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Regenerative Methods in Pressure Ulcers

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Abstract

Pressure ulcers are the common problem in the bed ridden and Intensive care patients in any hospitals. In initial stages, if proper prophylaxis and attention not paid it may progress to deep ulcers. Pressure ulcers are difficult to treat, various regenerative methods of pressure ulcers will be described in this article.

Keywords: Pressure ulcers; Regenerative medicine; Bed sores.

INTRODUCTION

Pressure ulcers, also known as pressure sores, bed sores or pressure injuries, are localized damage to the skin andor underlying tissue that usually occur over a bony prominence as a result of usually long-term pressure, or pressure in combination with shear or friction. The most common sites are the skin overlying the sacrum, coccyx, heels, and hips, though other sites can be affected, such as the elbows, knees, ankles, back of shoulders, or the back of the cranium.[1,2]

MATERIALS AND METHODS

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A. In this case report, a 25 years old male came with complaints of ulcer in the left ischial region. Patient had history of fall from tree following which he developed acute flaccid paralysis of lower limbs and spinal surgery was done for the same. He had history of pressure ulcers in sacral region which healed spontaneously and right ischial region which was treated with skin flap. The patient developed swelling in the left ischial region which gradually increased in size and ruptured developing into an ulcer. On examination ulcers were present at bilateral ischial region, pus and slough was present and bone was exposed (Fig. 1). Decreased sensation and decreased power of lower limbs was present. Swab was taken for pus culture and sensitivity showed growth of proteus vulgaris. Biopsy of the ulcer was done and histopathology in the right ulcer was found to be hyperkeratotic acanthotic epithelium with focalulceration and right ulcer was skin with acanthosis parakeratosis with follicular plugging. PAS and GMS stain negative for any organisms. During the course of the hospital stay he was treated with wound debridement (Fig. 2),Collagen granules (Fig. 3), insulin spray (Fig. 4), prolotherapy (Fig. 5), APRP, LLLT (Fig. 6), Vit D

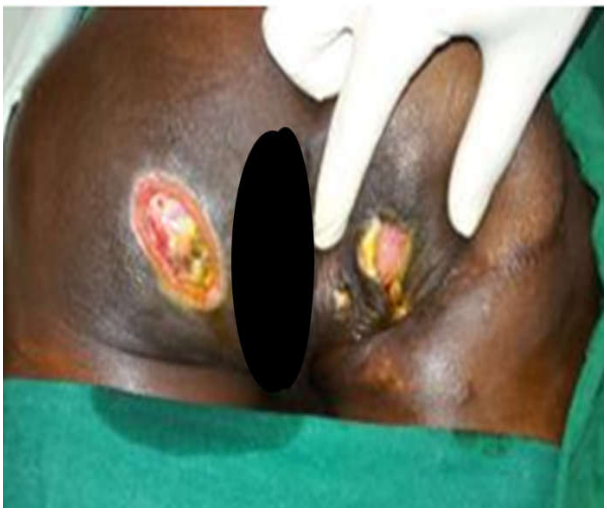


Fig. 1: Wound at Presentation.

