SCIENCE PHOTONS Publishers

Case Report

Innovative Insights in Case Reports and Reviews

Stimulation of the Wound Healing Process of an Electrocution-Associated Ulceration Using Amniotic Membrane: A Case Study

Alison Ingraldi[1], Daniel Davis[2], Darlene Lee[1], and Aaron J. Tabor [1,3,*]

1. *Department of Clinical Research, Axolotl Biologix, Scottsdale, AZ. USA.*
2. *Family Foot and Ankle Specialists, LLC, Bridgeport, CT. USA.*
3. *Department of Biological Sciences, Northern Arizona University, Flagstaff, AZ. USA.*

Received Date: 09 July, 2025;
Accepted Date: 15 July 2025;
Published Date: 18 July 2025.

[*]***Correspondence:*** *Aaron J. Tabor. Department of Clinical Research, Axolotl Biologix, Scottsdale, AZ. USA.*

Citation: Alison Ingraldi, Daniel Davis, Darlene Lee, Aaron J. Tabor. Stimulation of the Wound Healing Process of an Electrocution-Associated Ulceration Using Amniotic Membrane: A Case Study. Innov Insights Case Rep Rev. 2025; 1(1): 1-10.

ABSTRACT

Electrical injuries can result in significant tissue damage due to both thermal and electrical effects. The healing process for these wounds is complicated by factors such as vascular damage, tissue necrosis, and an increased risk of secondary infections. Approximately 3–5% of admissions to burn centers worldwide are due to electrocution injuries. Unlike thermal burns, electrical burns can penetrate deeply into the tissue because of the electrical current, leading to muscle necrosis and vascular injury, along with potential complications such as chronic ulcers. Healing from these injuries often progresses slowly; wounds may stall and necessitate aggressive treatments, including debridement, skin grafting, and additional therapies like hyperbaric oxygen treatment and biological skin substitutes. This clinical case study aims to assess the clinical efficacy of a commercial dehydrated amniotic membrane (dHAM) allograft in promoting wound healing in a chronic, non-healing neuropathic ulcer secondary to a high-voltage electrical injury in a 50-year-old male patient, resulting in extensive burns on his right arm, chest, and left lower extremity that have remained open since the etiologic event. The case report outlines the clinical interventions for wound management strategies using a commercial allograft dehydrated amniotic membrane (dHAM) product that demonstrated the stimulation of the wound healing cascade with a 44% reduction in wound size specifically for the patient's neuropathic ankle ulcer.

Keywords: *electrocution, wound healing, electrical injury, burn management, tissue regeneration, amniotic membrane.*

Abbreviations

dHAM : Dehydrated Amniotic Membrane, FDA : Food and Drug Administration, NPWT : Negative Pressure Wound Therapy.

Introduction

It is estimated that approximately 1000 individuals die of exposure to electricity annually in the United States [1,2]. Electrical injuries account for a substantial portion of burn-related hospital admissions, particularly in industrial settings. Each year, approximately 3,000 patients who survive electrical shock are admitted to specialized burn units [3,4]. High-voltage electrical injuries often result in deep tissue damage and require multifaceted treatment approaches to manage the immediate effects and promote wound healing.

The skin, especially the stratum corneum, has an electrical resistance of approximately 10,000 Ω, but this resistance can be bypassed by high voltage such as 500 V or more leading to injury [5]. Electrical burns are a common and severe form of trauma that may cause significant damage to the skin and also deeper tissues and organs. The extent and depth of burn injuries can determine the severity of the injury and influence the prognosis, with the deeper and more extensive burns possibly requiring emergent fasciotomy, debridement, and wound exploration. These injuries can have a

lasting negative impact on patients, affecting both their physical and mental health [6]. Individuals who suffer from burn injuries may experience severe psychological distress and anxiety due to the extent of their injuries and the societal stigma surrounding visible scars [7]. Although burn healing is a natural process that can be enhanced with appropriate medical treatment, patients may also face serious complications during recovery. Burn wounds have several key characteristics, including slow or stalled healing, the risk of infection, pain, contractures, and the potential for keloid formation [8]. The healing process for these wounds is complex and involves effective treatment, skin regeneration, and prevention of infection. Both local and systemic therapies may be necessary; however, local therapy is often the preferred option. This approach can reduce the risk of adverse effects, enhance treatment effectiveness, and help prevent antibiotic resistance [9]. Ideally, antibiotics should be used only at the site of application rather than through systemic or long-term treatments, as this helps minimize harmful effects on the microbiome and the development of drug resistance [4,9].

Patients with extensive burns often require a comprehensive pharmacological approach beyond standard infection control measures, including pain management, sedation, anxiolytics, blood products, and steroid-based interventions such as testosterone replacement [10]. While androgens have been shown to negatively impact healing in chronic cutaneous wounds, such as pressure ulcers and diabetic foot ulcers, emerging research suggests a potentially beneficial role in burn wound healing [11,12]. This apparent duality highlights the necessity of tailored wound care strategies that account for the underlying etiology of the wound, rather than a generalized approach.

Electrical burns pose a unique challenge due to their varying tissue damage, depth, delayed necrosis, and high risk of systemic complications. Effective treatment requires multidisciplinary management tailored to the patient, emphasizing early wound assessment, infection control, debridement, and strategies for tissue regeneration. Initial management includes fluid resuscitation using, for example, the Parkland formula, to address hypovolemia and prevent shock [13], as well as escharotomy or fasciotomy for compartment syndrome to relieve pressure and restore perfusion [14]. Wound care involves surgical debridement to remove necrotic tissue, which is critical for preventing infection and promoting healing [15], followed by optional topical antimicrobials such as silver sulfadiazine or mafenide acetate to reduce bacterial load [16]. Occasionally, advanced strategies such as negative pressure wound therapy (NPWT) have been shown to improve wound bed preparation and granulation tissue formation in burn wounds [17], while split-thickness skin grafts provide coverage to facilitate healing in severe cases [18,19]. Hyperbaric oxygen therapy (HBOT) has demonstrated efficacy in certain conditions by promoting tissue regeneration through increasing oxygen delivery to hypoxic tissues; however, evidence specific to electrical burns remains limited [20,21].

