

Advancing Wound Management: A Comprehensive Review of Marketed Formulations Linking Herbal and Conventional Therapeutics

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ABSTRACT

Wound healing is a highly regulated biological process that progresses through interconnected phases, namely hemostasis, inflammation, proliferation, and tissue remodelling. Managing wounds effectively continues to be a clinical concern, particularly in cases complicated by infection, metabolic disorders, or impaired vascular supply. Recently, increasing attention has been directed toward both herbal and non-herbal marketed formulations as therapeutic options to improve healing outcomes. Herbal agents such as *Curcuma longa*, *Aloe vera*, *Avena sativa*, and *Embllica officinalis* are known for their diverse pharmacological properties, including anti-inflammatory, antioxidant, antimicrobial, and collagen-promoting effects. Their natural origin and favourable safety profile make them attractive alternatives or adjuncts in wound care. Conversely, conventional non-herbal formulations, including hydrogel-based dressings, silver-containing preparations, and iodine-based antiseptics, remain widely utilized in clinical settings. These products provide benefits such as infection control, maintenance of a moist wound environment, and consistent therapeutic performance. The present review focuses on comparing herbal and non-herbal marketed wound healing formulations in terms of their mechanisms of action, efficacy, safety, and clinical applicability. Evidence from selected experimental studies and clinical observations is also discussed to evaluate treatment outcomes, including wound closure rate, pain reduction, and patient compliance. In summary, both categories of formulations demonstrate unique advantages, and their rational combination may offer improved therapeutic benefits. Further well-structured clinical investigations are required to validate their effectiveness and support evidence-based wound management practices.

Keywords: Wound healing, Herbal formulations, Marketed products, Hydrogel dressings, Curcumin, Clinical evidence

1) INTRODUCTION:

The primary function of the skin is to act as a barrier to protect against environmental insults. Seriously, morbidity and mortality can result from the skin's primary immune system function being compromised when its structural integrity is compromised. A wound occurs when physical, electrical, chemical, or microbiological assaults to the living tissue cause the cellular, anatomical, and functional continuity to be disrupted [1]. A wound is characterized by a disruption in the structure and function of the underlying tissues and consists of a break in the continuity of the skin's epithelium. In order to minimize fluid loss, prevent infection, and reestablish homeostatic mechanisms, skin integrity must be promptly restored following injury. This is accomplished through a complex process known as wound healing, in which a number

of interconnected and parallel pathways are triggered and synchronized to promote wound healing [2]. The physiological process of wound healing is crucial for preserving the skin's integrity following trauma, whether intentional or the result of an accident [3]. Homeostasis, inflammation, cellular migration and proliferation, protein synthesis, and wound contraction are proposed as the five stages of wound healing [4]. The biophysiological processes involved in these phases must take place in the right order, at the right time, and continue for a predetermined amount of time at the best possible intensity. Multiple factors can influence wound healing, interfering with one or more stages of the process and leading to inadequate or compromised tissue repair. The key elements that influence the healing of cutaneous wounds include age, sex, stress, obesity, tobacco, diet, drugs, alcohol, and infection etc [5]. In general, wounds that show impaired healing such as chronic wounds and delayed wounds have not advanced through the typical healing phases. This delayed, incomplete, or disorganized healing process often results in such wounds entering a state of pathological inflammation. The majority of chronic wound infections are ulcers linked to pressure, ischemia, diabetes mellitus, and venous stasis diseases [6]. Wounds can be divided into two main categories: acute and chronic wounds. An Acute wound can be full thickness, compromising the subcutaneous layer, or superficial, affecting both the epidermis and superficial dermis. Surgical wounds are an example of acute wounds. Acute wounds include surgical incisions, thermal wounds, abrasions, and lacerations; infection is the main complication of each of these injuries. Growth factors and cytokines are released near the wound healing. Neutrophils, macrophages, and lymphocytes migrate to the wound during the inflammatory phase linked to wound healing, resulting in the inflammatory symptoms that last for roughly two weeks. Inflammation that lasts for months or years is categorized as chronic, and it can be linked to a variety of pathological changes, such as infection and elevated protease activity. Following the inflammatory phase, the proliferative phase is marked by the formation of new tissue, granulation, and epithelial tissue, as well as restoration of the vascular network [7].

