Case Report

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Role of Negative Pressure Wound Therapy Assisted Biological Scaffold in Wound Bed Preparation in Diabetic Leg Ulcer

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Abstract

Wound bed preparation is a challenging, complex art. It can be fined as the process of removing local barriers which may facilitate healing and provide a more efficient means of wound healing.[1] It is a complex process that involves an understanding of the physiology of wound healing, the factors, which disrupt wound healing and methods to overcome them.Diabetic foot ulcer (DFU) is a debilitating and severe manifestation of uncontrolled and prolonged diabetes that presents as ulceration, usually located on the plantar aspect of the foot. Standard local and invasive care along with novel approaches like stem cell therapy pave the way to reduce morbidity, decrease amputations, and prevent mortality from DFU.[2]

Various modalities have been tried and described for accelerating the wound bed preparation of such wounds. The principles of wound bed preparation can be summarised as TIME an abbreviation stands for Tissue management, Infection control, Moisture regulation, and wound Edge management. In this article, we discuss the case of an 40 year old male patient who developed right lower limb diabetic foot ulcer from a blister, which progressed to necrotising soft tissue infection (NSTI) and underwent wound bed preparation for NSTI and regenerative techniques.

Key Words: Wound bed preparation; Diabetic foot ulcer; Necrotising soft tissue infection.

INTRODUCTION

Wound bed preparation is an ever-changing paradigm that has evolved rapidly in the past few years. It is defined as global wound management to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures.[3] Wound bed preparation has been classically managed by the TIME concept which is an acronym standing for T : Tissue Management I : Inflammation and infection control M : Moisture Balance E : Epithelial advancement (Edge).[4,5] The recent concept is the "Removal of Barriers" to wound healing which includes necrotic tissue, bio films, corrupt matrix, infection, edema, etc.[6,7] The selection of wound dressing and irrigation solution depends on these factors.

The therapeutic use of Amniotic Membrane (AM) has diversified over the years, since the beginning of the 20th century. However, the potential therapeutic spectrum of placental tissue has not yet been discovered. Among the advantages of AM is that it does not generate a rejection response and that it degrades days after placement in the tissue.

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After establishing the biosafety of the placental tissue by ruling out the presence of transmissible infections and its sterilization, AM can be used for any desired purpose with complete safety.

The proposed therapeutic axes of AM are immunomodulation[8], anti-scarring[9] and tissue regeneration.[10] These functions are achieved by the structural, protein and cellular components that AM possesses, all of which provide the therapeutic applications of AM. These three central functions are the minimum and most important in the resolution of most diseases.[11]

MATERIALS AND METHODS

This study was conducted in a Tertiary Care Centre in Department of Plastic Surgery after getting the departmental ethical committee approval. Informed consent was obtained. The subject was a 40-year-old male who had developed a blister over the sole of right foot, which progressed to diabetic foot ulcer and eventually necrotising soft tissue infection. He was initially admitted in the Emergency Ward where he underwent wound debridement of the NSTI under spinal anesthesia with Autologous Platelet Rich Plasma (APRP) (fig. 1) and Low Level Laser Therapy (fig. 2), following which continuous Negative Pressure Wound Therapy (NPWT) (fig. 3) was applied. Later he underwent repeated debridements (fig. 4, 5) followed by regenerative techniques like APRP (fig. 6), Amniotic membrane grafting over donor site (fig. 7), biological scaffold (fig. 9, 10) and finally Split Skin Grafting (SSG) (fig. 8) done. In the end splinting of limb done (fig. 11).