Additionally, growth factor-based therapies, such as recombinant human epidermal growth factor and amniotic membrane grafts, are emerging as promising adjuncts in the treatment of chronic, non-healing wounds by supporting re-epithelialization and reducing inflammation [22,23]. Given the high incidence of neuropathic pain and functional impairment in electrical burn survivors, a comprehensive rehabilitation program that incorporates pain management (e.g., gabapentin or amitriptyline for neuropathic pain), physical therapy to restore mobility, and psychological support to address trauma is essential for optimizing recovery outcomes. Future research should focus on biomaterial-based dressings and regenerative medicine approaches to enhance wound healing in electrical injuries [24].

Materials and Methods

This case study involves a 50-year-old male with complex chronic electrical burn wounds affecting a significant portion of his body. The patient experienced a near-fatal electrocution and now lives with long-term complications resulting from electrical injuries. The patient is a male electrician who suffered a high-voltage electrical shock of approximately 15,000 volts when an alternating current hit the patient on the left ankle and exited through the perineum, knocking them off a train car. This occupational incident resulted in muscle necrosis in the right forearm, genitals, thigh, and leg, including damage to underlying tissues and peripheral nerve involvement. The patient was admitted to the ICU for resuscitation, monitoring, and initial stabilization. Fluid resuscitation was initiated using the Parkland formula, targeting adequate urine output between 0.5 to 1.0 mL/hg/h [25]. Surgical debridement was performed to remove necrotic tissue, and an escharotomy was done to alleviate compartment syndrome in the right arm. A broad-spectrum antibiotic was administered prophylactically, and wounds were dressed in silver-impregnated dressings to prevent infection and reduce inflammation.

After the initial acute management of the patient, negative pressure wound therapy (NPWT) was utilized on the thoracic wound to promote the formation of granulation tissue. The burn wounds on the arm and forearm were treated with bioengineered skin substitutes, including Grafix® Core and Prime, to enhance epithelialization. The patient was prescribed physical therapy to encourage early mobilization of the right arm, focusing on range-of-motion exercises to prevent contractures.

A small collection of split-thickness skin grafts (STSG) was harvested from the patient's left thigh and applied to the thoracic wound. Additionally, dermal matrices were used to cover other areas of significant tissue loss, particularly in the left lower extremity. After the patient developed a sensitivity to the live cell graft, alternative treatment modalities were implemented, which included: Endoform™ (ovine-derived collagen ECM), Prisma™ (collagen & oxidized regenerated cellulose matrix), Excellagen® (bovine collagen allogeneic matrix), Helicoll® (bovine or ovine acellular Type-1 collagen matrix), G4Derm™ (flowable biomimetic matrix), Acell Micromatrix® (micronized powder derived from porcine), and Flexagen (collagen Micromatrix Hexagen blend).

Following the intermediate healing processes, late healing and rehabilitation for scar management, as well as neurological and psychological recovery, took place. For scar management, silicone gel sheeting and compression garments were employed to reduce hypertrophic scarring, while laser therapy was introduced to improve scar texture and pigmentation. Occupational therapy focused on regaining motor function and dexterity in the right hand. Psychological support was provided through counseling sessions addressing post-traumatic stress and anxiety related to the injury. After 6 months post-injury, the patient exhibited significant functional and cosmetic recovery, with complete epithelization achieved in both the upper limb and thoracic wounds. The grafted areas demonstrated good integration with minimal scarring. The grip strength in the right hand reached 80% of the baseline, allowing the patient to return to light-duty work with accommodations. The patients' psychological support reported an anxiety reduction and improved quality of life. The patient received extensive pharmacological interventional strategies, many of which are ongoing due to the severity of the injury. Monitoring medication usage is crucial in the context of burn wound healing. While it may be challenging to identify specific evidence that demonstrates how and when medications can significantly impede healing, it is essential to understand their purposes and effects on pain, mobility, sensation, and the immune system – factors that can indirectly influence the healing process. Below is a list detailing the various medications prescribed to the patient, their functions, and any known effects on burn wound healing associated with these medications. Refer to Table One for a summary of the pharmaceuticals prescribed to the patient for treatment and associated symptoms.

Table 1. Below details a selection of the pharmacological drugs administered to the patient and how they may impact the healing processes of an extensive burn wound.

Medication	Function	Effect on Burn Wound Healing
Cymbalta (30 mg capsule)	Primarily used to treat depression, generalized anxiety disorder, fibromyalgia, and chronic musculoskeletal pain. Acts as a serotonin-norepinephrine reuptake inhibitor	Not usually linked to direct wound healing, but can affect cytokine production and responses to infection, inflammation, circulation, nutrition, and pain.
Ferrous sulfate (65 mg iron)	An iron supplement, commonly used to treat & prevent iron deficiency anemia. Iron plays a crucial role in the production of hemoglobin, which is essential for transporting oxygen and maintaining a strong immune system.	While iron is essential for healing, excessive amounts can increase oxidative stress. In the case of burn wounds, this may create an imbalance between free radical production and antioxidant defenses, potentially worsening the inflammatory phase if not properly regulated.
Gabapentin (800mg capsule)	Anticonvulsive medication treats neuropathic pain by binding to calcium channels in the brain and spinal cord, calming nerve activity, and reducing pain signals.	It can disrupt the normal inflammatory response, resulting in slower healing. Pain management may cause reduced mobility and sensation; it can interact with other medications by affecting their metabolism.
Lidocaine (5% topical patch)	A topical patch is commonly used for localized pain relief; it functions by delivering the anesthetic that works to block sodium channels and prevent the initiation and conduction of nerve impulses, resulting in a numbing effect and temporary pain relief.	While helpful for providing pain relief for burn wounds, lidocaine patches should be used with caution. They may mask pain that serves as a protective mechanism, and improper use may cause skin irritation, infection, and blood flow concerns.
Oxycodone (10 mg tablet & 15 mg tablet)	Oxycodone is a semisynthetic opioid commonly used to manage moderate-to-severe pain. Works as a mu-opioid receptor agonist, meaning it activates receptors in the CNS to reduce the perception of pain.	There are several risks associated with its use that may negatively impact burn wound healing, including immune suppression, delayed healing, gastrointestinal issues, and psychological effects.
Sucralfate (1g tablet)	It is a cytoprotective drug widely used in clinical practice to prevent or treat several gastrointestinal diseases such as gastroesophageal reflux, gastritis, peptic ulcer, stress ulcer, and dyspepsia. Sucralfate is a sulfated polysaccharide that, when dissolved in acid, forms a viscous, sticky substance that adheres to the ulcer or wounded area, promoting healing and protecting it from further damage.	Monitoring is essential when sucralfate is used in burn patients to ensure proper drug interactions, gastrointestinal comfort, and nutritional support.