2) PHYSIOLOGY OF WOUND HEALING:

2.1) Stages of Healing (Figure1)

(i) Hemostasis: Coagulation and Hemostasis occur in the wound as soon as an injury occurs. This mechanism is primarily designed to avoid loss of blood. It is a means of preserving the vascular system, ensuring that vital organ function is unaffected in the event of damage. In addition to homeostatic processes, extrinsic and intrinsic pathways trigger the coagulation cascade, which causes platelet aggregation and formation of a clot to reduce blood loss [8]. During the initial stage of the injury, the inert zymogens become fully functional, catalytically active serine proteases which in turn cause platelets to activate and a fibrin clot to form. Along with hemostasis, platelet activation triggers the release of growth factors like platelet-derived growth factor and immune mediators, which trigger the immune system's activation and the shift to the inflammatory phase of wound healing. The hemostasis phase start when tissue damage permits blood to seep into the exposed wound site, starting the extrinsic clotting cascade and releasing mediators like serotonin that result in localized vasoconstriction [ps6]. Platelets and other components interact with exposed collagen and other extracellular elements as blood leaks into the injury site. When platelets come into contact with one another, clotting

factors are released, and a blood clot made of fibrin, vitronectin, thrombospondin, and fibronectin forms [8].

(ii) **Inflammation:** Within 24 hours of the injury occurring, the inflammatory phase of the healing process begins and typically lasts for 48 hours. After the completion of hemostasis and coagulation, the wound healing process progresses to the inflammatory phase. This stage includes an early response marked by neutrophil migration and a subsequent phase where monocytes are recruited and undergo transformation. [9]. Chemokines are essential modulators of the healing process of wounds. During this stage, the injured area may experience pain, redness, body heat and swelling, and other typical inflammatory signs. When collagen is exposed during wound formation, the intrinsic and extrinsic clotting cascades are activated, starting hemostasis. Furthermore, thromboxane A₂ and prostaglandin 2- α are released into the wound bed as a result of tissue damage, triggering a strong vasoconstrictor response. Additionally, the formation of the blood clot that reinforce the hemostatic plug is made possible by the extravasation of blood constituents. This first reaction supplies an initial extracellular matrix for cell migration and aids in limiting bleeding. One of the initial responder cells that is essential to the development of the hemostatic plug is platelets. Numerous chemokines, including platelet-derived growth factor (PDGF), histamine, von Willebrand factor, fibronectin, fibrinogen, epidermal growth factor, and serotonin, are secreted by the plug. These chemicals draw in and stimulate fibroblasts, neutrophil, and macrophages, which help in the stabilization of the wound healing [10]. Chemokines cause leukocytes, such as neutrophils and macrophage to migrate into damaged tissue as part of the inflammatory process [11].

Role of Neutrophils in inflammation: Neutrophils make up 50-70% of all leukocytes in healthy adult humans [12]. Generally, Neutrophils remove foreign debris from the wound and kill bacteria. The waste is then phagocytosed by macrophages or extruded with the eschar [13]. Their recruitment is started by the production of N-formyl peptides by bacteria and injured cells, as well as growth factors and chemokines by active platelets in the blood clot. During the early stage of inflammatory phase, neutrophils concentration in the wound rises, and four days later, it falls. Through the action of adhesion receptors like integrins and selectins, neutrophils bind to endothelial cells and undergo a sequence of events known as the leukocyte cascade, including rolling, firm attachment, crawling, and transmigration toward the site of inflammation. To continue recruiting neutrophils, neutrophils in the wound generate more neutrophil- chemoattractant mediators. Neutrophils are the most prevalent immune cells in the wounds, with a concentration of 5×10^6 within the first 24 hours and continuing to rise on day 2. Neutrophils in infected tissue bind to opsonized pathogens' Fc-receptors to aids in phagocytosis, and then release ROS and antibacterial protein from neutrophil granule into the phagosome to eradicate the pathogen [12].

Role of Macrophage: Macrophage are essential for wound healing, and it is evident how they contribute to angiogenesis, fibroplasia, cell division, and the removal of inflammation. Macrophages are phagocytic monocyte-derived cells that scavenge and eliminate necrotic tissue, dead cells, and the removal of inflammation. To aid in tissue repair, these homeostatic processes are boosted by a range of stimuli following injury. When there is cutaneous injury, circulating, monocytes leave blood vessels and move into the wound site, while local, skin-

resident macrophages are activated by danger signals and other injury by product molecules such as H₂O₂ [14].



