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Scar Acceleration Method—MAC® in the Treatment of Chronic Wounds in Lymphedema: Case Report

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Abstract

Wounds are a serious public health problem in our country. Access to effective, low-cost treatment is still far from the reality for many patients leading to a chronic and serious condition. They interfere in the quality of life of these individuals who isolate themselves socially constrained by the appearance of their wounds. The report shows the case of a patient with type II diabetes mellitus, with chronic wounds in lymphedema in the lower limbs who was treated with the Scar Acceleration Methodology associated with Complex Decongestive Therapy, leading to an improvement in her social participation and quality of life.

Keywords

Lympedema, Wound Healing, Diabetes Mellitus Complication

1. Introduction

Diabetes mellitus (DM) is an important and growing health problem for all

*Creator of the cicatricial acceleration method.

countries, regardless of their level of development [1]. Patients with obesity-associated DM have an increased risk factor for developing complications such as diabetic kidney disease (DKD) or diabetic nephropathy (DN), complicated skin and tissue infections (cSSTIs) and erysipelas [2] [3] [4].

Skin breakdown in patients with advanced diabetes mellitus provides a portal of entry for bacteria, the most common cSSTI pathogen being *S. aureus* [2] [3] [4] [5]. Erysipelas is a cutaneous infectious process, which can reach the fat of the subcutaneous cellular tissue, caused by a bacterium that spreads through the lymphatic vessels and when untreated in its acute phase, at the beginning of the disease, or when it is recurrent with repeated outbreaks, it can lead to some complications, including superficial or deep ulcerations (wounds), and the most common sequela is lymphedema, which is persistent and hard swelling in the leg and ankle [6] [7].

Chronic kidney disease affects more than 500 million people worldwide. In this context, the uremic toxins present are related to the worsening of tissue healing [4]. DN associated with pathophysiologically and immunologically compromised lymphatics in post-erysipelas lymphedema can delay and alter the wound healing process, leading to a chronic process, with risks of reinfection and with a significant loss of functionality and quality of life for the patient [4] [8].

The objective of this case report with literature review is to disclose the result of the conservative treatment of chronic wounds in lymphedema after an episode of erysipelas in a patient with DRD with the Scar Acceleration Method (MAC®) associated with Complex Decongestive Therapy (CFT).

2. Clinical Case Description

Patient MHRG, female, 63 years old, with type II diabetes mellitus, hypertensive and obese. She sought the physiotherapy service for tissue repair of leg wounds and functional change with a medical diagnosis of bullous erysipelas. As the main complaint, very strong pain in the lower limbs and wounds, from which exudate was leaking in large amounts, hindering activities of daily living and social participation.

In 2019, he already had symptoms of pain in the lower limbs and edema that gave in to rest. In the same year, he had a fall from a low height that caused a diaphyseal fracture in the left femur diagnosed late, which caused a vicious bone consolidation, where the bone healed in an incorrect anatomical position, causing changes in gait and functional losses, leading to walking with the aid of a walker.

After a walk on the beach, he noticed a redness in his legs that extended and limited to the region below the patella. Concurrent with this sign, she had a fever and chills. She went to an emergency medical service where she was diagnosed with erysipelas and immediately medicated. The wounds appeared after an injury to the anterior region of the left leg. After this tissue injury, bullous vesicles began to appear with liquid content inside which, when they ruptured, contaminate the adjacent tissues, developing new bubbles. She noticed a

worsening of the condition when symptoms such as nausea, vomiting, inappetence, weight and hair loss started to appear and she needed hospitalization for intravenous medication administration. His wounds did not heal and his legs swelled.

Upon inspection, he presented bilateral lymphedema in the lower limbs up to the infrapatellar region, stage II in the right leg and stage III in the left leg with alteration of the integumentary tissue with a fibrosclerotic aspect, "peau d'orange", hyperpigmentation (Ocre dermatitis) and erythema. Multiple wounds and ulcerations, intense exudate extravasation and adherent scars from past ulcerations.

