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Management of Venous Ulcer: Our Experience

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Abstract

Venous ulcers are common chronic leg ulcers that do not heal despite optimum wound-care practises. Pain and chronic/recurrent nature of venousulcer, poor quality of life, and poor outcome have significant negative impact on health, psyche and social life. Hence there is a need for exploration of effective techniques for healing of venous ulcers. In this article we report the manner in which we have explored various methods for augmentation of healing of venous ulcer such as hemoglobin spray, insulin therapy, vitamin D granule therapy, prolotherapy, low level laser therapy for wound bed preparation, heterografting with collagen and biosilk, hydrojet debridement, sucralfate therapy, RONPWT and finally split thickness skin grafting.

Keyword: Venous ulcer, APRP, LLLT

INTRODUCTION

Venous ulcers are common chronic leg ulcers that do not heal despite optimum wound-care practises. They are frequently recurrent, and can last anywhere from a few weeks to several years.[1-3] Cellulitis, osteomyelitis, and malignant transformation are all serious consequences.[4] Although the general occurrence of these ulcers is modest, their refractory nature increases the risk of morbidity and mortality, as well as having a major impact on patient's quality of life.[5,6] There is a link between a symptom cluster (pain, depression, fatigue, and sleep deprivation), delayed healing and poor quality of life as shown in a small number of studies. Wound healing can be slowed for a variety of reasons, including contamination, foreign material, large size of wound,systemic factors, and a lack of growth factors. Due to chronic and recurrent nature of venous ulcers there is a need for exploration of techniques that aid in wound healing in such patients.

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MATERIALS AND METHODS

This study was conducted in the department of Plastic Surgery at a tertiary care centre. The details of the patient are as follows:

A 50 years old lady, hailing from Tamil Nadu, with no known co-morbidities was admitted with infected ulcer over the left leg. On assessment, she was found to have incompetent venous perforators of the left lower limb. The patient was initially

treated with conventional dressing. As the ulcer did not show any evidence of healing and was infected (Fig. 1a and 1b), various techniques of wound bed preparation were attempted. We used methods of hemoglobin spray (Fig. 2), insulin therapy (Fig. 3), low level laser therapy (Fig. 4) for wound bed preparation. In addition, we used heterografting of wound with collagen (Fig. 5), and biosilk (Fig. 6) to supply growth factors to the raw area. Methods such asvitamin D granule therapy (Fig. 8), sucralfate therapy (Fig. 9), prolotherapy (Fig. 10) were done to aid granulation of raw area. Were also employedhydrojet debridement of wound (Fig. 7a) with post debridement picture as shown in figure 7(b). Finally, regulated oxygen negative pressure therapy was applied for wound bed preparation. Following adequate wound bed preparation, as shown in figure 12, split thickness skin graft was done to cover the raw area (Fig. 13). The donor site was covered by heterografting (Fig. 14).

