod[2]

HOW TO CITE THIS ARTICLE:
Sashi Walling T, K. Vinod. "To Study the Role of Compressive Therapy and Surgical Intervention in Treatment of Venous Ulcers". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 61, July 30; Page: 10697-10703, DOI:10.14260/jemds/2015/1542

ABSTRACT: OBJECTIVE: The aim of this study is to study the response of venous ulcers to compressive therapy plus surgical treatment. **STUDY DESIGN**: Patients presenting to our hospital from the period March' 2014 to December' 2014 with venous ulcers were examined and investigated. Thirty patients who had findings of venous ulcers along with varicose veins were selected for the study. Patients were initially treated with elastic compression bandages to allow ulcer healing followed by surgical intervention of varicose veins and incompetent perforators. The response to treatment was evaluated in terms of symptomatic improvement and ulcer healing. **RESULTS:** Venous ulcers respond well to both conservative treatment and surgical intervention. Meticulous assessment of patients is necessary for successful treatment. In our study period, elastic compression bandages combined with surgical treatment was successful in improving symptoms of venous ulcer as well as ulcer healing in the patients. Patient education regarding compliance to treatment is paramount to ulcer healing and preventing recurrence.

KEYWORDS: Venous ulcer, Varicose veins, Saphenofemoral junction (SFJ), Elastic compression bandage, SFJ ligation.

INTRODUCTION: Varicose veins primarily affect the lower limbs. The prevalence of varicose veins have been estimated to be between 5% and 30% in the adult population.(1) Varicose veins can be classified as primary or secondary. Primary varicose veins result from intrinsic abnormalities of the venous wall, whereas secondary varicose veins are associated with deep and/or superficial venous insufficiency. The findings of varicose veins include dilated and tortuous veins, telangiectasias and fine reticular varicosities. Varicose veins constitute a progressive disease that steadily becomes worse, even often with interventions. The most common complications are superficial thrombophlebitis, acute bleeding from one of the thin-walled varices, eczema, and finally skin ulceration.

Venous ulcers occur either in connection with varicose veins or post deep vein thrombosis in which recanalisation of the deep vein has occurred but the valves is either destroyed or incompetent due to damage. Various theories have been postulated regarding the pathogenesis of venous ulcers. Incompetence of valves in the deep venous system, communicating veins and less frequently in the superficial system produces stasis of blood and rise in the local venous pressure. This in turn produces thickening in the wall of the capillaries and stagnation of blood in the affected area. The transport of oxygen and nutrient substances required for cellular survival is thus affected and results in local cellular necrosis and ulceration.

A study by Lindemayr et al.(2) suggested that local venous hypertension opens up the normally closed arteriovenous shunts and arterial blood reaches the venous channels bypassing the capillary circulation. This shunting of arterial blood produces local tissue anoxia, necrosis and ulceration.

DOI: 10.14260/jemds/2015/1542

Precapillary fibrin has been found in the dermis of almost all patients with the pre-ulcerative condition of lipodermatosclerosis (pigmentation, inflammation and induration of the skin). Browse and Burnand.[3] demonstrated the effect of venous hypertension on the venous microcirculation and observed histologically that in large capillaries, pericapillary fibrin deposition, called the "fibrin cuff," occurred. Peripherally fibrin deposition has been shown to precede the development lipodermatosclerosis. Fibrin cuffing was found around the dermal capillaries with fibrin cuffs lowering the level of transcutaneous oxygen. This implies that as fibrin deposition occurs around capillaries, tissue anoxia occurs.

Thomas et al.[4] showed that significantly more white cells 'disappear' from the venous blood in the depending limbs of patients than from the dependent blood of normal limbs. They suggested that these vanishing leucocytes are being 'trapped' in the capillaries beneath venous ulcers where they cause capillary occlusion and tissue ischemia. Although leukocyte trapping within capillaries is probably not the sole cause, based on later evidence, it is likely that leukocytes become activated, transmigrate into the vein walls, and mediate some of the observed damage. Consistently, punch biopsy of ulcers suggests that leukocytes play a role in the manifestations of chronic venous insufficiency.[5]

Further studies are essential to elicit the mechanisms of ulcer development because a better understanding of causation will almost certainly leads to improved prospects for prevention.