Figure1: Stages of wound healing

(iii) Proliferative: The phase of proliferation starts as the inflammation subsides. This includes producing granulation tissue, re-epithelializing the wound surface, and re-establishing vascular channels. Keratinocytes from the wound margin start to migrate centrally within hours of tissue damage in physiologic wound repair, and two or three days after tissue damage, epithelial stem cells from the basal layer of the epidermis and hair follicle root sheath start to multiply [14][15]. Numerous chemical and physical signals, some of which originate from immune cells like pro-repair, anti-inflammatory macrophages, causes the formation of new blood vessels and reepithelialization. During the proliferative phase, vascular network restoration is crucial. The process of angiogenesis, or the creation of new blood vessels, involves two stages: vessel sprouting and vessel anastomosis. Fibroblast are the primary cell involved in the formation of granulation tissue, and they can be stimulated to produce pro-reepithelialization molecules by a number of macrophage-derived molecules, including TNF- α , IL-1, PDGF-bb, and platelets derived growth factor. Without IL-6, wounds exhibit reduced angiogenesis, collagen accumulation, and re-epithelialization in addition to an appropriate inflammatory response. TGF- β , a molecule primarily produced by wound-associated, pro-repair macrophage, also has a remarkable effect on fibroblasts. Both pro-repair macrophages and fibroblasts in the granulation tissue have an impact on keratinocyte re-epithelialization. Keratinocytes, Platelets, and activated pro-repair, anti-inflammatory macrophages produced the transforming growth factor- α , keratinocytes growth factor, a d epidermal growth factor, which start the process of re-epithelialization [16-17].

(iv) Maturation: When the granulation tissue has finished developing, the remodelling phase begins [18]. When granulation tissue turns into scars tissue, angiogenesis slows down and type III collagen in the granulation tissue is replaced by stronger type I collagen. It is important to remember that fully developed scars 80 percent of their original tensile strength [14]. After three weeks following tissue damage, there is net increase in collagen deposition, indicating that fibroblasts continue to deposit collagen for an extended period of time [13].

2.2 FACTOR AFFECTING HEALING

1) Diabetes: One of the main complications of both type 1 diabetes and type 2 diabetes is abnormal healing of wounds. In chronic conditions, nonhealing foot ulcers results in lower limb amputation. According to reports, 30-50% of diabetes individuals have peripheral neuropathy, demonstrating a clinically evident connection between wound healing and the

nervous system [18]. However, the majority of people are susceptible to developing chronic non-healing diabetic foot ulcer, which are thought to affect 15% of all diabetic all diabetics. Hypoxia remains prevalent in Diabetics foot ulcers, such as venous stasis illness and pressure-related chronic non- healing wounds. Chronic hypoxia, which can result from inadequate perfusion and angiogenesis, is harmful to the healing of wounds. By raising the amount of ROS, hypoxia can intensify the early inflammatory response and prolong damage. Diabetic foot ulcers are characterized by elevated metalloprotease levels, with chronic wound fluid containing nearly 60 times more MMP than acute wound healing. This elevated protease activity promotes tissue destruction and obstructs typical healing mechanisms. Another likely factor in wound healing is the neuropathy that develops in diabetes. Since they encourage cell chemotaxis, boost cell proliferation, and trigger growth factor synthesis, neuropeptides such as substance P, nerve growth factor, and calcitonin gene-related peptide are important for wound healing. Diabetics foot ulcer is caused due to reduction in neuropeptides [19].

2) Medications

i) NSAID'S: It has been demonstrated that non- steroidal medications reduce the granulocytic inflammatory response and have a depressive impact on wound healing. NSAID'S can lessen the pain by preventing the inflammatory prostaglandin PGE₂ from being produced. NSAID'S may promote the production of scars, particularly if they are used in the proliferative phase of healing as severe wound scarring also suppress PGE₂. NSAID'S also slows the process of haling by having antiproliferative impact on the skin and blood vessels [20]. In animal models, systemic ibuprofen has been shown to have an anti-proliferative effect on wound healing, leading to impaired angiogenesis, delayed epithelialization, decreased fibroblast counts, weakened breaking strength, and decreased wound contraction [19].

ii) Steroids: Steroid are used to treat conditions like autoimmune disease, cancer, and asthma. The anti-inflammatory and immunosuppressive properties of glucocorticoids can cause delayed healing, even though they are beneficial in treating rheumatoid arthritis and bronchospasm. Steroids' propensity to prevent wound contraction and reduce tensile strength has a detrimental effect on wound healing [20].