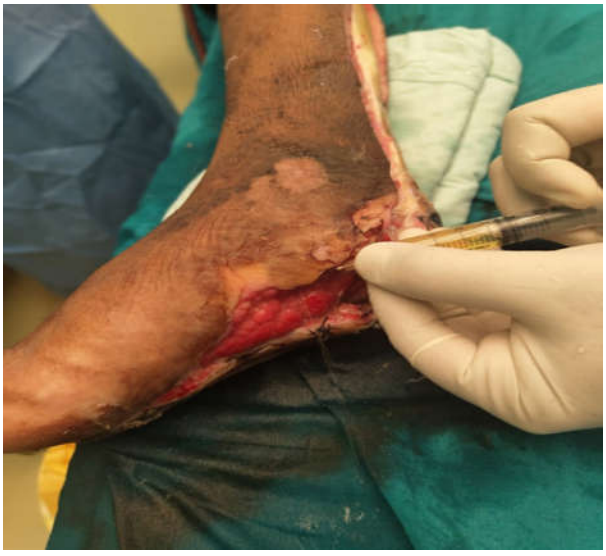

Fig. 1: Wound bed preparation with Activated Platelete Rich Plasma

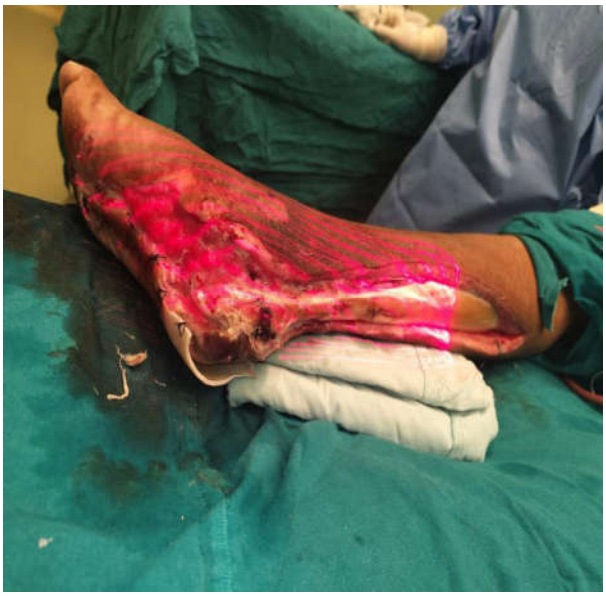

Fig. 2: Wound bed preparation with Low Level Laser Therapy

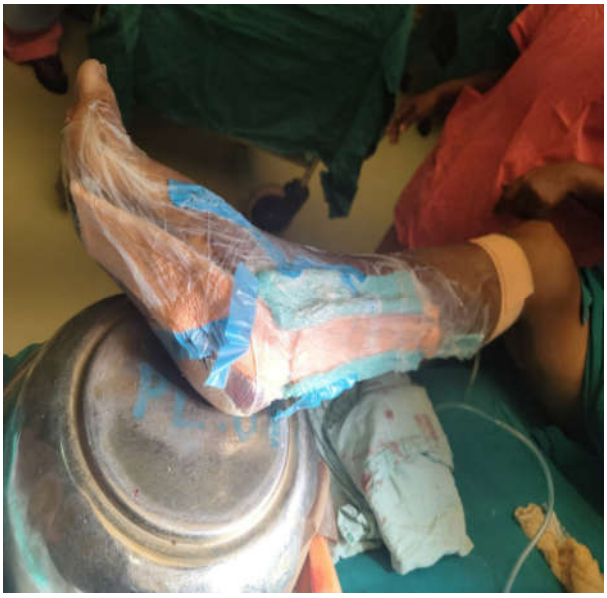

Fig. 3: Application of Continuous Negative Pressure Wound Therapy for wound bed enhancement

RESULTS

The wound bed preparation of right lower limb NSTI wound was done using various regenerative techniques like Autologous Platelet Rich Plasma, Low Level Laser Therapy, amniotic membrane and biological scaffold. He underwent multiple sessions of regenerative therapy. The wound granulated well and split skin grafting done over it.