It has a history of previous therapies that had no effect, such as dressings with varied coverage and treatment with hyperbaric oxygen therapy.

After biochemical examination, important metabolic alterations were detected, among them: urea, uric acid, creatinine, glucose and antibiogram with wound secretion accusing *Staphylococcus aureus* contamination and resistance to beta lactam antibiotics.

She was referred by the physiotherapist responsible for the treatment, for evaluation by an endocrinologist to normalize metabolic rates, to an angiologist for evaluation of venous and arterial structures, where neither the physical examination nor the doppler ultrasound examination found changes in flow and morphology. of vases. She was also referred to a nephrologist for evaluation of altered uremic rates and treatment of diabetic nephropathy with which she is currently following up (**Figure 1**).

Treatment using MAC started on 04/19/2021 and ended on 07/05/2021. Short, medium and long-term goals were set after the physical therapy assessment. Short term: MAC® doping. Single doses/descending—LED, in Amber, Blue and Violet wavelengths—300 s/180 s/120 s. Medium term: After exudate extravasation ceases and skin infection control—Single doses/decreasing—LED, in Red, amber, blue and violet wavelengths—300 s/120 s/60 s/30 s. Long-term: After controlling the skin infection, advancing to the proliferative phase in the wound healing process, and doppler ultrasound examination showing no arterial involvement, TCD was added to the treatment with the MAC method, mainly Compressive Therapy with Multilayer Bandages. Follow-up: (**Figure 2**).

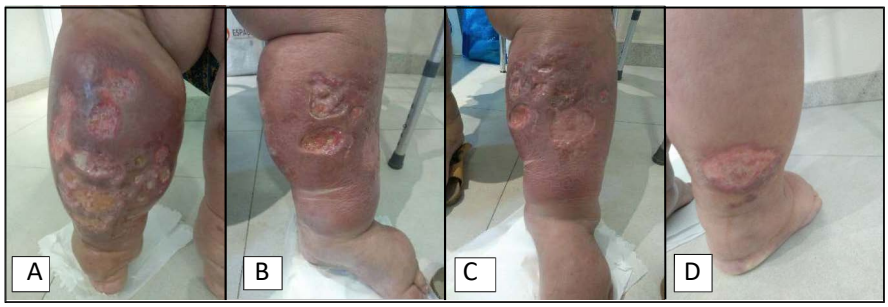

Figure 1. Lower limbs on April 19, 2021, before treatment with the Scar Acceleration Methodology—MAC® associated with Complex Decongestive Therapy. (A) Posterior leg in left lower limb; (B) Medial surface of leg in left lower limb; (C) Anterior surface of leg in left lower limb; (D) Lateral aspect of leg in right lower limb.

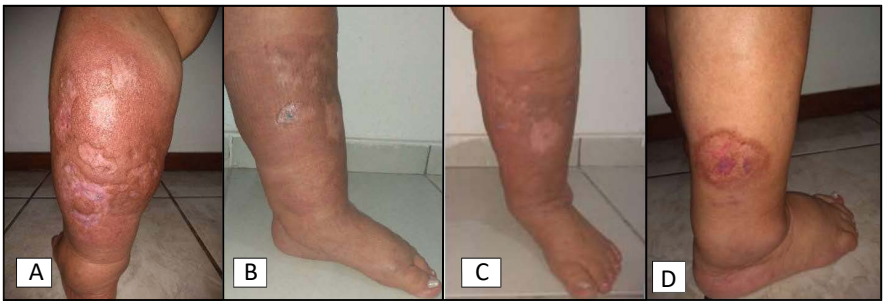

Figure 2. Lower limbs on July 5, 2021, after completion of treatment with the Scar Acceleration Methodology—MAC® associated with Complex Decongestive Therapy. (A) Posterior leg in left lower limb; (B) Medial surface of leg in left lower limb; (C) Anterior surface of leg in left lower limb; (D) Lateral aspect of leg in right lower limb.