iii) Stress: Stress ha a significant effect on social behaviour and human health [19]. Stress can prevent the wound healing and a number of systemic diseases by causing endocrine hormone to become dysregulated. The nervous system and hypothalamus are affected by stress, which can cause an increase in the release of cortisol, glucocorticoid, norepinephrine, and adrenaline. These compounds may cause a reduction in leukocytes, immune response and cytokine release. These compounds may cause a reduction in leukocyte immune response and cytokine release, which may disrupt inflammatory processes and postpone the healings of wounds [20].

iv) Ageing: The cellular function of the body gradually and irrevocably deteriorates with age, making the elderly body more susceptible to environmental attacks and, ultimately, to chronic illness or even death [21]. One of the important factors that affect process of wound healing is aging. The epidermis layer thins with age, and metabolic and systemic changes can result from growing older. The inflammatory response of the elderly is altered in a number of ways, including a delayed migration of leukocytes to the area, a decrease in macrophage activity as phagocytosis, and a reduction in the release of growth factors and cytokines. In addition, re-epithelialization, delayed angiogenesis, decreased fibroblast activity, and collagen remodelling can all be caused by aging [20].

v) Alcohol: Ethanol intoxication at the time of injury is a risk factor for increased susceptibility to infection in the wound, and alcohol reduces host resistance. It appears that wound angiogenesis is most severely impaired, with a reduction of up to 61% after a single ethanol

exposure. Ethanol exposure also affects collagen synthesis, wound closure, re-epithelialization, and the proliferative stage of healing [22, 23].

vi) Smoking: Smoking cigarettes increases the risk of infection and scarring, making it a major risk factor that cause poor wound healing. There is various chemical derived from tobacco, such as nicotine, affects leukocytes and fibroblasts, impair the immune response and tissue oxygenation response, cause necrosis and poor collagen production and insufficient microbial eradication, all of which contribute to low- tensile wound strength [22]. Smoking is associated with impaired wound healing following surgery, along with an increased incidence of complications including infection, wound breakdown, anastomotic failure, tissue necrosis, and decreased wound strength. Nicotine induces vasoconstriction, limiting blood perfusion and resulting in ischemic conditions at the wound site. Cigarette smoke contains carbon monoxide, which causes tissue hypoxia in addition to nicotine [23].

vii) Sex Hormones: Acute wounds heal more slowly in older males than in older females. Then process of wound healing is significantly impacted by male androgens, female estrogens, and their steroid precursor dehydroepiandrosterone (DHEA). Estrogen can improve age- related healing impairment, whereas androgens negatively regulate cutaneous wound healing [24].

2) MARKETED FORMULATION AVAILABLE FOR WOUND HEALING:

i) Urgotul: Urgotul is a novel idea in wound care called lipido-colloid technology [25]. Lipido-colloid dressing technology accelerates wound healing and tissue regeneration in burn cases by stimulating fibroblast proliferation. Silver sulphadiazine, an antimicrobial agent and source of silver nanoparticle, incorporated into both hydrophilic and lipophilic matrices in Urgotul, a lipido- colloidal dressing technology. A thin, flexible layer of pure polyester gauze impregnated with silver nanoparticles embedded in a mixture of petroleum and hydrocolloidal polymers make up UrgoTUL, a non- occlusive dressing [26] (**Table 1**).

ii) CuraGel: Hydrogel wound dressing include Curagel Hydrogel Wound Dressings. Intended to keep the wound moist and protect it [25]. Hydrogel dressing, in contrast to conventional dressing like bandages and gauzes, are well known for their superior qualities, which include mechanical qualities that work with biological tissue and a remarkable capacity to retain water, which can keep the wound moist and continuously absorb exudate [26]. Clear gel makes it possible to examine the wound visually without taking off the dressing. Medical-grade hydroxypropyl methylcellulose's viscosity concentration in 2.0 mm per second. It has special rheological qualities that preserve the CuraGel's anatomical spaces because of its high viscosity and elastic behaviour. This solution has a new formula of 2.0% or 2.4% Plus, making it genuinely high viscosity. During an eye procedure, the high anterior segment is used. The viscosity of this product has been changed to allow for unrestricted manipulation during surgery while achieving the maximum zero shear rate viscosity [25].