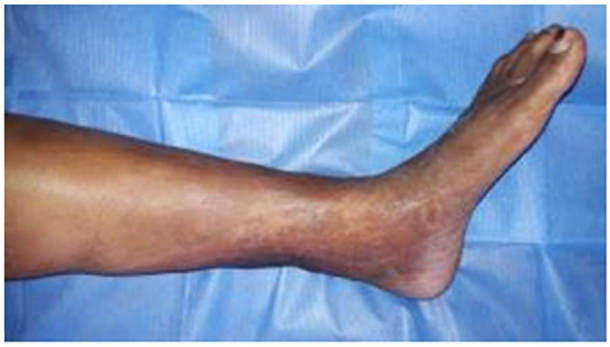

Fig. 1 (a): Wound at presentation

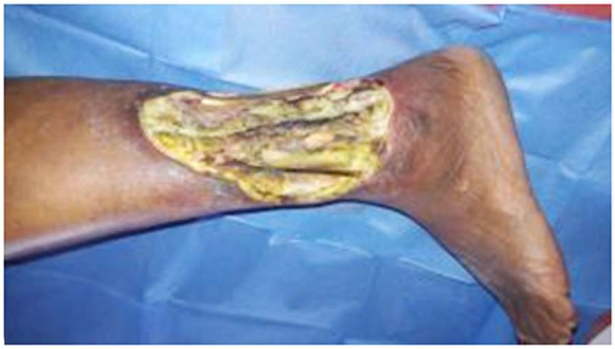

Fig. 1 (b): Wound at presentation

Fig. 2: Hemoglobin spray

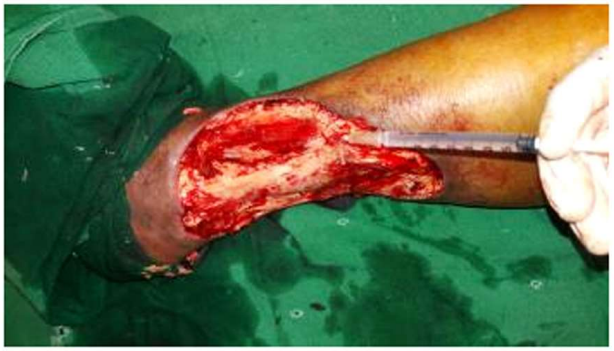

Fig. 3: Insulin therapy

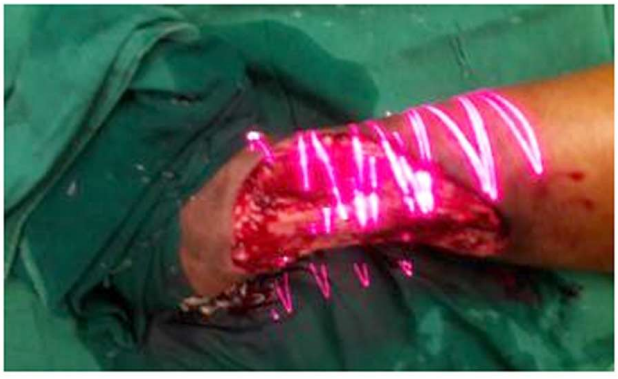

Fig. 4: LLLT (low level laser therapy) for wound bed preparation

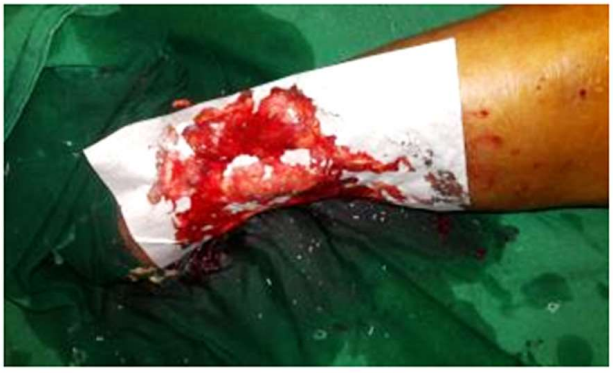

Fig. 5: Heterografting with collagen

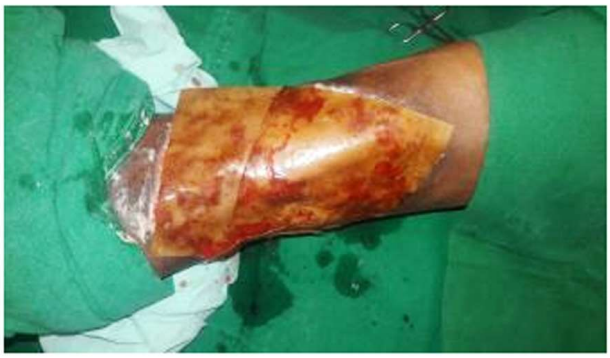

Fig. 6: Heterografting with biosilk

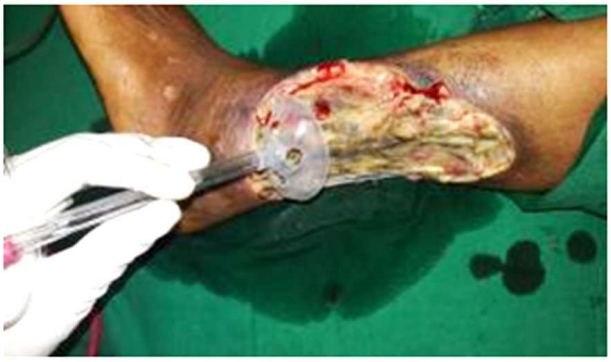

Fig. 7(a): Hydrojet debridement

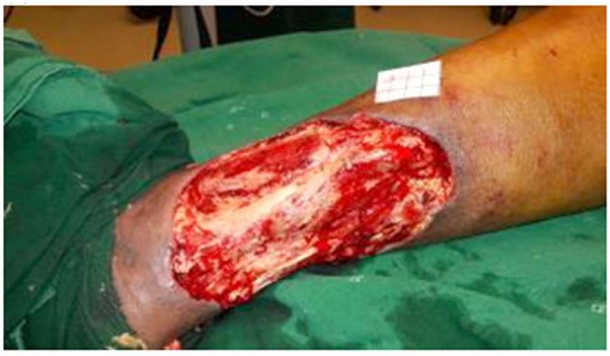

Fig. 7(b): Wound after hydrojet debridement