3. Discussion and Literature Review

3.1. Diabetes Mellitus

The International Diabetes Federation estimated in 2019 that 31.6 million adults aged between 20 and 79 years in the South and Central America region, or 9.4% of the regional population in this age group, has diabetes. Of these, 13.3 million (41.9%) are undiagnosed. Brazil is the country with the highest number of adults with diabetes (16.8 million). The prevalence of diabetes is higher in women (17.9 million; 10.4%) than in men (13.8 million; 8.4%). Complications of diabetes are categorized as microvascular and macrovascular disorders. Immune dysfunction, diabetic neuropathy, poor circulation, and chronic kidney disease in patients with diabetes mellitus put these patients at high risk for many types of typical and atypical infections and impaired wound healing [9].

3.2. Diabetic Kidney Disease (DKD) ou Diabetic Nephropathy (DN)

One of the most frequent complications in patients with DM is DN. Currently, it is the leading cause of end-stage renal disease and the main cause of cardiovascular morbidity and mortality in these patients [9].

Chronic low-grade inflammation and activation of the innate immune system are key factors in the pathogenesis of diabetes mellitus. Several inflammatory parameters are elevated in diabetic patients and constitute strong predictors of the development of this disease. The increase in inflammatory cytokine levels in the diabetic patient leads to microvascular complications, such as the development of nephropathy [9] [10]. Activated immune cells migrate and infiltrate kidney tissue locally, producing more inflammatory mediators and chemokines that recruit more immune cells to the kidney. In addition, activated resident kidney cells can also produce additional pro-inflammatory mediators, contributing to sustained inflammation and the induction of kidney damage [11].

Early detection of DKD, characterized by an increase in urinary albumin excretion and/or a reduction in TFG, should be performed by measuring the albumin concentration in an isolated urine sample, and the diagnosis should be confirmed on a second occasion, and TFG estimation by equations based on se-

rum creatinine.

Inflammation, along with oxidative stress and fibrosis plus numerous deviations from normal homeostasis, including hemodynamic abnormalities, which trigger increased systemic and intraglomerular pressure, metabolic abnormalities, and activation of the renin-angiotensin system is the link to understanding of the pathogenesis and progression of DN [9].

Uremic toxins, generated in renal dysfunction, are responsible for the progression of Chronic Kidney Disease (CKD) by inducing the loss of residual renal function, triggering systemic and vascular inflammatory responses and, thus, increasing renal endothelial dysfunction. Uremic toxins are responsible for the progression of CKD and loss of residual renal function [4].

3.3. Diabetic Kidney Disease and Wound Healing

Healing is developed by a harmonic set of local cellular and biochemical events, common to different sectors of the organism, it can be said that these influence its basic intermediary mechanisms such as hemostasis, inflammation, cell proliferation and wound remodeling [4].

Renal dysfunction has been associated with poor wound healing outcomes in the diabetic population. Adverse effects of CKD include: decreased phagocytic activity of polymorphonuclear cells, impaired tissue healing, delayed inflammatory healing process, low proliferation of fibroblasts and endothelial cells, low tissue levels of hydroxyproline and collagen, subcutaneous connective tissue and granulation tissue [4] [12].

According to Silva *et al.*, 2018, studies have shown that granulation tissue was reduced in uremic mice by performing histological analysis five days after the procedure for inducing renal dysfunction, as well as inhibiting cell proliferation in fibroblasts and tissue endothelial cells of granulation [4].

In studies, Xie *et al.*, 2019, used diabetic mice with kidney injury that showed remarkably impaired wound healing processes, concomitant with reductions in cell proliferation and angiogenesis, as well as increases in M1 polarized macrophages, infiltrating neutrophils, oxidative stress, and cell apoptosis. Furthermore, quantitative polymerase chain reaction (qPCR) results showed corresponding alterations of related genes (TNF-α, IL-1β, SOD2) in wounds from db/db mice with kidney injury. Renal manipulation in this study accelerated the progress of renal impairment, which has been shown to worsen impaired skin wound healing in diabetic mice leading to a significant increase in urinary protein excretion [10] [12].