iii) Xeroform: Xeroform is a medical dressing intended to shield and encourage wound healing. Its affordability, ease of application, steady healing rate, and low risk of infection make it a popular choice for split-thickness donor sites. Xeroform dressings offer antimicrobial and protective properties by coating non-adherent gauze with a petroleum and 3% bismuth tribromophenate mixture [25]. As the exudate drain, xeroform stays attached to the wound and serve as a scaffold for re-epithelialization [27]

iv) Polyskin: Polyskin Transparent Dressings represents a sophisticated approach to wound management, specially engineered to establish a protective barrier while maintaining breathability for the best possible healing. It is composed of a thin polymer that has been covered with a hypoallergenic glue. These dressing is particularly suitable for use on

intravenous access sites, ulcers, donor sites, surgical sutures and thermal injuries. Their transparent and semi-permeable configuration permits the transmission of oxygen and water vapour for optimum healing while obstruct the entry of pathogenic bacteria, water and other contaminants [24].

v) Permafoam: Permafoam is a sterile, non- medicated, single-us dressing made of polyurethane foam. The top layer of polyurethane is permeable to water vapour and has a special hydrophilic pore structure that promotes a high capillary effect, allowing wound exudate to be transported quickly and preserving a sterile and balanced moist wound environment. This facilitates autolytic debridement and encourages the growth of granulation tissue [24]. It comes in a variety of sizes and shapes. It is advised for both moderate to highly exudating acute and chronic wounds, such as abrasions, donor sites, incisions, leg ulcers, diabetic foot ulcers, and pressure ulcers grad II to IV [28].

vi) Duoderm: Duoderm is a hydrocolloid dressing that retain the moisture and is used on both partial and full thickened wounds that generate exudate. Its unique Convatec hydrocolloid formulations sets it apart from other commercially available standard dressings. Leg ulcers, traumatic ulcers. Acute wounds, donor sites, pressure ulcer, and partially thickened burns are amazing the conditions for which Duoderm Dressings is advised. Duoderm hydrocolloid dressings are effective in treating both superficial and deep partial- thickness burns. There are two types of Duoderm dressings: (i) duoderm Extra Thin dressing and (ii) Duoderm Signal dressing. The ultra-thin design of the Duoderm Dressings enables them to closely follow the body's natural contours, offering a discrete and comfortable fit. This makes it perfect for area that are visible, like the hands and face, where flexibility and appearance are crucial. The dressings is appropriate for active people and areas that are prone to friction because of thin profile, which guarantees that it adheres well without causing discomfort or limiting movement [24].

vii) Comfeel: Comfeel Plus is a hydrocolloid dressing that is semi-permeable and contains calcium alginate to enhance its absorption of low to moderate amounts of exudates. Transparent hydrocolloid dressing Comfeel Plus is used to treat low to moderately wounds, including surgical wounds, donor sites, leg ulcer, skin abrasion, pressure ulcers, and superficial partial-thickness burns [24].

Case on the effectiveness of Comfeel dressing: A Community- and Hospital- based comparative evaluation of Comfeel ulcer Dressing for Chronic leg ulcers

76 patients with 94 chronic leg ulcers were selected from community or hospital-based practises over a 21-month period and they were randomized to either Jelonet (Smith and Nephew) or Comfeel Ulcer Dressing (Coloplast Ltd). Three of the 48 ulcers treated with Jelonet in the 12-week assessment group healed, while 14 of the ulcers in the Comfeel group did the same. Additionally, Comfeel-treated ulcers responded noticeably better. The waterproof and cosmetic appearance of Comfeel Ulcer Dressings was praised by patients [29].

viii) Opsites: Opsite is a waterproof, biocompatible, adherent polyurethane film that is permeable to oxygen and water vapour. It can be used clinically as a secondary dressing and to create a moist wound environment in superficial wounds [30].

ix) Tegisorb: Tegisorb is a hydrocolloid composed of a polyisobutylene-based hydrocolloid adhesive with absorbent particles and an outer protective film.

On contact with exudate, it forms a gel that maintains a moist healing environment, and it is indicated for light to moderate wound [31]. Tegaderm hydrocolloid dressing serve two purposes in wound care: (i) The hydrocolloid adhesive's inner layer absorbs exudate rapidly,

outperforming the leading competing the dressing in absorbency over the first 48 hours. (ii) The permeable outer film layer consistently sustains a high rate of moisture vapour transmission in addition to having exceptional absorbency [24].

x) Vaseline Gauge: Vaseline gauze is a type of absorbent, fine-mesh gauze that has been impregnated with Vaseline to create a moist environment that aids in wound healing. Its functions as a primary dressing in conjugation with a secondary dressing that aids in exudate absorption, such as gauze, Comfeel, or changes. By keeping the secondary dressing from those aids in exudate absorption, such as gauze, Comfeel, or Duoderm, the Vaseline layer lessens trauma during dressing changes [24].