There are various modalities of treatment for venous ulcers. Compressive therapy is the mainstay of medical management. Other conservative measures include pharmacologic treatment, wound and skin care, and structured calf muscle exercise. Interventional management includes venous sclerotherapy either as a primary therapy or in combination with surgical procedure, endovenous radiofrequency and thermal ablation, venous valve reconstruction of the deep veins, open perforator ligation surgery or SEPS (Subfascial Endoscopic Perforator Surgery), saphenofemoral junction (SFJ) ligation and stripping of Great saphenous vein (GSV) for SFJ incompetence. In our study, we observe the response of venous ulcer to elastic compression therapy followed by SFJ ligation and stripping of GSV with subfascial ligation of incompetent perforators.

MATERIALS AND METHODS: During March'2014 to December'2014, patients who presented to the Surgical OPD with venous ulcer in the leg were evaluated clinically and investigated. Thirty patients who had venous ulceration along with varicose veins were selected for the study. A detailed history was taken in regard to symptoms, duration of varicose veins and ulceration, occupation, previous surgical and medical treatment. The ulcer was examined in detail and the extent of superficial varicosities was also noted.

The venous examination was done with the patients in the standing and supine positions. The limbs were inspected for dilated Great saphenous vein and Small saphenous vein. Incompetence of saphenous veins and perforators were determined by Trendelenberg and multiple tourniquet tests.[6] Perforator incompetence was localized clinically (confirmed by Duplex scan) by palpating defects in the deep fascia at relevant anatomical sites. Abdominal and pelvic examinations were done. Cardiovascular system and peripheral pulses were carefully assessed to exclude arterial disease. Routine blood and urine investigations, ECG, Chest X-ray, Echocardiogram in relevant patients, were done. Patients were sent for Duplex scan assessment of the varicose veins and deep veins.

Venous ulcer was treated initially with Bisgaard regimen. This consists of- Massage and elevation of the leg to soften the indurated area around the ulcer; Passive movements to maintain the

DOI: 10.14260/jemds/2015/1542

mobility of the foot and ankle; Active movements to the calf muscles in elevation and in standing (With bandages on); Teaching correct walking, placing heel down first and using the calf muscles to lift the heel of the back foot giving 'spring' to the walk and hence improving the venous pump; A firm elastic compression bandage was applied spirally from the base of the toe to the knee so that movements in walking alternately stretch and relax the bandage and produce an added venous pumping effect; The ulcer was cleaned with hydrogen peroxide initially till the slough separated and then EUSOL (Edinburgh University Solution of Lime) and saline dressing was done on alternate days; They were kept under proper antibiotic cover till healthy granulation tissue was seen. The limb was elevated by placing wooden blocks at the foot end of the bed. Eczematous dermatitis was treated with topical steroids.

All the patients underwent surgical procedure under spinal anaesthesia after obtaining fitness for surgery. Preoperatively, with the patient standing to fully dilate the veins, the varicosities and location of perforator incompetence are marked with a permanent marker for later operative visualization. The procedures done were ligation and disconnection of the GSV at the SFJ (Trendelenberg operation) followed by complete removal of the vein up to the knee, subfascial ligation of below knee and ankle perforators, stripping of small saphenous vein after ligation at saphenopopliteal junction. Large varicosities were excised using the "stab avulsion" technique. Stab avulsions were performed by making 2-mm incisions directly over branch varicosities, and the varicosity was dissected from the surrounding subcutaneous tissue as far proximally and distally as possible through the small incisions. The elastocrepe bandage applied to the limb at the time of surgery was removed on the third post-operative day and wounds were inspected. The sutures were removed between 8th and 10th post-operative day and patients were advised to continue the elastocrepe bandaging for 6 months after surgery. Patients were reviewed two weeks and 3 months after surgery.