Patients with advanced DM have an increased risk factor for developing many types of infections. Of these, cSSTIs are of concern for two reasons. First, immunological defects in patients with diabetes mellitus lead to a reduced response to the most common cSSTI pathogen, *S. aureus*. Second, the breakdown of the skin that leads to ulceration provides a route of entry for the bacteria [2] [5] [13].

All DM-related comorbidities are directly or indirectly related to chronic

hyperglycemia. Maintaining adequate glycemic control can reduce risk and is important for the prophylaxis and treatment of infections in patients with DM [1].

Several factors may contribute to the risk of developing infections in diabetic patients, such as decreased activity of polymorphonuclear neutrophils, alteration in adherence, chemotaxis and leukocyte opsonization, inefficient and delayed cellular immune response to harmful agents, alteration of antioxidant systems and lower production of interleukins (IL-2), reduced vascular response to inflammatory mediators such as histamine and bradykinin, vascular insufficiency, peripheral and autonomic neuropathy, decreased protein binding with consequent edema, reduced mast cell degranulation, worsening of tissue oxygenation and skin and skin colonization mucosa with pathogens such as *Staphylococcus aureus* and Candida [1].

Resident bacteria are often considered commensals, meaning the microbes are not harmful and can be beneficial to the host. The resident flora consists mainly of Gram-positive cocci (Staphylococcus epidermidis), diphtheroids (Corynebacterium and Brevibacterium) and anaerobic rods (Propioni-bacterium). Resident flora organisms contribute to resistance against colonization by pathogenic bacteria by hydrolyzing lipids and producing fatty acids, which are toxic to many bacteria. Staphylococcus epidermidis is a cutaneous commensal but can be an opportunistic pathogen in immunocompromised hosts. The transient flora is mainly represented by *Staphylococcus aureus* (coagulase-positive) and Streptococcus pyogenes. These bacteria, originating from the environment [14].

3.4. Erysipela

According to the Brazilian Society of Angiology and Vascular Surgery, one of the most common local complications in diabetics and obese patients is erysipelas. Erysipelas is also known as "St. Anthony's Fire" (due to its intense fire eruption) is a skin infection involving the dermis layer of the skin that may extend to the superficial cutaneous lymphatics, caused by group A streptococci and *Staphylococcus aureus*, but other streptococci and Gram-negative bacteria. appear to be the causative agents in some cases. The skin infection spreads through a crack in the skin, directly invading the lymphatic system and causing erysipelas. Therefore, it is important to assess the patient for any skin trauma such as portals of entry or recent pharyngitis. Erysipelas can be serious.

Risk factors that predispose people to developing erysipelas are obesity, lymphedema, athlete's foot, leg ulcers, eczema, intravenous drug abuse, poorly controlled diabetes, and liver disease.

No laboratory tests are necessary for the diagnosis of erysipelas, its findings are nonspecific and may involve an elevated leukocyte count with a predominance of neutrophils and elevated C-reactive protein. Clinical findings are predominant for the diagnosis, which is characterized by an area of well-demarcated, elevated erythema and faster development. Patients may complain of burning, tenderness, and itching at the site. It most often affects the lower limbs, the face

being the second most affected site.

Local complications are more common than systemic complications. When the patient is treated early in the disease, complications are not as obvious or severe. However, cases not treated in time can progress to abscesses, superficial or deep ulcerations (sores), and vein thrombosis. The most common sequel is lymphedema, which is persistent, hard swelling located mainly in the leg and ankle, resulting from repeated outbreaks of erysipelas [6].