Case Study on Vaseline Gauze:

A third- degree burn that covered less than 10% of his total body surface area was sustained by a 67-year-old man with hypertension on his lower extremities. Two treatments were created from the wound: (i) Section A- Vaseline gauze was used extensively alone (ii) Section B- Vaseline gauze was applied followed by another Layer of dressing. External dressings were taken off for semi- exposure therapy nine days after surgery. In Section A, where full epithelialization was seen by day 11, healing proceeded more quickly. Section B, on other hand, healed to the same degree every day. These results demonstrate the benefits of Vaseline gauze in accelerating wound healing, preserving moisture balance, and lowering the risk of pain and infection in burn injuries [31].

xi) Allevyn: Allevyn is a highly absorbent, non-adhesive dressing made of hydrocellular foam that is antibacterial and composed of permeable, waterproof polyurethane film. The dressing is advised for wounds with moderate to extensive exudation since it is resistant to both water and germs. The hydro-cellular structure enables the wound to retain moisture and prevents adhesion to the wound surface. The hydro- cellular structure enables the wound to retain moisture and prevents adhesion to the wound surface [32].

xii) Multisorb: Multisorb swabs have a soft- textured, non- woven structure that minimizes linting and lowers the possibility of wound contamination because they are made from a viscous/polyester blend. These swabs maintain a low- lint profile, which make wound care safer than with traditional swab. Multisorb Non-Woven swabs are a better option than conventional gauze because they provide the ideal ratio of softness, absorbency, and low-linting qualities. Their capacity to absorb 40-65% more fluid than traditional alternatives guarantee efficient exudate management, which help to promote a safer and cleaner wound healing process.[33]

xiii) Purilon: Purilon gel is a safe and effective wound debridement solution because it is a mild, sterile hydrogel made entirely of natural ingredients without any additives. It is intended for necrotic and sloughy wounds, including pressure ulcers, leg ulcers, diabetic foot ulcers that are not infected, and burns of the first and second generation. It absorbs exudate, slough, and debris while also playing a vital role in moisturizing necrotic tissue. This dual- action characteristics promotes autolytic debridement and helps establish the ideal healing environment. Even after absorbing wound debris, Purilon gel's thick, cohesive consistency keeps it in place and stops leaks and maceration [34,35].

xiv) Silver Sulfadiazine: Since Fox created Silver Sulfadiazine from silver nitrate and sodium sulfadiazine in 1968 for its greater potency and minimal effects, including little pain upon application, Ag-SD has been a common treatment for burns for the past thirty years [36]. A common topical antibacterial for treating burns and chronic wounds is silver sulfadiazine. It combines sulfadiazine, an antibiotic belonging to the sulphonamides class, which has broad-

spectrum antibacterial qualities. AgSD is a recommended treatment choice for controlling and preventing infections in burns and other skin lesions because of its dual- action. The primary mechanism by which silver ions are released and interact with bacterial cell wall to cause bacterial cell death is the antibacterial action of silver. The silver component of Silver Sulfadiazine is effective against both Gram positive and Gram negative [37].

xv) Silver Alginate Gel Fibre:

Alginate, a naturally absorbent substance produced from seaweed, and silver are combined in Silver Alginate Fibre Gel, a topical wound care treatment, to promote wound healing and infection prevention. SAF Gel is a flexible choice for wound that require the capacity to absorb large volumes of exudate in addition to antimicrobial protection. It is used to treat moderate to heavily exudating, wounds, such as acute wounds like burns, trauma wounds, and surgical wounds, as well as chronic wounds like diabetic foot ulcers, and pressure sores [37].

Table 1: Comprehensive detail notes on commercial product available for wound healing