Testosterone Cypionate (200mg/mL intramuscular oil)	It is a synthetic form of the hormone testosterone, which is commonly used to treat conditions caused by low testosterone levels in men. This form of testosterone binds to androgen receptors throughout the body, leading to various physiological effects.	While testosterone cypionate is primarily used for hormonal replacement and muscle growth, its potential effects on burn wound healing are more complex. Androgens influence various bodily functions that are involved in tissue repair, including protein synthesis, collagen production, immune response, and inflammation.
Tizanidine (4mg tablet)	It is a muscle relaxant that works by inhibiting spinal cord activity, primarily by acting on alpha-2 adrenergic receptors in the central nervous system. It is commonly used to treat conditions involving muscle spasticity caused by multiple sclerosis, an acquired brain injury, or a spinal cord injury.	The potential risks associated with its use in burn patients include: • Hypotension and dizziness that can impair circulation • Sedation and drowsiness • Gastrointestinal issues such as nausea and constipation • Immune suppression • Respiratory depression, which can exacerbate hypoxia
Tramadol (50mg tablet)	An opioid-like analgesic that is used in the management of moderate to moderately severe pain; it works by affecting the CNS to alleviate pain. It binds to opioid receptors in the brain and spinal cord to block the transmission of pain signals.	
Xyosted (75mg/0.5mL subcutaneous auto-injector)	A testosterone replacement therapy, used in adult males to treat low testosterone levels. It comes as an autoinjector for home use to self-inject under the skin in the stomach.	Some negative effects of excess testosterone could suppress immune function, increasing the risk of wound infection, fluid retention, and edema, which could exacerbate swelling and impair proper wound healing, and increased red blood cell production, leading to a higher risk of clotting or circulatory issues.

After many years of various treatment therapies, the lower leg containing the ulcerations failed to close with no improvement with the above modalities (Endoform™, Prisma™, Excellagen®, Helicoll®, G4Derm™, Acell Micromatrix® and Flexagen). This ultimately led to stalled wounds that created a full-thickness, neuropathic, traumatic, ulcerated, chronic wound, including one at the location of the left medial malleolus. A biopsy was not taken for fear of enlarging or creating a new wound space. Ulcers of this nature have a high rate of failure to heal, with high rates of infection, which can lead to amputation, other morbidities, and even mortality. Therefore, the decision for advanced treatment in the form of a commercially available dehydrated amniotic membrane (dHAM) biological skin substitute product known as Axolotl DualGraft™ (Axolotl Biologix, USA, Scottsdale AZ.) was utilized. This was selected as an aid to reduce the size of the lesion, prevent worsening conditions in the patient's mobility, prevent developing infection, and overall loss of limb and life. See Figure 1 for a visual representation of the dehydrated amniotic membrane appearance, ex-vivo.

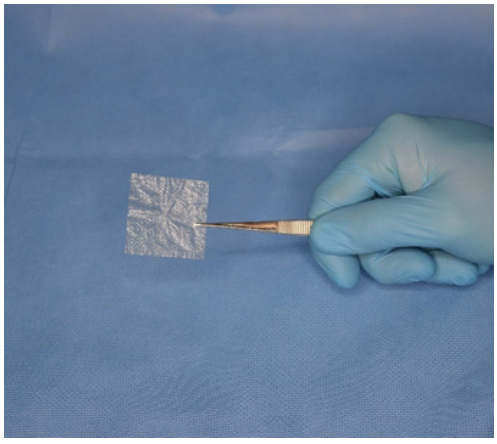

Figure 1

Figure 1. is an example of an amniotic membrane product applied during the presented electrocution wound care case study.

The conditions, etiologies, and options for care, treatment plan, and prognosis were all reviewed with the patient, with both conservative and surgical options for care discussed. Together, the physician and patient agreed that the chosen tissue-based product, due to its unique properties, including wound protection, the potential for decreased scarring, pain reduction properties, and epithelialization initiation capabilities, would be a proper clinical strategy [26]. The dHAM product is regulated by the Food and Drug Administration (FDA) as a human cell, tissue, and cellular and tissue-based product or HCT/P under 21 CFR 1271. It is a resorbable amniotic membrane used as a structural wound barrier for varying acute or chronic wound conditions. Figure 2 below details the initial visit wound in 2024 with the first allograft application.

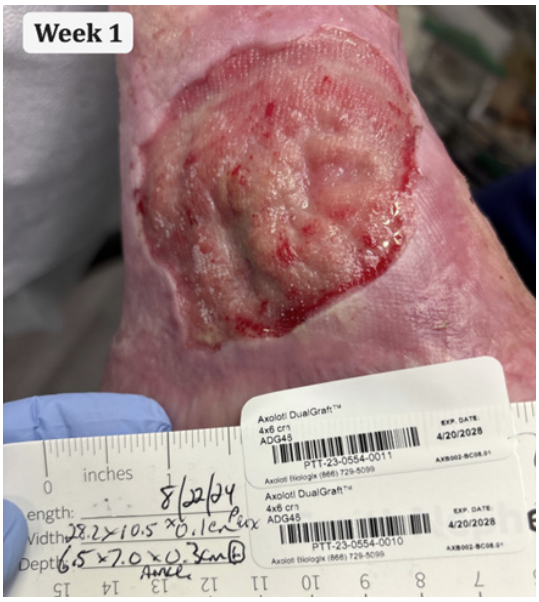

Figure 2

Figure 2. First Application (8/22/24):
The skin ulcer has irregular, sharply defined borders and is situated within background skin that shows extensive scarring. The surrounding skin lacks normal epithelial tissue and accessory structures, giving it a shiny and rigid appearance. The ulcer bed is moist and does not exhibit any purulent discharge.