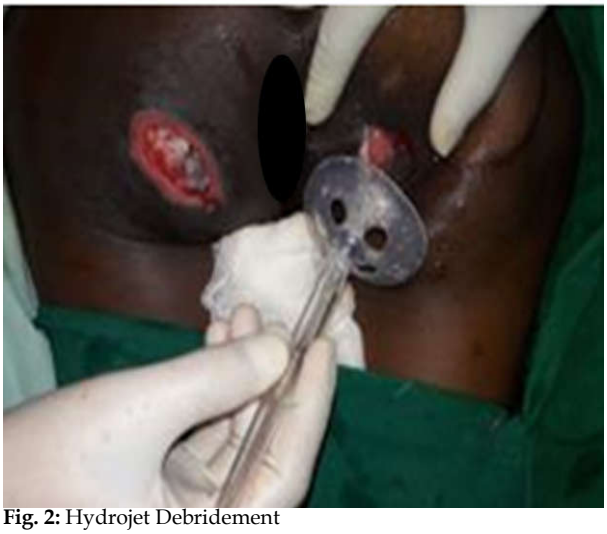


Fig. 2: Hydrojet Debridement

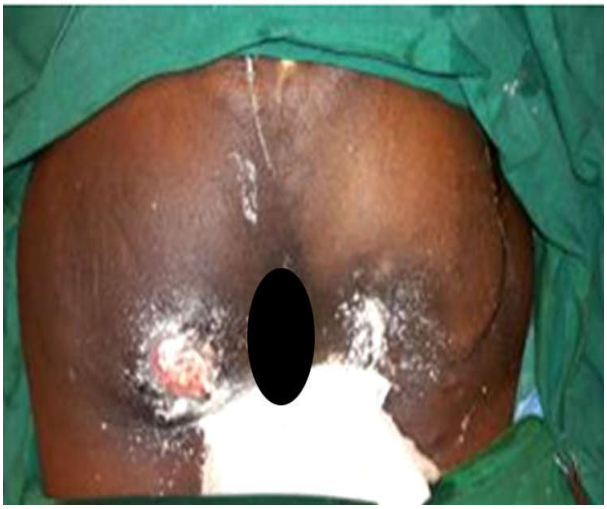


Fig. 3: Application of collagen granules

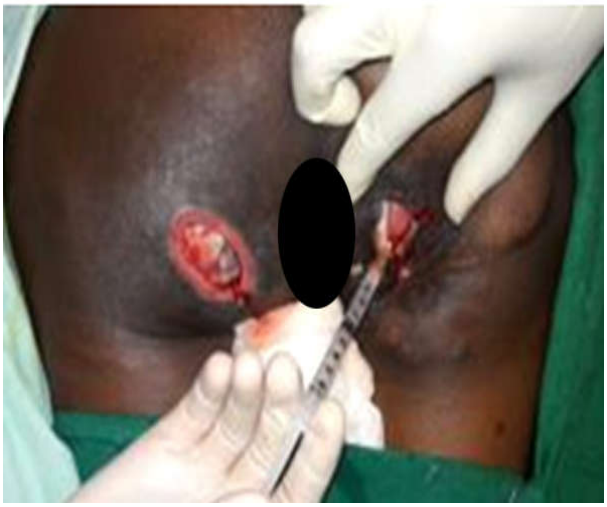


Fig. 4: Insulin therapy

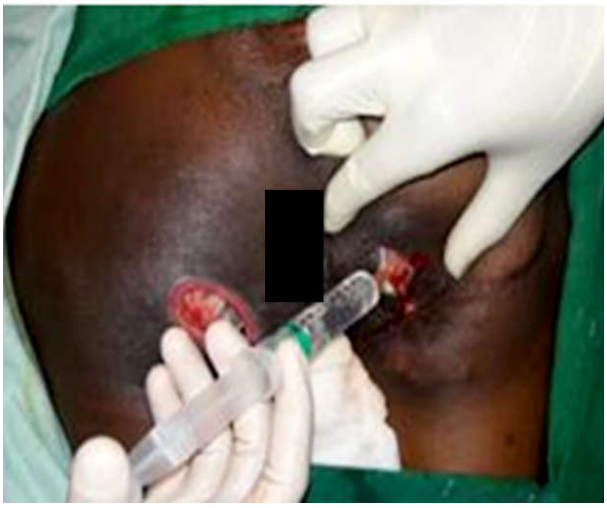


Fig. 5: Prolotherapy

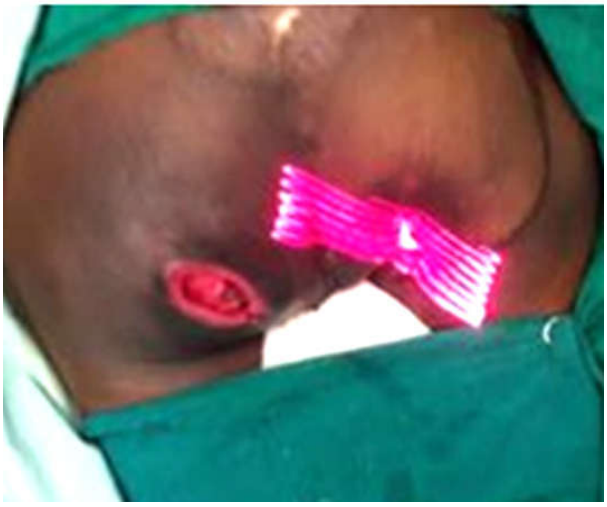
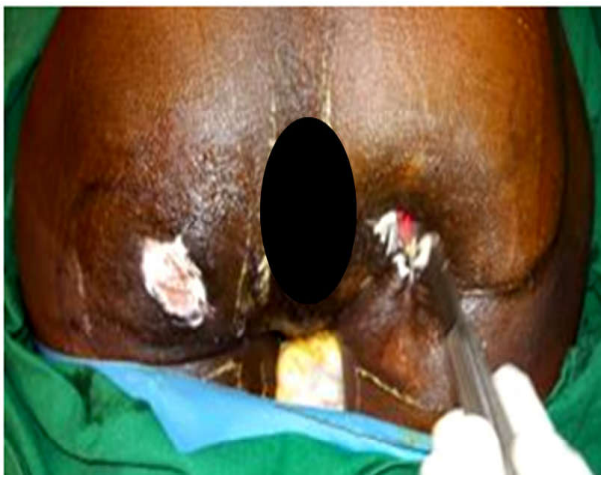