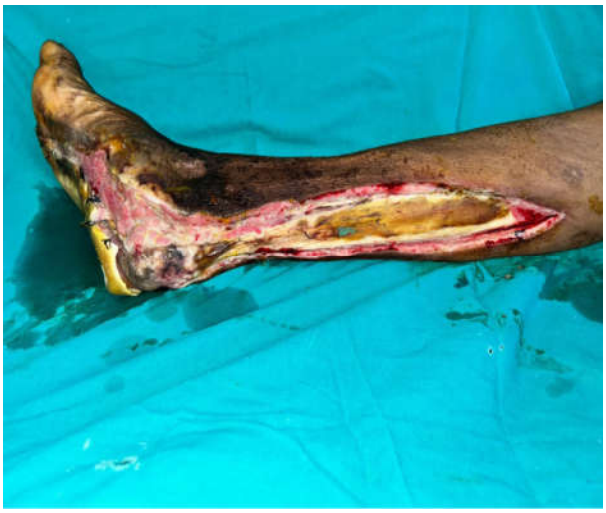

Fig. 4: Wound bed before undergoing repeat debridement

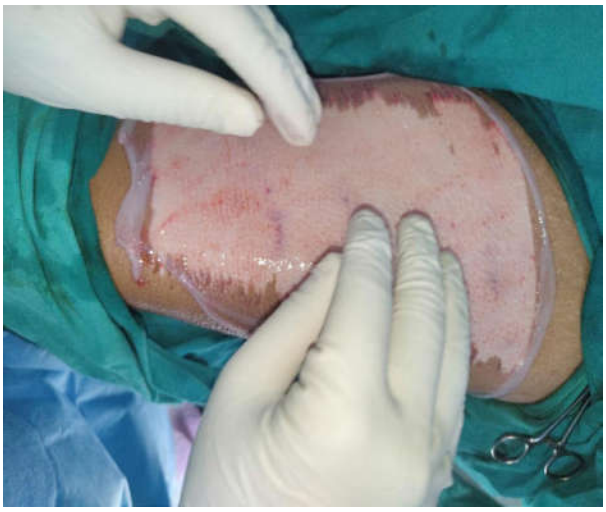

Fig. 7: Amniotic membrane application over donor site for split skin graft

Fig. 5: Woundbed after debridement with bone abraded

Fig. 8: Application of Split Skin Graft over wound bed

Fig. 6: Application of Autologous Platelet Rich Plasma

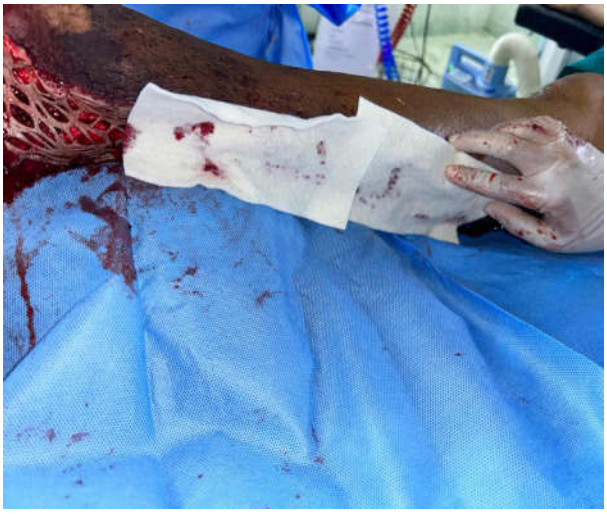

Fig. 9: Application of collagen sheets over split skin graft to enhance uptake of graft