Before the dHAM was initially applied to the left LE ulceration, the patient received several conservative treatments, including debridement, offloading, medications, and collagen topicals. Unfortunately, these treatments did not yield a tissue response, prompting the initiation of advanced therapies.

Attention was directed to the left medial ankle ulceration with sharp and blunt dissection with a scalpel to remove the hyperkeratotic rim and any debris present from the wound bed. After debridement, the wound was flushed with normal sterile saline. Hemostasis was obtained with pressure, and the wound was ready to receive the dHAM graft. The amniotic allograft was prepared aseptically, measured, and fitted to the wound bed using sterile saline; no suturing was necessary, and any excess graft was folded into the wound bed. The graft was covered with a non-adherent dressing and fixed in place with Mepitel® and Steri-Strips™, a dry sterile offloading dressing. Coban was applied to complete the dressing and to achieve a degree of compression to address edema. No local anesthesia was used, and the patient reported tolerating the procedure well and would report pain on a numerical pain rating scale (standard 0-10 scale), see figure 3 below in results section.

The patient proceeded with weekly clinical check-ins, with reapplication of the amniotic membrane at physician-selected intervals. Before application, the wound bed would be measured using standard wound sizing measurements. The images (figures 4-9) are gross photos of the patient's medial ankle ulceration after treatment application, illustrating the wound healing response in the ulcer with the utilization of dHAM. The surrounding tissue of the medial ankle ulcer is not healthy and resembles the tissue architecture of scar tissue that has healed by secondary intention or fibrosis. This is common in deep partial-thickness burns and full-thickness burns, where the regenerative layers of the skin are destroyed [8,13]. The healing by substitution with an amniotic membrane graft means the body is still forming scar tissue, but it is resulting in more functional, less fibrotic tissue than would occur without the amniotic allograft present.

Results

In this clinical case study, the patient's neuropathic ankle ulcer was monitored using a combination of a 0-10 numeric pain intensity scale, serial wound measurements, and photographic documentation to track healing progress with dHAM treatments. Initial assessments recorded the wounds surface area (SA)to be 45.5cm2 and a 13.65 cm3 volume (V), with a reported pain level of 5.

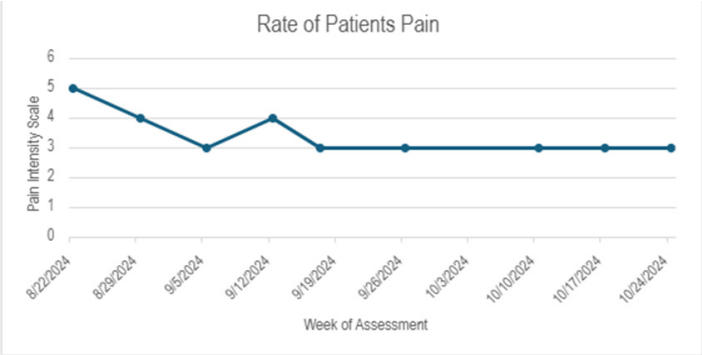

Figure 3

Figure 3. Graphical illustration of the patient's pain response correlating to the initial application of the allografts. A notable decrease in pain occurred during the first 5 weeks of the allograft treatment regimen.

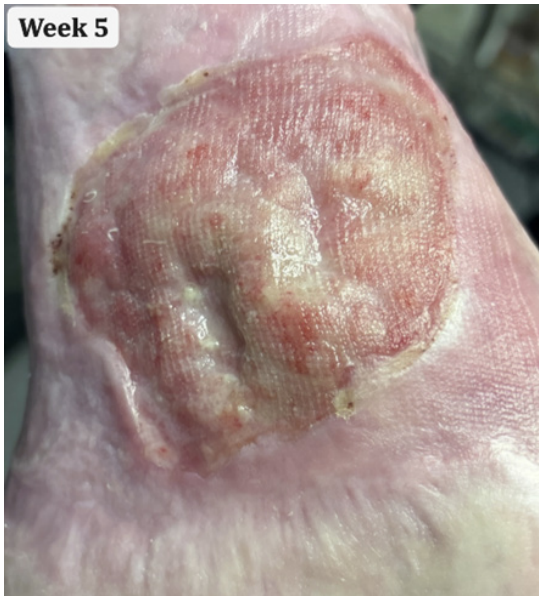

Figure 4

Figure 4. Week Five (9/26/2024):
The skin ulcer is similar in appearance, but small, with healing by the production of undifferentiated connective tissue (cicatrix) around the wound edges. The ulcer bed remains moist, without purulent exudate, but also lacks islands of epithelialization due to the limited regenerative ability of the background skin. Wound measurement post-debridement: 5.4x6.7x0.3 cm.

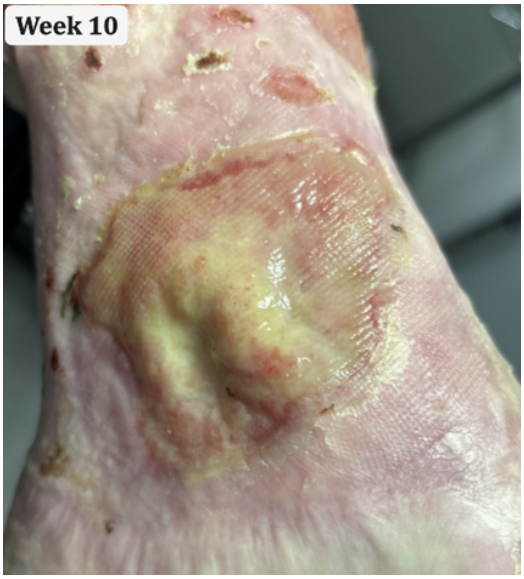

Figure 5

Figure 5. Week Ten (10/24/24):
The skin ulcer is significantly smaller, showing further healing with dHAM controlling inflammation. The borders of the healed ulcer closely resemble the surrounding skin, featuring reduced pigmentation and altered texture. Wound size post-debridement: 5.0x6.3x0.3 cm.