3.5. Lympedema

According to the 6th Latin American Consensus for Treatment of Lymphedema, lymphedema comprises the accumulation of water, salts, electrolytes, high molecular weight proteins and other elements in the interstitial space, leading to an increase in the volume of the body region as a result of an alteration dynamics and/or mechanics of the lymphatic circulation that leads to a progressive and evolutionary increase in the volume of the limb or body region with a decrease in functional and immunological capacity, weight gain and morphological changes [15].

Genetic, iatrogenic, traumatic or infectious abnormalities of the lymphatic system are factors for the development of dysfunctions and serious complications in the lymphatic vasculature, including lymphedema. Recent research suggests that many of these abnormalities are related not only to changes in lymphatic fluid transport function, but also loss of lymphatic regulation of normal immune responses [16] [17].

Lymphedema can be classified as primary, when hereditary defects in lymphatic vascular development or function are the cause, and secondary, a result of obstruction or disruption of the acquired lymphatic vascular system [16] [17].

The International Society of Lymphology rates a lymphedematous limb on a three-stage scale with recognition of Stage 0 (**Table 1**).

3.6. Wound in Lympedema

The lymphatic system is a third circulation that is part of our body's vascular network. Lymphatics contribute to extracellular space homeostasis and normal wound healing. Lymphatic vessels control interstitial microcirculation, removing macromolecules and particles from the extravascular space that are too large to re-enter the blood capillaries. If these materials and water are not removed by failure of the lymphatics, the balance of osmotic and hydrostatic forces within the tissues shifts, builds up—proteins and other macromolecules and fluids around the cells result in tissue toxicity, cell damage, delayed healing and pathologies [8] [18].

Lymphedema is known to delay wound healing, swelling of the interstitium leads to disruption of normal nutrient delivery pathways to cells, which impairs normal wound healing. In addition to the pathophysiological mechanisms, consisting of impaired tissue remodeling, increased internal tissue pressure (leading

Table 1. Prepared by the authors based on the article: Executive Committee of the International Society of Lymphology. The diagnosis and treatment of peripheral lymphedema: 2020 Consensus Document of the International Society of Lymphology. Lymphology. 2020 development of verrucous overgrowths).

Stages	Characteristics
stage 0	Latent or subclinical condition Swelling is not yet evident May be transient and may take months or years before overt swelling occurs (Stages I-III)
Stage I	Initial fluid accumulation relatively high in protein content (compared to "venous" edema) Decreases with elevation of the limb
Stage II	Involves more changes in tissue structures that increase the risk of fibrosis, infection, and skin lesions. Limb elevation alone rarely reduces tissue swelling.
Stage III	Lymphostatic "elephantiasis". Locker sign is absent. Trophic skin changes (acanthosis, changes in skin character and thickness, subsequent fat deposition and fibrosis.

to microcirculatory ischemia) and accumulation of cellular debris, it is suspected that the balance of immune cells involved in wound healing is disrupted in lymphedema [8].

One of the functions of the lymphatic system besides draining interstitial fluid and absorbing gastrointestinal lipids is trafficking of immune cells. It regulates immune responses by transporting bacteria, foreign antigens, particles, exosomes and immune cells to regional lymph nodes and lymphoid structures. The intensive defense provided by acquired immunity requires functioning lymphatic organs [8] [16] [17]. Lymph nodes provide a specialized microenvironment for migrating immune cells to meet, especially T lymphocytes and antigen-presenting cells, including dendritic cells (DCs) that exist in peripheral tissues and enter blood capillaries. Lymph nodes coordinate the traffic of cells from two sources: 1) blood vessels, through which most lymphocytes enter lymph nodes, and 2) lymph vessels, which transport interstitial fluid, including DCs.

If the lymphatic pathway is blocked, as in lymphedema, the immune system would be unaware of an inflammatory process occurring in the afferent tissue and would remain unengaged, resulting in immune failure [8] [19]. Lymphedema is a condition in which the lymphatic ducts are impaired and the expression of acquired immunity is impossible.