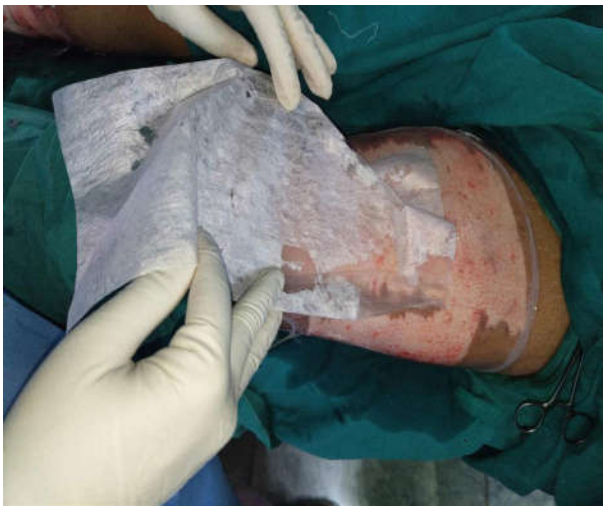

Fig. 10: Application of collagen sheets over donor site to enhance healing

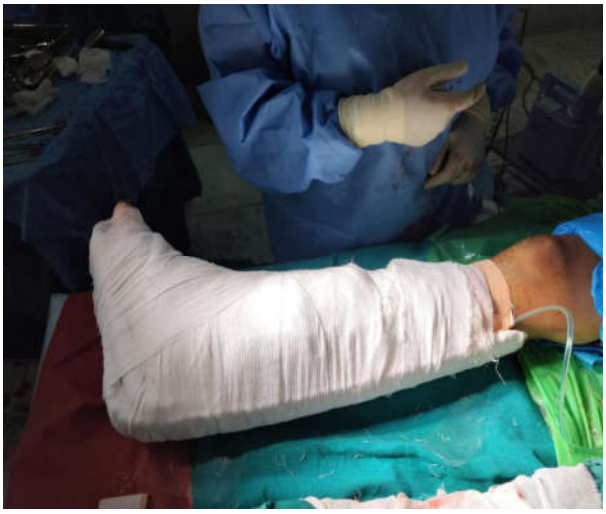

Fig. 11: Application of splint at wound site to provide support to limb and prevent microtrauma to enhance uptake of graft

DISCUSSION

Wound healing is a complex phenomenon that is divided conventionally into four phases- hemostasis phase, inflammatory phase, proliferative phase, and phase of maturation. Each phase overlaps with the other. Soon after the injury, the hemostasis phase begins leading to the formation of the platelet plug.[12] Activation of platelets and the complement system leads to release of several growth factors that activate the inflammatory phase. Recruitment of leucocytes, initially neutrophil followed by lymphocytes and macrophages, is the hallmark of this phase.[13] Macrophages release several growth factors like platelet-derived growth factor (PDGF), transforming growth factor (TGF-beta and TGF-alpha), basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF).[14] These growth factors are responsible for the proliferation, angiogenesis, deposition of collagen, and extracellular matrix (ECM) and the maturation phase. Non-healing wound is caused by an imbalance of growth factors so that these phases do not occur in a timely fashion or their progression is stopped at a different level.

To accelerate wound healing, adjuvant methods of treatment NPWT and APRP were given. NPWT requires a device which is connected through a special set that generates a negative pressure over the wound bed.[15] Various mechanisms that are thought to act both at tissue and cellular level include reduction of the edema, improvement of local blood flow, induction of angiogenesis and granulation, wound margin epithelialization, and facilitation of cell migration and proliferation. Macrostrain mechanisms of NPWT involve removal of exudates and infectious materials and contraction of wound margin. NPWT has been shown to be safe and effective in post debridement wounds. Hence NPWT was started, and size of the wound was measured at the time of change of dressing.

Platelets act as regulators of inflammation, angiogenesis, cell migration, and proliferation with the release of various growth factors and anti-inflammatory cytokines which is thought to help in faster and better healing of the wounds. APRP has growth factors which when injected in the wound site or sprayed, act at the intracellular level to bring about cell proliferation and healing of a wound.

CONCLUSION

There are multiple modalities in wound bed preparation that include debridement, Autologous Platelet Rich Plasma, Amniotic membrane grafting, Regulated Oxygen Enriched Negative Pressure Wound Therapy, regenerative grafting and biological scaffolding. Each modality contributes in some way to make the wound fit for grafting and ultimately speeds up wound healing and patient discharge timing.

Conflict of Interest: None declared.

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