S.NO.	TYPE OF FORMULATION	ADVANTAGE	DISADVANTAGE	REFERENCE
i)	Vaseline Gauge	Easy to apply	Interrupt wound healing	[24, 31]
ii)	Xeroform	Lower infection risk	May harm forming tissue	[25,27]
iii)	Tegasorb	Support autolytic degradation	Can stick to wound	[24,31]
iv)	Urgotul	Speeds healing	Do not retain water	[25,26]
v)	polyskin	Highly flexible	Tough to remove, may disturb skin layers.	[24]
vi)	Opsite	Act as secondary dressing	Hard to remove, may disturb epidermal layer	[30]
vii)	Curagel	Preserve high moisture wound	Adherence problem	[25,26]
viii)	Permfoam	Very absorbent	Firmly stick wound when dry	[33]
ix)	Duoderm	Waterproof and provide protection against contamination	Very thick	[24]
x)	Multisorb	Easily applied on skin	Not effective for wound with lot of exudates	[33]
xi)	Silver Sulfadiazine	Low cost	Lack mechanical strength	[36]
xii)	Purilon	Maintain a moist environment	Unsuitable for heavily exudating wounds	[34,35]

xiii)	Allevyn	Highly absorbent	Need secondary dressing for secure coverage	[32]
xiv)	Silver Alginate Gel fibre	Hydrate wound bed and support skin condition	Lack mechanical strength and prone to tear	[37]
xv)	Comfeel	Promote autolytic debridement	Strong adhesion may stick to wound	[24]

3) LITERATURE REVIEW (EXPERIMENTAL STUDIES):

(i) Curcumin as a Wound Healing Agent (Akbik et al., 2014): This article reviews curcumin's wound healing qualities, emphasizing the necessity of optimal topical application while highlighting its capacity to improve granulation tissue formation, tissue deposition, collagen deposition, and wound contraction, as well as to lessen inflammation and oxidation [38].

(ii) Wound Healing Activity of Topical Application Forms Based on Ayurveda (Datta et al., 2011): They selected excision and incision wound healing models for the initial efficacy assessment of anti-aging activity. They examined parameters such as collagen content, wound contraction, and skin breaking strength, which are indicative of the skin's mechanical strength, collagenation capacity, and tissue cell regeneration capacity. The formulations containing Yashada bhasama, Shorea robusta resin, and flex seed oil were found to be effective potential anti-aging formulations because the group treated with them demonstrated significantly better contraction, higher collagen content, and better skin breaking strength [39].

(iii) A Prospective Randomized Study to Compare the Effectiveness of Honey Dressing vs. Iodine Dressing in Chronic Wound Healing (Gulati et al., 2014): In this study, a total of 45 participants were randomly assigned to one of two groups: the Honey & Povidone iodine dressings group. For the duration of the 6-week follow-up period, dressing was done every other day. The primary outcome measure was full recovery after six weeks. Up to six weeks, the status of wound healing was evaluated every two weeks. The results indicated that no patients in the povidone iodine group (n=20) experienced complete healing, while 7 of 22 patients receiving honey dressing achieved full recovery. Additionally, at the 0.05 significance level, honey treatment was associated with significant improvements in wound size reduction, pain relief, and overall comfort compared to povidone iodine. When it comes to healing chronic wounds, honey dressings works better than povidone iodine dressings [40].

(iv) Curcumin: A novel therapeutic for burn pain and wound healing (Cheppudira et al., 2013): This article discussed recent clinical preclinical research on the effects of analgesic and wound healing. Studies aiming at creating better curcumin delivery system that enhance its bioavailability. Based upon the evidence it is hypothesized that curcumin's has two useful effects-analgesic and improved wound healing – are mediated by common anti-inflammatory pathways. Recent research has shown that curcumin is a promising experimental for the treatment of wounds and pains [41].

(v) Topical Application of Aloe vera Accelerated Wound Healing, Modelling, and Remodelling: An Experimental study (Oryan et al., 2016): As per the studies conducted, the biological, morphological, and biochemical properties of the rat's healing cutaneous wounds were enhanced by the topical application of Aloe Vera. The methodology involved a

rectangular 2*2 cm cutaneous wound was made on rats' dorsum back. The animals were split into three groups at random: 20 control, 20 low-dose, and 20 high dose A. vera. For 10 days, control treated with saline, while low dose (25mg/mL), and high dose (50mg/mL) A. vera gel were applied topically to the treated animals, respectively. Monitoring was done on wound contraction, wound surface, and epithelialization [42].

(vi) Beneficial Effects of the Genus Aloe on Wound Healing, Cell Proliferation, and Differentiation of Epidermal Keratinocytes (Moriyama et al., 2016): According to findings, human primary epidermal keratinocytes and a model that mimics human skin showed markedly better wound healing when exposed to both aloe vera gel (AVG) and cape aloe extract (CAE). Furthermore, flow cytometry analysis showed that HPEKs treated with AVG and CAE had higher cell surface expression of $\alpha 6$, $\beta 1$, $\beta 4$ - integrin and E-cadherin. These increases might aid in wound healing and cell migration. During the Aloe treatment significant alterations in cell cycle progression and increased in cell number was observed. Aloe boosts HPEK differentiation marker gene expression, indicating that AVG and CAE may play a part in enhancing keratinocyte function [43].