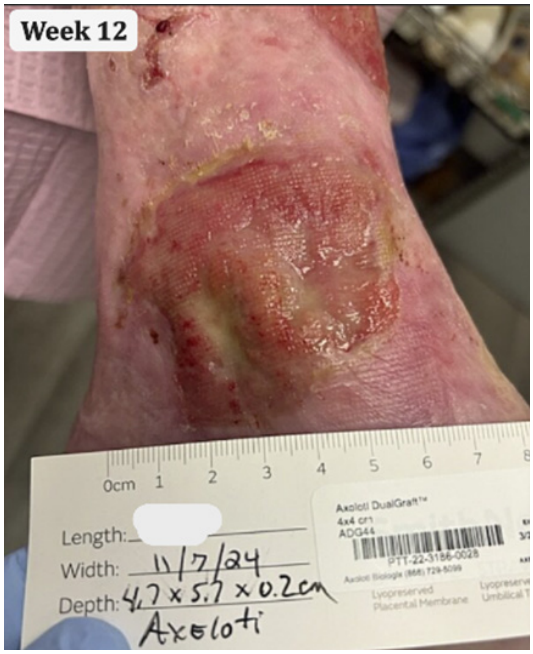

Figure 6

Figure 6. Week Twelve (11/7/24):
The skin continued to maintain a similar size to the previous week (ten) and demonstrated ongoing tissue response. Again, the borders of the healed ulcer closely resemble the surrounding integument, featuring reduced pigmentation and altered texture. Biofilm is present, while no odor is present. The wound did not contain any purulent exudate. Wound size post-debridement: 4.7x5.7x0.2cm.

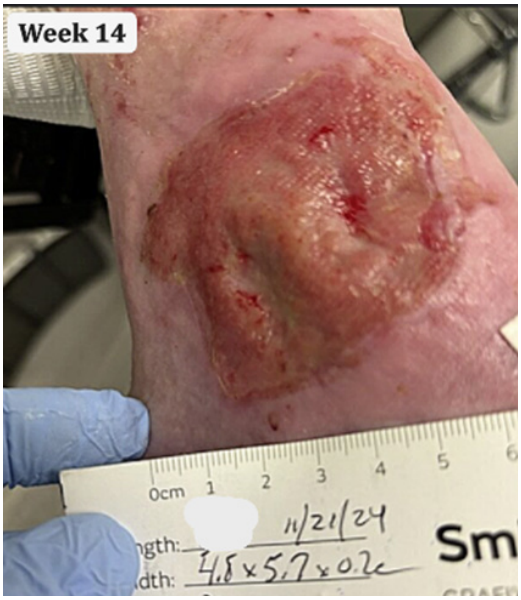

Figure 7

Figure 7. Week Fourteen (11/21/24):
At week fourteen, the ulcer borders continue to gain similar pigmentation as surrounding previously healed tissue, as noted by the black arrow, and wound size remained the same. Wound odor, biofilm presence, and moisture appeared to remain as in previous assessments. Wound size post-debridement: 4.8x5.7x0.2cm.

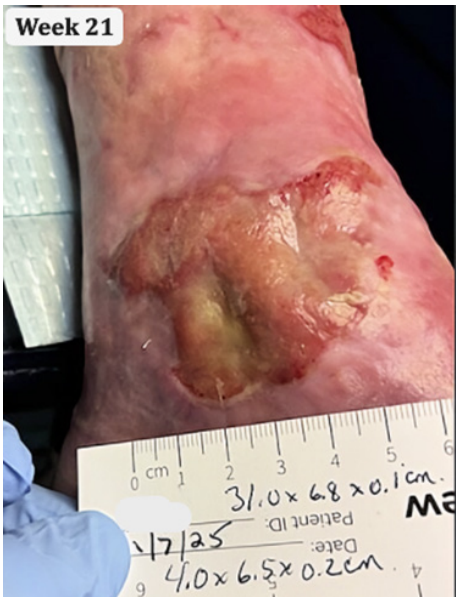

Figure 8

Figure 8. Week twenty-one (1/7/25):
After intermittent appointments due to the 2024 holiday season, the wound measurements did change. However, the wound bed moisture and peri-wound edges still appear to be responding. Wound size post-debridement: 4.0x6.5x0.2cm.

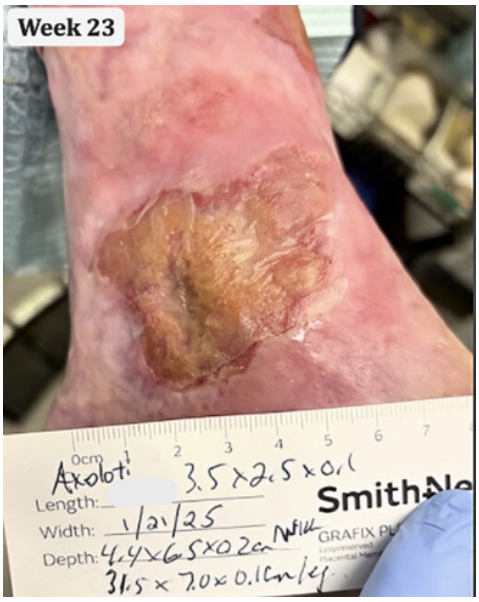

Figure 9

Figure 9. Week twenty-three (1/21/25):
Following allograft placement, the wound demonstrated tissue regeneration beginning at the peri-wound edges. No odor, biofilm, or excessive exudate was observed. Wound size post-debridement: 4.4 x 6.5 x 0.2 cm.

A graph plotting the measured surface area (SA) and volume (V) of the ankle ulcer each week of treatment is captured below. See Figure 10: Graph A Wound Surface Area & Graph B Wound Volume.