Excessive chronic inflammation and fibrosis can have a chaotic effect on the healing process [11]. Lymphedema results in a significant inflammatory response [20]. T cells comprise almost 70% of all inflammatory cells in chronic lymphedema and are associated with a mixed T-helper 1 (Th1) and T-helper 2 (Th2) response [18] [21]. Th2 cytokine expression is markedly increased in lymphe-

dema. Th2 cytokines (IL-4 and IL-13) and IFNγ derived from Th1 cells have profound anti-lymphangiogenic effects, inhibit lymphatic function, increase fibrosis and promote changes in the extracellular matrix, may impair lymphatic repair and regeneration by through direct effects on survival, proliferation, migration and formation of tubules on lymphatic endothelial cells (ELCs) [21] [22]. T lymphocytes migrate to the wound after inflammatory cells and macrophages after tissue injury during the proliferative phase of healing [11] CD4+ T lymphocytes can differentiate into Th1, Th2, Th17 (T helper or Th in English), or even other subtypes, each of which secretes a particular set of cytokines [23].

In tissues, macrophages are matured and activated in certain phenotypes, depending on the stimulus, to acquire specialized function. M1 is characterized as pro-inflammatory, providing tissue damage, while the M2 population is involved in tissue repair, with an anti-inflammatory profile. M1 and M2 are critical for natural wound healing. Polarization of M1 to M2 is a vital step in wound healing and can be amplified by cytokines, in particular IL-4, as well as increasing the number of M2, resulting in elevated levels of IL-10, transforming growth factor-β, and IL-12 [8]. Subsets of T helper (Th) 1 and 2 cells and macrophages are closely related in such a way that they mutually regulate their differentiation [8].

The differentiation of Th1 cells from naive T cells is driven primarily by interferon-gamma (IFN-γ). IFN-γ inhibits the differentiation of naive T cells into Th2 cells. When the influence of IFN-γ is weak, it is assumed that natural killer T cells produce interleukin (IL)-4 without being affected by dendritic cells. IL-4 causes naive T cells to differentiate into Th2 cells that promote chronic inflammation with the help of M2 macrophages that promote fibrosis. Th2 self-produces IL-4, which creates a state of Th2 and M2 dominance through positive feedback. In the lymphedema region, dendritic cell activity is suppressed, which diminishes the ability of IFN-γ to inhibit Th2 cell differentiation. Fibrosis is promoted in response to chronic inflammation driven by Th2 cells and M2 macrophages [8] [19] [24].

Yoshida *et al.*, 2019, presented in their work, two case reports where they obtained satisfactory results in lymphedema wound treatment with the use of the microsurgical procedure called Lymphovenous Anastomosis, associated with skin grafts and cotton elastic bandage. Despite the scientific literature showing through results that the supermicrosurgical lymphaticvenular anastomosis has a valuable place in the treatment of lymphedema, with significant improvement of symptoms, the microsurgical procedure is limited to specific cases of lymphedema, it is not the first treatment option, which is the conservative, do not address the underlying pathophysiology of lymphedema and the long-term effects are still unclear [24].

3.7. The Method of Acceleration of Science (MAC®) and Complex Decongestive Therapy (CDT)

MAC® Method

The MAC® Method is a treatment methodology for tissue repair (integumentary, musculoskeletal, bone and cartilaginous) and its concept is based on clinical reasoning through three pillars: Identification, observation and interpretation of the patient's health condition through complementary exams (image and biochemical) and pathognomonic signs. Clinical sovereignty directs the elaboration of the physiotherapeutic diagnosis and consequently the short, medium and long term goals, in the treatment plan, leading it to be more effective and accurate in physical-functional rehabilitation and improvement of the patient's quality of life.

This method uses laser and LED as a treatment tool to perform photodynamic therapy, through cell markers, photosensitive substances, photopharmaceuticals and photobiomodulation, to modulate cellular biological processes.