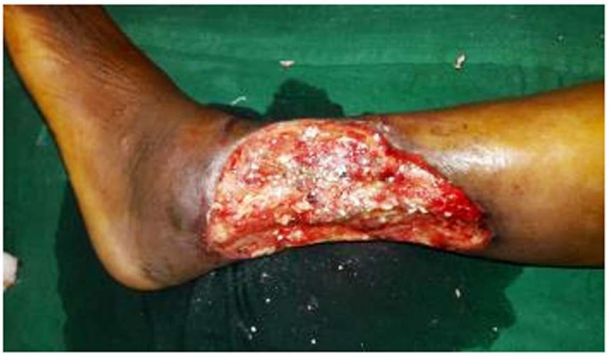

Fig. 8: Vitamin D granule therapy

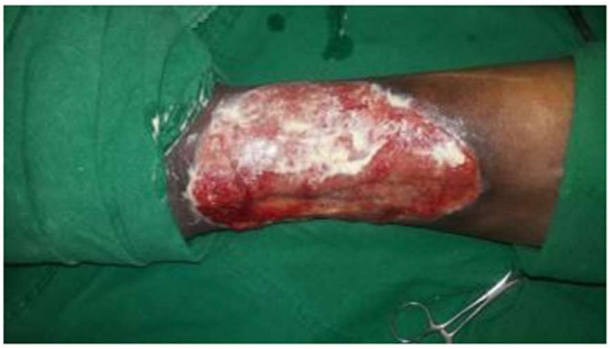

Fig. 9: Sucralfate therapy

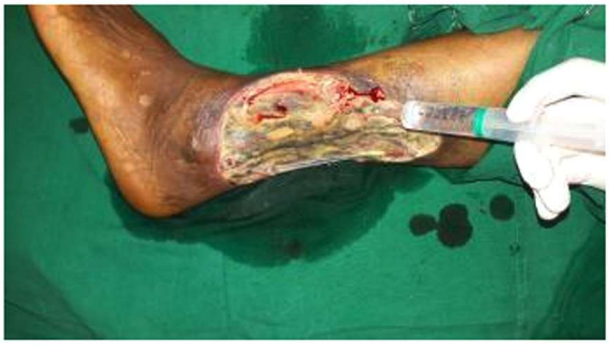

Fig. 10: Prolotherapy

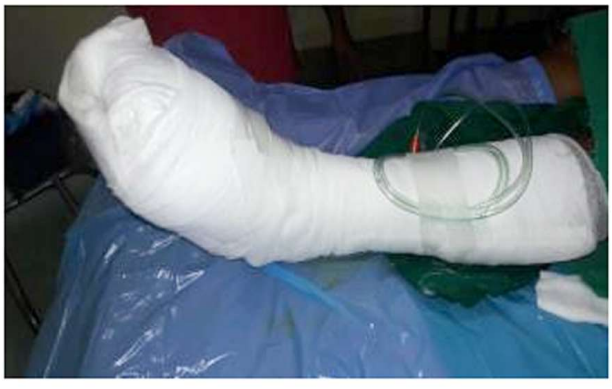

Fig. 11: RONPWT- Regulated oxygen negative pressure therapy

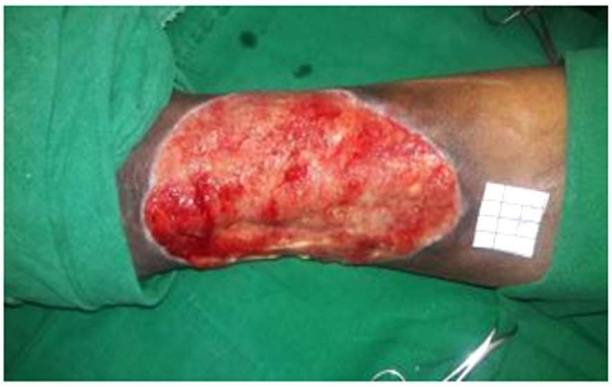

Fig. 12: Well prepared wound bed

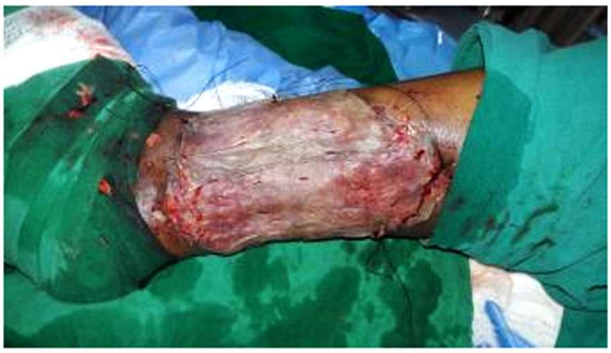

Fig. 13: Split thickeness skin grafting

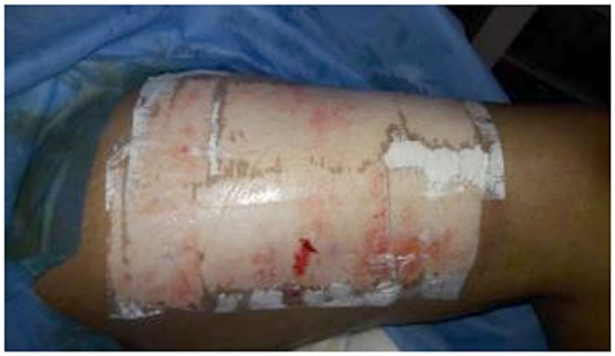

Fig. 14: Heterografting of donor site

RESULTS

The wound healed with skin grafting and there has been no recurrence over a follow-up period of 2 years (Fig. 15).