Fig. 6: Application of Low level laser therapy



`**Fig. 7:** Sucralfate therapy

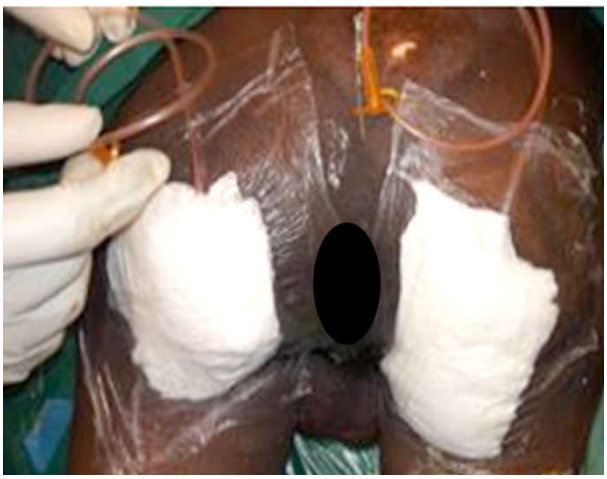


Fig. 8: Negative Pressure Wound Therapy

therapy , Sucralfate therapy (Fig. 7), RONPT therapy (Fig. 8), phenytoin therapy. Patient is further planned for Posterior thigh VY advancement flap on the left side. Patient recovered well and was discharged from hospital.

RESULT

Pressure ulcers are treated with the various methods for regeneration of the tissues. Pressure ulcer treated with the multiple therapies found to be useful in the process of regeneration of tissues in the pressure ulcer. Patient was compatible with all regenerative methods. The methods mentioned for the tissue regeneration are easily adaptable and reproducible in any other hospital for pressure ulcers.

DISCUSSSION

Pressure on soft tissue that entirely or partially obstructs blood flow to the soft tissue can lead to pressure ulcers. Shear can strain on the blood vessels that supply the skin, which is another cause[3,4]. People who are immobile, such as those who are frequently in a wheel chair or on pro-longed bedrest, are more likely to develop pressure ulcers. It is commonly accepted that other factors may affect how well skin can with stand pressure and shear, raising the possibility of pressure ulcer development[5]. These include under nutrition in protein and calories, microclimate (sweating or incontinence related skin wetness), disorders that lower blood flow to the skin, such as arteriosclerosis, or diseases that lower skin sensibility, such as paralysis or neuropathy. Age, illnesses (such as arteriosclerosis, diabetes, or infections), smoking, or medications like anti-inflammatory drugs can all impair the healing of pressure ulcers[6].

Pressure ulcers can be exceedingly difficult to prevent in critically sick patients, elderly seniors, and people with limited mobility such as wheelchair users, despite the fact that they are frequently preventable and treatable if discovered early (especially where spinal injury is involved). The main method of prevention is to regularly rotate the person to redistribute pressure.[7,8] Since at least the 19th century, turning is well known to help prevent new sores. A balanced diet with enough protein and protecting the skin from urine and faces are crucial in addition to rotating and repositioning the client in the bed or wheel chair.[9]

Pressure sores, venous stasis, diabetic ulcers, traumatic wounds, and burs have all been treated with phenytoin. Through a variety of methods, including stimulation of fibroblast proliferation, facilitation of collagen deposition, glucocorticoid antagonistic activity, and antimicrobial activity, phenytoin aids in wound healing. According to a study, phenytoin helped a large necrotizing soft tissue wound recover after other treatments had failed to help it.[8,9]

By controlling oxidative and inflammatory reactions, topical insulin promotes faster wound healing. Reactive oxygen species, which have negative effects on lipids, proteins, and DNA, are reduced by insulin treatment. Additionally, topical insulin causes early neutrophil recruitment and reduces inflammation in wounds by raising macrophage numbers and IL-10 levels to get rid of dead tissues.[10] By controlling MCP-1 expression at wound sites, insulin regulates the chemotaxis and phagocytosis of macrophages as well as the release of inflammatory mediators. Insulin used topically promotes keratinocyte migration, speeds up re-epithelialization, and heightens fibroblastic response.[11]