RESULTS: The study included 30 male patients with age ranging from 29 to 70 years. Small saphenous varicosity was found in two patients. Duplex scan assessment showed patent deep veins in all patients. Twenty six patients had ulcer healed by 12 weeks of elastic compressive treatment. For the remaining four patients, split skin grafting was done during the surgical treatment for varicose vein. During the follow up period of 3 months after surgery, all the patients gave symptomatic improvement and a better quality of life. Wound infection of the transverse groin incision was seen in 1 patient, while there were 4 wound infections at the surgical site for perforators in the leg.

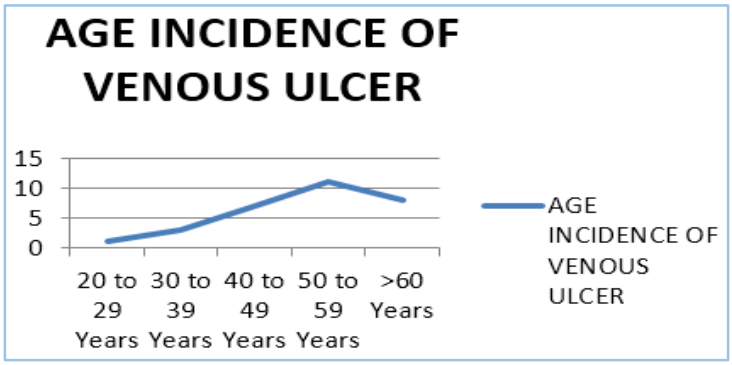

Figure 1

DOI: 10.14260/jemds/2015/1542

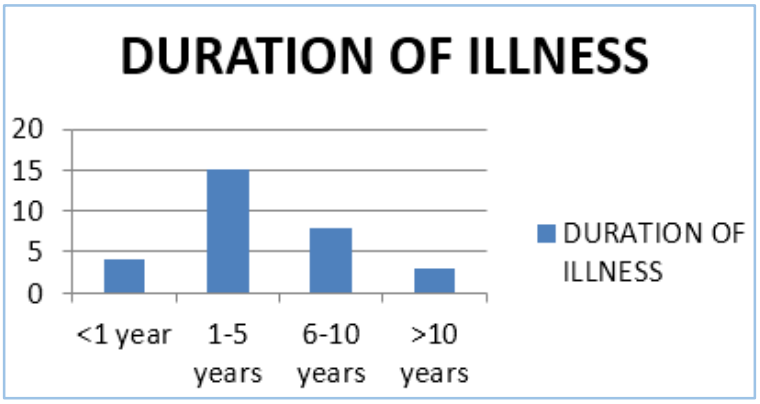

Figure 2

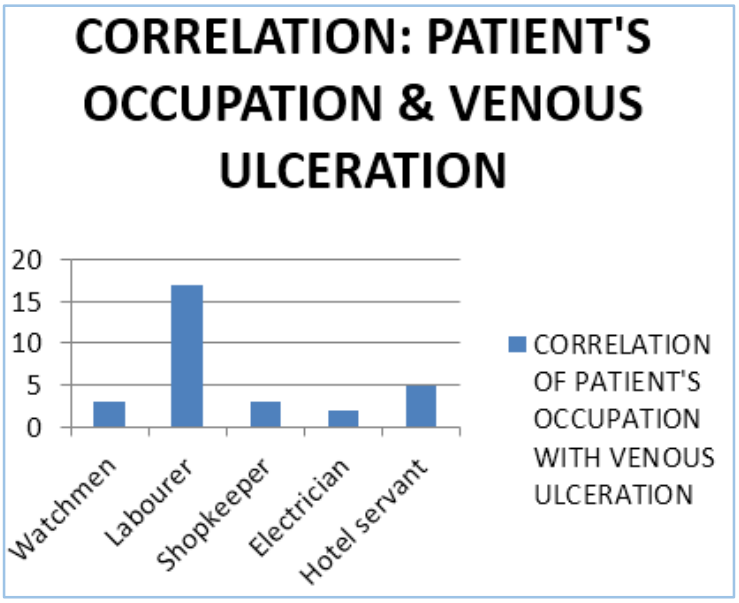

Figure 3

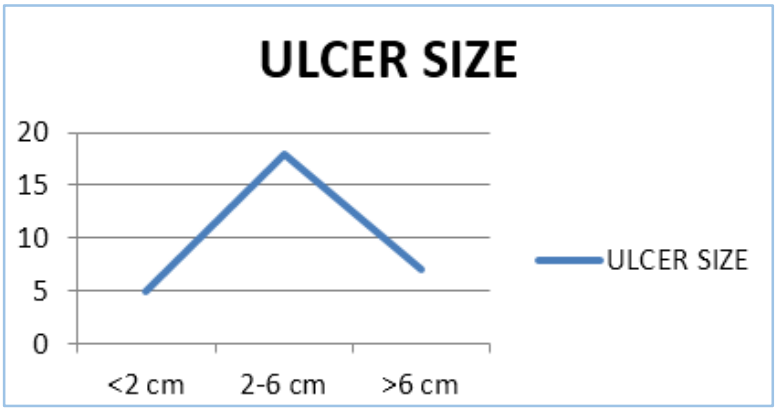

Figure 4

DOI: 10.14260/jemds/2015/1542

	Clinical assessment of 30 limbs	Number
1.	Great saphenous vein varicosity with saphenofemoral incompetence	30
2.	Perforator incompetence	
	Medial ankle perforators	18
	Calf perforators	5
3.	Saphenopopliteal incompetence with small saphenous vein varicosity	2

Table 1: Clinical assessment of 30 limbs

	Type of Surgery	Number
1.	Trendelenberg operation with stripping of Great saphenous vein upto knee	30
2.	Subfascial ligation for incompetent below knee and calf perforators	23
3.	Stab avulsion of large varicosities	14
4.	Small saphenous vein ligation and stripping	2
5.	Skin grafting of resistant venous ulcer	4

Table 2: Type of Surgery

DISCUSSION: Venous ulcer therapy has two goals: heal the ulcer and prevent recurrence. Elevation and compression are important to control edema and facilitate ulcer healing. Multilayer dressings provide compression and absorbency to protect the ulcer bed from the wound fluid which can impede wound healing.[7] For resistant venous ulcers, skin grafting is an option. Once the ulcer has a clean base of granulation tissue, a split-thickness skin graft can be applied. Skin grafting allows for coverage of raw surfaces to speed the healing process. There are reports on improved healing with skin grafting for chronic venous ulceration.[8] Venous ulcer responds promptly to ambulatory treatment or ligation operation but post thrombotic ulcers tend to be refractory to treatment and may require bed-rest, curettage and skin grafting.