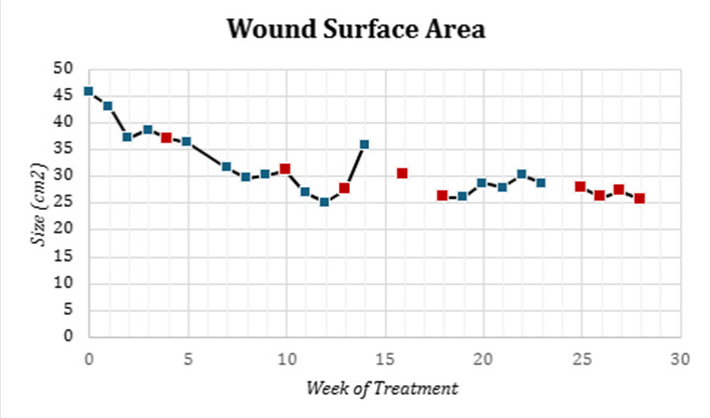

Figure 10. Graph A

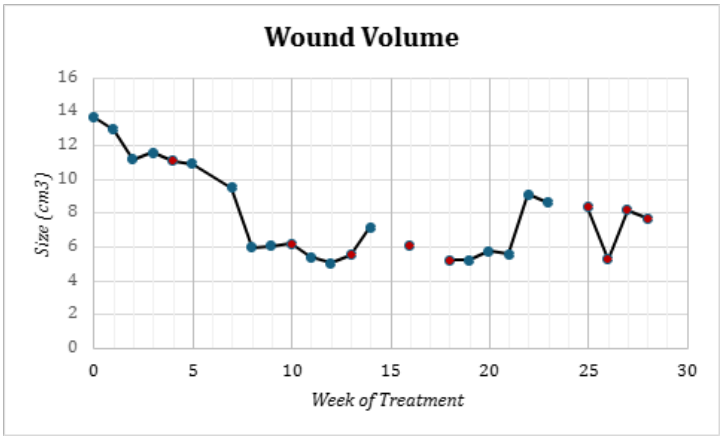

Figure 10. Graph B

Figure 10. Graph A & Graph B:
The graphs illustrate the size of the wound by plotting the measurements SA or Volume over the weeks of treatment applications. Graph A represents the calculated surface area of the wound (length x width) in cm2, while Graph B indicates the calculated volume (cm3) (surface area x width x depth) of the wound following 28 weeks of treatment. The red points on each graph indicate the weeks when membrane application did not occur, and the skipped weeks are due to the patient's absence.

The patient was unable to make every weekly appointment; thus, weeks 15, 17, and 24 have no recorded data. During weeks 4, 10, 13, 16, 18, and 25 through 28, no dHAM membrane was applied to the wound bed, however, measurements were still taken and dressing reapplied. From week 13 to week 14 and week 21 to week 22, there is an increase in SA and volume due to the location of the wound. The decrease in pain masked the patient's unintentional non-adherence to wound protection protocols, and the patient was inadvertently placing excessive mechanical stress on the wound bed. This increased ambulation causes the wound margins to stretch towards the dorsum of the foot. The wound demonstrated tissue response and consistently decreased in size over 28 weeks, showing a 44% reduction in wound surface area and volume. Concurrently, the patient reported progressive pain relief, with scores declining to a steady 3, correlating with visible improvement in tissue quality and granulation. Photographic evidence further supports the observed healing trajectory, capturing the reduction in wound size and the enhancement in wound bed appearance. Together, these tools provided a comprehensive and objective view of the patients' healing process, demonstrating the effectiveness of the dHAM treatment.

The wound size has reduced, and the previously stalled healing process has begun to respond, marking a significant and positive development overall. When a wound that has remained in the inflammatory phase finally begins to heal, it signifies a return to the body's natural healing process. Resumption of healing in chronic burn wounds reduces the risk of infection, amputation, and further tissue breakdown, and supports improved functional outcomes and quality of life.

Discussion

In this case study, a 50-year-old male sustained a high-voltage electrical injury in 2015, creating a lower extremity stalled wound that had remained open for approximately nine years. The complexities of an electrical burn can create stalled tissue responses. As such, the complex nature of electrical injuries necessitates a multidisciplinary approach to begin to stimulate a localized tissue response to aid in the wound healing progression. Early surgical intervention, combined with advanced wound care technologies and rehabilitative therapies, is pivotal in these types of wounds and a patient's recovery. Treatment modalities that include allograft skin substitutes may create a tissue response. One such example of these allograft tissue products, dHAM, was applied over a 28-week treatment regime, creating a visible tissue response that had not been present for years. The mechanism of action to stimulate tissue cannot be derived from a single cellular pathway, growth factor, or biological agent; rather, biological skin substitutes create a multifaceted approach to simulate tissue. These allograft amniotic membrane products have been demonstrated to contain numerous proteins, growth factors, and peptides that have been scientifically demonstrated to initiate cellular pathways [27].

The biological structure of allograft amniotic membrane consists of a dense basement membrane and a stromal matrix, which provides a supportive and structurally protective scaffold that covers exposed tissues and modulates the wound microenvironment. Studies have demonstrated that amniotic membrane application to electrical burns reduces inflammation, minimizes fluid loss, and accelerates epithelialization, offering a significant advantage over traditional dressings and standard of care [28,29]. A recent review by Yang et al supports the use of amniotic membrane as an effective adjunct in burn wound management. A review of eleven randomized controlled trials involving a total of 816 participants found that AM treatment was more effective than conventional approaches, silver sulfadiazine, and polyurethane membranes in promoting burn wound healing [30]. These findings, alongside this unique clinical case, suggest that AM may represent a promising option in the treatment of electrical burns, particularly in cases where minimizing infection risk, enhancing epithelization, and reducing inflammation are clinical priorities.