According to Pinto *et al.* (2021), the method uses a technique for spatio-temporal control, similar to the "self-flying focus", which is a behavior of the laser beam in nonlinear modeling that produces an arbitrary, flexible path intensity peak that can be sustained over distances comparable to the focal length. The bending of time over time is used, causing the parabolic profile of the wave's amplitude to begin to change, tending to maintain and control the speed of the peak irradiance of the laser/LED, controlling the beam divergence over a long distance, increasing and concentrating potency and the non-declining and longer therapeutic window (**Figure 3**) [12] [19].

The method uses doping in two treatment modalities as Monodoses and Doses. Therefore, it is necessary to evaluate the type of tissue, taking into account its density, redox states, severity and depth of tissue injury, type of microorganism that may be colonizing the tissue. Monodoses are subdoses. Time folds over time are used, time is fractioned into each wavelength. This time double can be increasing or decreasing depending on the duration of the comorbidities (chronic or acute), the characteristics of the lesion, the objective of the treatment, the clinical evaluation. Dosimetry is increased or decreased depending on each case.

Doses are fixed time folds at each wavelength. The dose is medialized. It is more used in organisms where the metabolism is high through a clinical evaluation (such as athletes and children).

Complex Decongestive Therapy (CDT)

CDT or Combined Physical Therapy or Complex Decongestive Physical Therapy (among others) involves a two-step treatment program that can be applied to children and adults, according to the International Society of Lymphology [15]. It consists of therapeutic procedures or guidelines, which will be performed according to the classification or staging of lymphedema [8]. Wound healing and skin and attachment care, Manual lymphatic drainage (MLD), Multilayer bandage (or elastic stocking/sleeve) and Myolymphokinetic exercises (before or after these procedures, taking care as long as the patient is wearing the multilayer bandage or socks elastic bands or sleeves in place).

The first phase of treatment is aimed at maximal reduction of lymphedema

volume and consists of skin care, MLD (depending on lymphedema staging), muscle pumping exercises, and compression typically applied with a multilayer bandage. Phase 2 (started immediately after Phase 1) aims to conserve and optimize the results obtained in Phase 1. It consists of compression by a low-stretch elastic stocking or sleeve, skin care, continued exercise and MLD as needed (**Figure 4**) [15].