In prolotherapy, an irritant is injected or sprayed into the wound to create an inflammatory response that is thought to speed up the healing process. Dextrose, with concentrations ranging from 12.5% to 25%, is the most used prolotherapy drug used in clinical practice. Because it is water soluble, a normal component of blood chemistry, and can be safely injected into numerous locations in huge quantities, dextrose is regarded as the ideal proliferant.[12,13] Hypertonic dextrose solutions work by causing local tissue trauma at the injection site, which draws granulocytes and macrophages and aids in the healing process. In this post, we discuss our experiences managing pressure ulcers using prolotherapy. Proponents of the technique hold the view that the injection of hypertonic dextrose causes cell dehydration and osmotic rupture at the injection site, which results in local tissue injury and induces granulocyte and macrophage migration to the site, with release of the growth factors and collagen deposition. The precise mechanism of prolotherapy is unknown. Dextrose concentrations as low as 5% have been demonstrated to induce the production of a number of growth factors necessary for tissue repair in in vitro tests. PDGF, TGF, EGF, b-FGF, IGF-1, and CTGF are a few of these growth factors. Studies conducted in vitro have demonstrated that cells grown in high glucose growth media can produce more PDGF14. In skin wounds, PDGF promotes angiogenesis, fibroblast proliferation, and extracellular production, among other pro-reparative actions. High glucose levels also increase TGF- expression. Inflammation, angiogenesis, fibroblast proliferation, collagen synthesis, matrix deposition and remodeling, and wound epithelialization are just a few of the processes that TGF- is involved in during the healing process. EGF, b-FGF, IGF, and CTGF are other growth factors that are elevated by high glucose levels. Each of these proteins performs a variety of preparative tasks and promotes healing in various animal models of defective wound healing.[15]

The terms low power lasers (LLL), soft laser, cool laser, bio stimulating laser, therapeutic laser, and laser acupuncture are also used to refer to LLL. LLLT has analgesic and anti-inflammatory properties in addition to stimulatory effects on tissue regeneration, wound healing, and repair[16]. At the cellular level, the LLLT stimulates cell growth, increases fibroblast proliferation, decreases the formation of fibrous tissue, promotes cell regeneration, increases the production of collagen, decreases the formation of oedema, increases the synthesis of growth factors, decreases the number of inflammatory cells, decreases the synthesis of inflammatory mediators like substance P, bradykinin, histamine, and acetylcholine, and stimulates the production of nitric oxide The power, wave length, and duration of LLLT treatment all affect the photobiological effects. Gallium Arsenide Ga-As, Gallium Aluminium Arsenide, Krypton, Helium Neon He-Ne, Ruby, and argon are among the regularly utilized LLLT LASERS. It has been utilized to manage burn wounds as well as acute and chronic pain, wrinkles, scars, hair loss, and photo rejuvenation of photo damaged skin. Due to its bio stimulatory qualities, LLLT has been demonstrated to be beneficial as an adjuvant therapy in the care of wounds. Pressure sore treatment using low-level laser therapy (LLLT) can enhance and hasten the healing process.[17]

The foundation of tissue expansion is the idea that all living tissues react dynamically to mechanical stress. The phenomena of biological creep and physiological creep are included in tissue expansion. The use of this technique has increased since it was first proposed by Neumann and made popular by Radovan and Austad. These ideas are not just applicable to the skin; they have also been applied to bones. Internal tissue expansion is not without difficulties, though. The protracted duration, cosmetic deformity, and the requirement that the field be free of infection are the most crucial elements. Internal tissue growth is therefore only seldom useful for covering raw areas. The development of external tissue expansion was made possible by this. Numerous methods for expanding external tissue, such as negative pressure and various expansion tools like Wise Bands and Derma-Close, have been documented. Rubber bands and blouse hooks, which are both readily available items, can also be employed for this therapy.[18,19]

Studies and rare case reports all show that topical sucralfate therapy is effective for treating wounds. Sucralfate suppresses the release of interleukin-2 and interferon-gamma damaged skin cells while promoting the growth of dermal fibroblasts and keratinocytes in vitro. Sucralfate has a physical barrier effect that reduces inflammatory response and promotes mucosal repair. Additionally, sucralfate promotes angiogenesis, which speeds up wound healing. Sucralfate raises the levels of basic fibroblast growth factor (bFGF) and epidermal growth factor in the wound. Similar to how heparin stabilizes blood clots, it binds with bFGF. Small blood vessel development is induced by stabilized bFGF, and cell division in fibroblast and epidermal cells is triggered. Additionally, sucralfate promoted the release of IL-6 and PGE2 from skin cells, which aided in the healing process.[20]

According to the literature, negative pressure wound therapy is thought to have four main

mechanisms of action: contraction of the wound, stabilization of the wound environment, removal of extracellular fluids, and micro deformation at the foam-wound interface.[21,22] It has been widely used to treat wounds, particularly pressure ulcers and diabetic foot ulcers.

CONCLUSION

Our experience in management of Pressure sores has showed to have positive results with usage of methods such as sucralfate cream application, platelet rich plasma application, low level laser therapy, and split thickness skin graft. There was significant improvement noted with the above methods in healing of raw areas. However, to strengthen the concept, multicentric experiments with a larger sample size are required.

Conflicts of interest: None

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