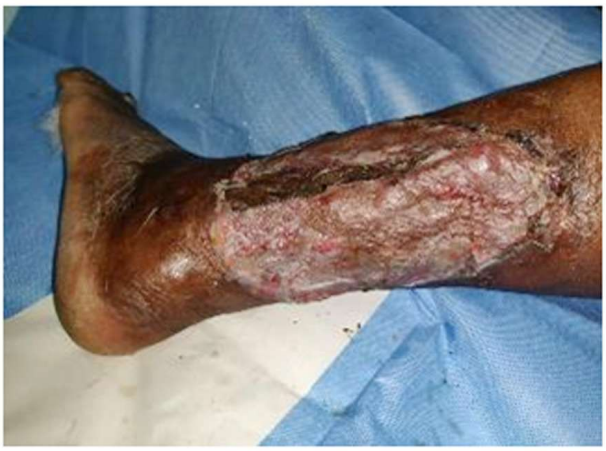

Fig. 15: Healed wound

DISCUSSION

Local oxygen delivery is critical for wound healing, and it is well known that a lack of oxygen can result in a chronic non-healing ulcer.[8] To fulfil the increased metabolic needs of the wound healing process, injured tissue requires a steady supply of oxygen.[9,10] Hemoglobin spray is a novel technique for enhancing oxygen availability in the wound bed. It is composed of purified haemoglobin and works by binding oxygen from the atmosphere and diffusing it into the wound bed to accelerate healing of slow-healing wounds.[11]

Low level laser therapy (LLLT) is the use of photons at a non-thermal irradiance to alter the biological activity.[12] Studies have shown that LLLT at low doses enhance cell proliferation of fibroblasts,[13-15] keratinocytes,[16] endothelial cells[17] and lymphocytes.[18,19]

Clinical trials of dextrose prolotherapy for musculoskeletal indications, although inconsistent, have shown to have pro-reparative responses and decrease in pain in treated patients. Dextrose injections in the wound periphery and base are thought to induce the production of growth factors such as PDGF 37, which could speed up wound healing.[20]

Sucralfate application on wound has shown to affect neoangiogenesis, increase wound contraction, promote re-epithelialization of the wound area and diminish the inflammatory reaction and anoverall improvement patients with chronic venous ulcers after the use of topical sucralfate.[21]

In vivo investigations have revealed that insulin-like growth factor (IGF) stimulates the proliferation, migration, and extracellular matrix excretion of keratinocytes, endothelial cells, fibroblasts, and even promotes the rebuilding of granulation tissue hence contributing to wound healing.[22]

The beneficial effects of vitamin D on wound healing, is thought to be mediated by stimulation the phagocytosis and killing the bacteria by macrophages[23], suppressing interferon-g-mediated macrophage activation[24], activating insulin receptor expression, and the downregulation of cytokine generation.[25] Local use of Vitamin D granules for wound healing is a novel technique and is yet to be explored on a larger study group.

Optimal debridement requires a balance between removal of necrotic tissue and preservation of healthy tissue without obstruction of healing.[26] Hydrojet dissection refers to use of water jet and/or micro water jet technology to emit pressurized sterile saline that simultaneously debrides, irrigates and removes non viable tissue.[27] Furthermore, application of this micro water jet application removes the need to touch the wound during debridement and minimizes possible cross-contamination.

The collagen tissue acts as a matrix material, and an

anchoring substance in the wound healing process. It acts as a decoy for the excess Matrix Metallo Proteinases (MMP) found in chronic wounds[28,29] Collagen heterografting done in chronic ulcers are thought to benefit by this mechanism.

Finally, after adequate preparation of the wound bed in a chronic non-healing venous ulcer, split thickness grafting to cover the wound has been tried. By definition, a split-thickness skin graft (STSG) is one that contains the epidermis and a piece of the dermis. Skin grafts, unlike flaps, do not have their own blood supply, thus rely on graft in-growth in a well-vascularized wound bed. Therefore, a well prepared wound bed is a pre-requisite for adequate STSG uptake.

CONCLUSION

Several methods were implemented to augment the wound bed preparation process of venous ulcer as mentioned and were found to be useful in this patient. Since the methods were applied only on a single patient, its applicability on all cases of venous ulcers is questionable. This could be alleviated by conducting randomised control trials in this regard.

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