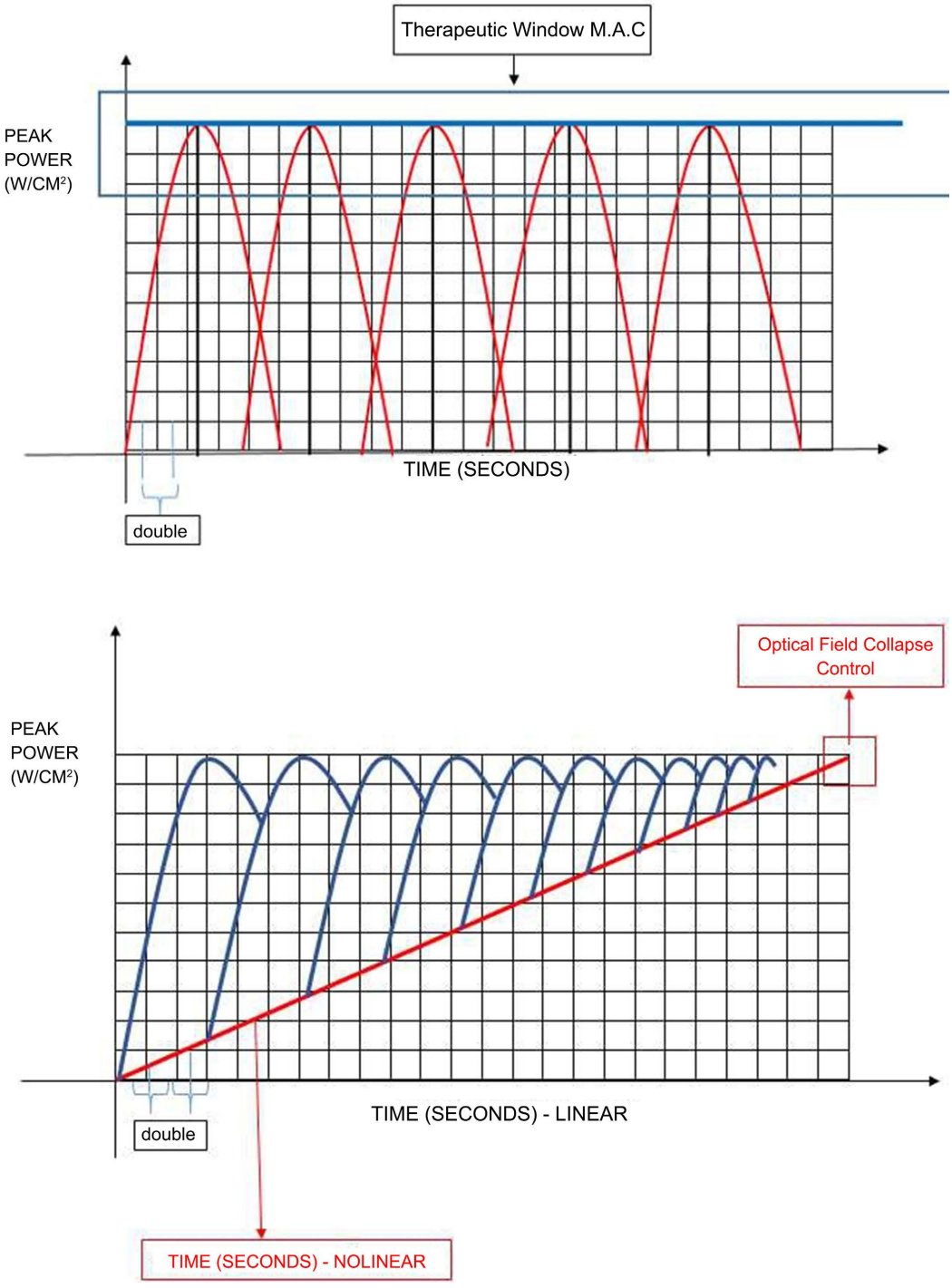

Figure 3. Graphic schemes of temporal folds—therapeutic window MAC® Method.

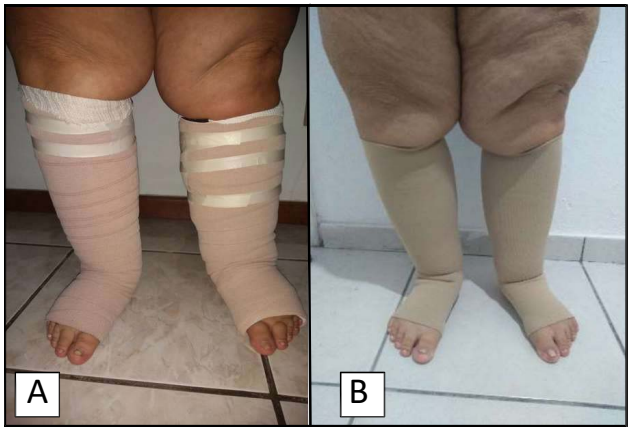

Figure 4. Patient in the two stages of treatment with Accelerated Scarring Methodology—MAC® and Complex Decongestive Therapy (A) First phase of Complex Decongestive Therapy—Multilayer bandage with short extensibility bandage; (B) Second phase of Complex Decongestive Therapy with flat knit elastic socks.

4. Conclusion

It is known that the microsurgical procedure is limited to specific cases of lymphedema, not being the first treatment option, being conservative, they do not address the underlying pathophysiology of lymphedema and the long-term effects are still unclear. More studies addressing the long-term follow-up after surgical treatment are still needed. MAC associated with CDT proved to be an effective method of conservative treatment of chronic wounds in lymphedema, with short-term results, more affordable, promoting an improvement in the patient's quality of life.

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Conflicts of Interest

The authors declare having no conflict of interest regarding this article.

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