A common misconception is that all amniotic graft membranes are the same. However, processing modalities and donor-specific requirements all affect the final product's characteristics, and it is important to characterize these advanced therapy medicinal biologics, such as amniotic membrane. The tissue response potential of amniotic membrane in electrical injuries is largely attributed to its numerous bioactive growth factors. This specific dHAM product utilized in this case study has been biologically characterized to contain over 7,500 proteins, ~83,000 peptides, and ~97,000 peptide ion variants [26]. Certain growth factors found within the amniotic membrane can stimulate tissues and subsequently impact the phases of a wound healing response. For example, epidermal growth factor (EGF) and transforming growth factor-beta (TGF-β) found in amniotic membrane promote keratinocyte proliferation and fibroblast activity, critical for restoring the epidermal and dermal layers [31]. Vascular endothelial growth factor (VEGF) facilitates neovascularization, an essential process for managing electrical burns where significant vascular damage affects blood flow. The basic fibroblast growth factor (bFGF) and platelet-derived growth factor (PDGF), further stimulate fibroblast migration, granulation tissue formation, and matrix remodeling [31]. Together, these growth factors create an environment conducive to controlled, functional tissue response and ultimately may initiate repair.

Beyond growth factors, the extracellular matrix components of amniotic membrane, such as collagen types I and III and fibronectin, provide mechanical stability and biochemical cues that guide cellular infiltration and tissue organization [32]. These matrix proteins, together with the membrane's anti-inflammatory cytokines (e.g., interleukin-10), can mitigate the excessive inflammatory and fibrotic responses often seen in severe electrical wounds [29]. Consequently, the amniotic membrane may not only facilitate a tissue response but can also aid wound closure, making it an invaluable tool in the management of electrical burn injuries.

Advanced wound treatment has become a vital component of burn care, as the focus shifts towards optimizing recovery and preventing unwanted complications. Severe burn injuries result in a prolonged immune response, which can exacerbate the injury by causing multiple systemic effects and increasing the risk of invasive infections [8]. Generally, the complex healing response is targeted towards dermal and epidermal regeneration, to restore closure of the skin barrier as well as pliability and functionality of the skin [13]. The healing process for this patient does not adhere to the typical regenerative path seen in most cases due to the severity of the injury. It is essential to consider the patient's extensive history of care and the quality of that care. The dHAM product offered coverage, regained mobility, and facilitated the tissue to respond, causing a healing response that had not been present for numerous years in this wound's specific location.

Throughout a 28-week treatment, the use of a regenerative therapy, dHAM, not only restarted the healing process and showed signs of the wound responding to treatment after numerous unsuccessful options (by a decline in wound surface area and volume), but it also reduced the patient's pain. Treatment for chronic illnesses or wounds can prolong life; however, it often comes with side effects such as fatigue, nausea, pain, and emotional distress, which can reduce the quality of life that it aims to enhance. For many patients facing serious illnesses or chronic wounds, the goal of treatment shifts from seeking a cure to maximizing their time in a way that is fulfilling and dignified.

Dehydrated human amniotic membrane was considered for this patient to stimulate a tissue response due to its minimally processed amniotic tissue and its ability as a successful non-immunogenic barrier with potential antibacterial properties that provide necessary cytokines and growth factors within a matrix that facilitate migration and proliferation of cells. As of now, the patient's wound has reached an optimal state, making it a notable candidate

for more advanced treatment options, such as autografting or cellular therapy. These future interventions could further enhance the recovery process and improve the patient's overall quality of life.

Conclusion

This electrical burn case highlights the importance of tailored interventions in managing electrical-associated wounds. Advances in wound care and interdisciplinary collaboration can optimize outcomes, enabling patients to regain functionality and quality of life.

This clinical case study presents several important limitations that should be acknowledged. The absence of standardized wound assessment tools, such as the Pressure Ulcer Scale for Healing (PUSH) and the Bates-Jensen Wound Assessment tool, limited the ability to track and quantify wound healing over time objectively. Similarly, the reason for not utilizing non-invasive tools (such as thermography or Doppler) was based on physician discretion during a prolonged and evolving course of treatment, where clinical judgment guided care over time. Additionally, the chronicity and severity of the patient's condition contributed to a distorted treatment timeline, complicating efforts to specifically track the patients' lower left leg ulceration interventions. Microbial monitoring was not consistently performed, further limiting insight into infection dynamics during the healing process. Moreover, this case was not initiated as a formal clinical investigation but emerged as a unique observational opportunity following the application of dHAM.

Further research is needed to refine treatment protocols and explore emerging technologies in this field. The research into dHAM therapy applications is in an expanding phase, working to establish useful applications, since clinical outcomes may vary significantly, as not all dHAM products are processed or preserved in the same manner. Additionally, data demonstrates if these tailored solutions can offer cost savings to the patient and the insurance system throughout all facets of the treatment regime should be gathered.

Despite a lengthy treatment history, multiple co-morbidities, and concurrent medications known to impair wound healing, this patient demonstrated a favorable response to dHAM following the failure of alternative treatment modalities. These findings highlight the potential value of dHAM in complex wound care and supports the need for further rigorous studies using standardized methodologies and broader patient populations.

Author Contributions

» **Conceptualization:** Tabor AJ, Ingraldi A;
» **Methodology:** Davis D;
» **Formal analysis:** Tabor AJ, Ingraldi A, Lee D, Davis D;
» **Resources:** Tabor AJ;
» **Data curation:** Tabor AJ, Ingraldi A;
» **Writing—original draft preparation:** Tabor AJ, Ingraldi A;
» **Writing—review and editing:** Tabor AJ, Ingraldi A, Davis D, Lee D;
» **Visualization:** Tabor AJ, Ingraldi A;
» **Supervision:** Tabor AJ;
» **Project administration:** Tabor AJ.

All authors have read and approved the final version of the manuscript.

Funding

This research received no external funding.

Informed Consent Statement

Individual patient informed consent was obtained along with medical release by the patient; all personal information has been deidentified to respect the patient and their anonymity.

Data Availability Statement

The datasets presented in this article are not readily available because of corporate policy and an ongoing study with dehydrated human amniotic membrane. Requests to access the datasets should be directed to Dr. Aaron J. Tabor at atabor@axobio.com.

Acknowledgments

The authorship group would like to acknowledge the patient and all patients who allow their clinical data to be an educational source for the translational and clinical scientific field.