5) Ayurveda Approach of Wound Healing:

The universal approach of aging started ago, when life first began. The combination of extrinsic (primarily photoaging) and intrinsic (chronological) factors causes skin aging.

Elderly people may be more susceptible to tissue breakdown because metalloproteases, which are proteases linked to intrinsic aging, seem to be more prevalent and less challenged. Therefore, wound healing animal models can be used to evaluate the integrity of the skin structure, collagenation (collagen synthesis), epithelialization, connective tissue deposition, and tissue cell regeneration [39].

The ancient Indian medical system known as Ayurveda has been used for thousands of years. The three major classics that provide in-depth descriptions of more than 700 herbs are Atharvaveda, Charak Snhita, and Sushrut Samhita. Chronic and complicated wounds continue to pose a significant challenge in clinical practices worldwide due to impaired healing process and their limitations to currents treatments. Traditional herbal medicine has a long, culturally rooted history in promoting wound healing and remains trusted by many due to its perceived safety and accessibility. Unlike isolated synthetic pharmaceuticals used in allopathic medicine, whole herbs contain a complex mixture of bioactive constituents that work synergistically to promote wound healing [43]. Some of the herbs that promote wound healing are described below (Table 2):

Table 2: Medicinal Plants in Wound Healing: Mechanism of Action, Adverse Effects, and Evidence-Based Review

Herb	Part used	Side effects	Clinical Evidence	Interaction	MOA	Reference
Curcuma longa	Rhizome	Could increase the anti-inflammatory effects of other medicines	Animal trails and clinical studies	Interact with NSAIDS, warfarin, antiplatelets drugs and antihyperlipide mic drugs	Increase the activity of fibroblast and collagen synthesis.	[38]

Avnena	Fruits	Skin reaction in patients with contact dermatitis	Clinical & dermatologic al studies	Minimal	Improves skin barrier, anti-inflammatory	[44]
Emblica officinalis	Crushed bark	Not significant	Animal trails	Minimal	Antioxidant property and enhance collagen synthesis.	[45]
Aloe Vera	Inner leaf mucilage	Hypersensitivity of aloe	Animal trails	Minimal	Angiogenesis, growth factor production cytokine synthesis	[42]
Ficus religiosa	Roots, hydroalcoholic extract of leaves, bark	Not significant	Animal studies	Minimal	Contract wound, angiogenesis	[46]
Ginkgo biloba	Leaves	Headache, hypersensitivity to skin, Dizziness	Case studies and animal studies	Anticoagulant, NSAID, Antidiabetics,	Encourage epithelialization	[47]
Allium Sativum	Outer layer of garlic bulb	allergy, rashes	Animal study	Anticoagulant drugs	Enhances re-epithelialization, antioxidant	[48]
Hypericum perforatum	Full plant [23]	phototoxicity	Case studies and animal studies	Indinavir, anticoagulant, etc.	Contract wound and re-epithelialization.	[49]

Conclusion

In summary, wound healing is a complex and strictly controlled biological process that is impacted by a wide range of physiological and pathological variables. The current evaluation emphasizes the relative importance of conventional commercial formulations and herbal remedies in supporting efficient wound care. Because of their natural source and variety of pharmacological effects, including anti-inflammatory, antioxidant, antibacterial, and collagen-enhancing qualities, herbal agents provide a viable and safer substitute or supplement to traditional treatments. Curcumin, aloe vera, and other substances derived from plants show great promise for speeding up tissue repair and enhancing overall healing results.

However, non-herbal formulations such as hydrogels, hydrocolloids, and silver-based dressings remain essential in clinical practice because they provide structural support for wound healing, infection prevention, and regulated moisture balance. These formulations are essential to contemporary wound care because of their reliable clinical performance and simplicity of use.

Crucially, the research indicates that by using the benefits of both systems, an integrated strategy combining herbal and conventional medications may provide better therapeutic results. Nevertheless, more thorough, well-designed clinical studies are still required to confirm the effectiveness, safety, and standardization of herbal formulations despite promising experimental and clinical results.

All things considered, improving wound care requires a well-rounded, evidence-based approach that combines conventional wisdom with cutting-edge scientific research to improve patient care and healing effectiveness.

Conflict of Interest

The authors have no conflict of interest.

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