Compression therapy is most commonly achieved with graduated elastic compression stockings. The benefits of elastic compression stocking therapy in the healing of ulcerations have been well documented.[9,10] An improvement in skin and subcutaneous tissue microcirculatory hemodynamics as well as a direct effect on subcutaneous pressure have been hypothesized as the mechanism of compression therapy.[11] Ligation with stripping of the great saphenous vein results in significant improvement in venous hemodynamics, may eliminate concomitant deep venous reflux, provides symptomatic relief, and assists in ulcer healing.[12,13] In a study by Dwerry house et al among properly selected patients undergoing vein stripping, recurrent saphenous varicosities will be noted in less than 15% of cases.[14]

Although venous ulceration is a benign condition, it has social and economic implications due to loss of work. A definitive diagnosis of venous ulceration must be made before treatment is initiated. There is considerable morbidity due to recurrent cellulitis, phlebitis and stiffness of joints. Meticulous clinical assessment leads to successful treatment. Before the initiation of therapy for venous ulcer, patients must be educated about their chronic disease and the need to be compliant with their treatment plan to heal ulcers and prevent recurrence. Systemic conditions that affect wound healing and leg edema, such as diabetes mellitus, immunosuppression, malnutrition, and

congestive heart failure, should be improved as much as possible. Duplex ultrasound is a useful tool in assessing the varicose veins and incompetent perforators. Deep vein thrombosis must be ruled out in all patients undergoing surgery. In our study period, elastic compression bandages combined with surgical treatment was successful in improving symptoms of venous ulcer as well as ulcer healing in the patients.