Conflicts of Interest

The authors have the following relevant disclosures: Dr. Aaron J. Tabor and Alison Ingraldi, PhD student, are employed by Axolotl Biologix, LLC (Scottsdale, AZ).

References

1. Cooper MA. Electrical and lightning injuries. Emerg Med Clin North Am. 1984; 2: 489–501.
2. Zemaitis MR, Cindass R, Lopez RA, Huecker MR. Electrical injuries. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448087/
3. Spies C, Trohman RG. Narrative review: electrocution and life-threatening electrical injuries. Ann Intern Med. 2006; 145: 531–537.
4. Kumar M, Mahmood S, Mandal UK. An updated account on formulations and strategies for the treatment of burn infection – a review. Curr Pharm Des. 2022; 28: 1480–1492.
5. Fish RM, Geddes LA. Conduction of electrical current to and through the human body: a review. Eplasty. 2009; 9: e44.
6. Mason SA, Nathens AB, Byrne JP, Ellis J, Fowler RA, et al. Association between burn injury and mental illness among burn survivors: a population-based, self-matched, longitudinal cohort study. J Am Coll Surg. 2017; 225: 516–524.
7. Rehan M, Tariq R, Iqbal T, Sarwar MA, Tul Ain Q, et al. Impact of burns on anxiety, depression and self-esteem among patients with burn injuries: a cross-sectional study. Ann Burns Fire Disasters. 2024; 37: 134–139.
8. Burgess M, Valdera F, Varon D, Kankuri E, Nuutila K, et al. The immune and regenerative response to burn injury. Cells. 2022; 11: 3073.

9. Lu J, Yang M, Zhan M, Xu X, Yue J, et al. Antibiotics for treating infected burn wounds. Cochrane Database Syst Rev. 2017; 2017: CD012084.
10. Rojas Y, Finnerty CC, Radhakrishnan RS, Herndon DN. Burns: an update on current pharmacotherapy. Expert Opin Pharmacother. 2012; 13: 2485–2494.
11. Wicke C, Halliday B, Allen D, Roche NS, Scheuenstuhl H, et al. Effects of steroids and retinoids on wound healing. Arch Surg. 2000; 135: 1265–1270.
12. Shi H, Cheer K, Simanainen U, Lesmana B, Ma D, et al. The contradictory role of androgens in cutaneous and major burn wound healing. Burns Trauma. 2021; 9: 046.
13. Jeschke MG, van Baar ME, Choudhry MA, Chung KK, Gibran NS, et al. Burn injury. Nat Rev Dis Primers. 2020; 6: 11.
14. Orgill DP, Piccolo N. Escharotomy and decompressive therapies in burns. J Burn Care Res. 2009; 30: 759–768.
15. Taran A. Treatment approaches of electrical injuries. Mold Med J. 2021; 64: 38–41.
16. Storm-Versloot MN, Vos CG, Ubbink DT, Vermeulen H. Topical silver for preventing wound infection. Cochrane Database Syst Rev. 2010; 3: CD006478.
17. Kantak NA, Mistry R, Varon DE, Halvorson EG. Negative pressure wound therapy for burns. Clin Plast Surg. 2017; 44: 671–677.
18. Vig K, Chaudhari A, Tripathi S, Dixit S, Sahu R, et al. Advances in skin regeneration using tissue engineering. Int J Mol Sci. 2017; 18: 789.
19. Singh M, Nuutila K, Kruse C, Robson MC, Caterson E, et al. Challenging the conventional therapy: emerging skin graft techniques for wound healing. Plast Reconstr Surg. 2015; 136: 524e–530e.
20. Villanueva E, Bennett MH, Wasiak J, Lehm JP. Hyperbaric oxygen therapy for thermal burns. Cochrane Database Syst Rev. 2004; 3: CD004727.
21. Weitgasser L, Ihra G, Schäfer B, Markstaller K, Radtke C. Update on hyperbaric oxygen therapy in burn treatment. Wien Klin Wochenschr. 2021; 133: 137–143.
22. Brown GL, Nanney LB, Griffen J, Cramer AB, Yancey JM, et al. Enhancement of wound healing by topical treatment with epidermal growth factor. N Engl J Med. 1989; 321: 76–79.
23. Kesting MR, Wolff KD, Hohlweg-Majert B, Steinstraesser L. The role of allogenic amniotic membrane in burn treatment. J Burn Care Res. 2008; 29: 907–916.
24. Chua AW, Khoo YC, Tan BK, Tan KC, Foo CL, et al. Skin tissue engineering advances in severe burns: review and therapeutic applications. Burns Trauma. 2016; 4: 3.
25. Mehta M, Tudor GJ. Parkland formula. StatPearls. 2025; Jan.
26. Ingraldi AL, Allen T, Tinghitella JN, Merritt WC, Becker T, et al. Characterization of amnion-derived membrane for clinical wound applications. Bioengineering. 2024; 11: 953.
27. Munoz-Torres JR, Martínez-González SB, Lozano-Luján AD, Martínez-Vázquez MC, Velasco-Elizondo P, et al. Biological properties and surgical applications of the human amniotic membrane. Front Bioeng Biotechnol. 2023; 10: 1067480.
28. Parungao RJ, Cheer K, Simanainen U, Ma D, Hew JJ, et al. Electrical burns: challenges and strategies in wound healing. Burns Trauma. 2020; 8: tkaa012.
29. Niknejad H, Peirovi H, Jorjani M, Ahmadiani A, Ghanavi J, et al. Properties of the amniotic membrane for potential use in tissue engineering. Eur Cell Mater. 2008; 15: 88–99.
30. Yang C, Xiong AB, He XC, Ding XB, Tian XL, et al. Efficacy and feasibility of amniotic membrane for the treatment of burn wounds: a meta-analysis. J Trauma Acute Care Surg. 2021; 90: 744–755.
31. Koob TJ, Rennert R, Zabek N, Massee M, Lim JJ, et al. Biological properties of dehydrated human amnion/chorion composite graft: implications for chronic wound healing. Int Wound J. 2014; 11: 477–483.
32. Diller RB, Tabor AJ. The role of the extracellular matrix in wound healing: a review. Biomimetics. 2022; 7: 87.