BIBLIOGRAPHY:

1. Beebe-Dimmer JL, Pfeifer JR, Engle JS, et al: The epidemiology of chronic venous insufficiency and varicose veins. Ann Epidemiol 2005; 15:175-184.
2. Lindemayr W, Lofferer O, Mostbeck A, et al: Arteriovenous shunts in primary varicosis? A critical essay. Vasc Surg 1972; 6:9-13.
3. Browse NL, Burnand KG: The cause of venous ulceration. Lancet 1982; 2:243-245.
4. Coleridge Smith PD, Thomas P, et al: Causes of venous ulceration: A new hypothesis. Br Med J (Clin Res Ed) 1988; 296:1726-1727.
5. Scott HJ, Coleridge Smith PD, Scurr JH: Histological study of white blood cells and their association with lipodermatosclerosis and venous ulceration. Br J Surg 1991; 78:210-211.
6. Das S: A manual of clinical surgery: Examination of varicose veins. 10th edition. Kolkata: S Das; 2013. P102-103.
7. Nicoloff AD, Moneta GL, Porter JM. Compression treatment of chronic venous insufficiency. In Gloviczki P, Yao JST, eds. Handbook of venous disorders, 2nd ed. London: Arnold; 2001, pp. 303-308.
8. Schmeller W, Gaber Y. Surgical removal of ulcer and lipodermatosclerosis followed by split-skin grafting (shave therapy) yields good long-term results in non-healing: venous leg ulcers. Acta Derm Venereol. 2000; 80:267.
9. Mayberry JC, Moneta GL, Taylor LM Jr., et al: Fifteen-year results of ambulatory compression therapy for chronic venous ulcers. Surgery 109:575, 1991. [PMID: 2020902]
10. Kitahama A, Elliott LF, Kerstein MD, et al: Leg ulcer. Conservative management or surgical treatment? JAMA 247:197, 1982. [PMID: 7053458]
11. Nehler MR, Porter JM: The lower extremity venous system. Part II: The pathophysiology of chronic venous insufficiency. Perspect Vasc Surg 5:81, 1992.
12. Padberg Jr FT, Pappas PJ, Araki CT, et al: Hemodynamic and clinical improvement after superficial vein ablation in primary combined insufficiency with ulceration. J Vasc Surg 1996; 24:711-718.
13. MacKenzie RK, Allan PL, Ruckley CV, et al: The effect of long saphenous vein stripping on deep venous reflux. Eur J Vasc Endovasc Surg 2004; 28:104-107.
14. Dwerryhouse S, Davies B, Harradine K, et al. stripping the long saphenous vein reduces the rate of reoperation for recurrent varicose veins: five-year results of a randomized trial. J Vasc Surg. 1999; 29:589.

DOI: 10.14260/jemds/2015/1542

ORIGINAL ARTICLE

AUTHORS:

1. Sashi Walling T.
2. K. Vinod

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of General Surgery, Shri Sathya Sai Medical College and Research Institute, Ammapettai, Nellikuppam, Kancheepuram, Tamilnadu.
2. Associate Professor, Department of Anaesthesiology, Shri Sathya Sai Medical College and Research Institute, Ammapettai, Nellikuppam, Kancheepuram, Tamilnadu.

FINANCIAL OR OTHER COMPETING INTERESTS: None

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sashi Walling T,
Doctor's Quarters,
Shri Sathya Sai Medical College and Research Institute, Ammapettai,
Nellikuppam, Kancheepuram,
Tamilnadu-603108, India.
Email: sashiwalling@rediffmail.com

Date of Submission: 09/07/2015.
Date of Peer Review: 10/07/2015.
Date of Acceptance: 23/07/2015.
Date of Publishing: 29/07/2015.