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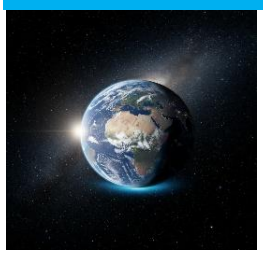

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Management of chronic wounds: a current review for internal medicine physicians

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Abstract

Background: Chronic wounds are a growing clinical problem in older patients especially those with multimorbidity. Their healing is impaired by constant inflammation, skin cell dysfunction and bacterial biofilm presence.
Aim: The aim of this review article is to present the current management of chronic wounds in internal medicine practice.
Methodology: The study was conducted as a narrative review. A structured literature search was carried out using the following electronic databases: PubMed, Scopus, Web of Science, and Google Scholar. The search included publications released mainly between 2017 and 2025, in order to reflect current clinical practice and guideline recommendations.
Results: The reviewed literature and articles confirm that internal medicine doctors play a key role in the chronic wound treatment. Firstly, they should concentrate on the etiology of the wound. Comorbidities are also worth considering. The TIME strategy is a structured framework for dealing with wounds. Selected additional therapies are mentioned including negative pressure wound therapy and larval therapy which are used to improve debridement and reduction of bacterial biofilm. All of these actions contribute to reducing the risk of complications, hospitalizations and amputations.
Conclusion: Chronic wounds are still a complex and heterogeneous clinical problem. It requires an individualized diagnostic and therapeutic approach based on etiology. The internist actions are essential in early patient assessment, management of comorbidities and coordination of multidisciplinary care. In some cases advanced therapies such as negative pressure wound therapy or larval therapy need to be performed in order to achieve a better outcome.

Keywords: chronic wounds; TIME; internist; larval therapy; ulcers

Introduction

Chronic wounds are defined as skin defects that fail to heal within at least six weeks. They represent a growing clinical problem, particularly in the ageing population and among patients with chronic conditions such as diabetes mellitus, chronic venous insufficiency, peripheral arterial atherosclerosis, or prolonged immobilization [1],[2]. The presence of a chronic wound leads not only to pain and functional impairment but also to a significant reduction in patients' quality of life and an increased demand for medical care, generating substantial costs for healthcare systems [3],[4].

In clinical practice, the internist is often the first specialist to assess a patient with a chronic wound. The internist initiates the diagnostic process, identifies the potential etiology of the lesion, implements causal treatment, optimizes the management of comorbid conditions, and coordinates further specialist care [5],[6]. Therefore, up-to-date knowledge of wound pathophysiology, principles of comprehensive wound assessment, and modern therapeutic strategies is essential for the appropriate management of these patients in everyday internal medicine practice [7],[8].

The aim of this article is to present current, evidence-based principles for the management of chronic wounds in internal medicine practice, with particular emphasis on the TIME strategy as the foundation of local wound care [7], modern dressing techniques, negative pressure wound therapy [9],[10], larval therapy [11] and adjunctive methods supporting wound healing within the context of causal and multidisciplinary treatment.

Methodology

This study was conducted as a narrative review of the literature. The aim was to present and systematize current knowledge on the pathophysiology, prevention, management of chronic wounds with particular attention to diabetic foot ulcers, chronic venous disease, peripheral arterial disease and selected advanced wound treatment methods.

A structured literature search was carried out using the following electronic databases: PubMed, Scopus, Web of Science, and Google Scholar. In addition, documents published by international and national scientific societies and professional organizations were reviewed. The search included publications released mainly between 2017 and 2025, in order to reflect current clinical practice and guideline recommendations.

The search strategy was based on combinations of the following keywords: chronic wounds, diabetic foot ulcer, wound management, wound pathophysiology, negative pressure wound therapy, larval therapy, peripheral arterial disease, chronic venous disease, compression therapy, and off-loading.

Results

Pathophysiology of Chronic Wounds

Wound healing is a dynamic biological process comprising three consecutive phases: the inflammatory, proliferative, and remodeling phases. In chronic wounds, the balance between these stages is disrupted, leading to an arrest of tissue repair at the level of a persistent inflammatory response. A key role is played by the complex interaction of dysfunctional cells, molecular disturbances, alterations in the wound microenvironment, and chronic microbial colonization.

One of the earliest abnormalities observed in chronic wounds is keratinocyte dysfunction, characterized by impaired migration and differentiation, which prevents proper re-epithelialization. Contemporary studies demonstrate dysregulation of signaling pathways responsible for epidermal cell proliferation and migration, resulting in ineffective restoration of the skin barrier.

Cellular senescence, defined as premature aging of skin cells, also plays a significant role. Senescent cells exhibit an altered secretory profile (senescence-associated secretory phenotype, SASP), including excessive production of pro-inflammatory cytokines, proteases, and regeneration-inhibiting factors. This phenomenon perpetuates chronic inflammation,

suppresses reparative processes and affects not only keratinocytes but also fibroblasts and endothelial cells.

Another key component of chronic wound pathophysiology is the persistence of chronic inflammation. In normal wound healing, immune responses are reprogrammed to allow progression to the proliferative phase. In chronic wounds, a predominance of a pro-inflammatory phenotype is observed, particularly M1-type macrophages, resulting in excessive production of cytokines such as IL-1, IL-6, TNF-α, as well as dysfunction of neutrophils and mast cells.

Abnormalities also involve fibroblasts and the extracellular matrix (ECM). Fibroblasts in chronic wounds show reduced proliferative capacity and impaired collagen synthesis, often displaying features of cellular senescence. Concurrently, excessive activity of matrix metalloproteinases (MMPs) with a relative deficiency of their tissue inhibitors (TIMPs) leads to degradation of ECM components and growth factors, preventing the formation of stable granulation tissue [1],[2].

In addition, chronic wounds are characterized by impaired angiogenesis and microcirculation. Tissue hypoxia, dysregulated pro-angiogenic signaling, and a reduced number of endothelial progenitor cells result in insufficient neovascularization and compromised perfusion, significantly inhibiting the healing process [1].

A crucial factor sustaining the chronic nature of wounds is the presence of bacterial biofilm formed by microorganisms colonizing the wound bed. Biofilm acts as both a mechanical and biological barrier, limiting drug penetration and protecting pathogens from the host immune response. Its presence promotes persistent inflammation and further tissue destruction [11], [12].

The complexity of these mechanisms necessitates a multidirectional therapeutic approach to chronic wounds, aimed not only at infection control and appropriate dressing selection but also at modulation of the wound microenvironment, improvement of tissue perfusion, and restoration of normal cellular function involved in the healing process [1],[7].

The TIME Framework

The wound healing is complex proces. It depends on both patient-related factors and environmental influences [1]. Internal factors include overall health status, immune system function, age, extremes of body weight, the presence of diabetes, and nutritional status—all of

Element	What does it mean?	Therapeutic goal	Examples of interventions
T – Tissue	Assessment of tissue types in the wound bed: healthy granulation tissue, necrotic tissue, slough, eschar.	Removal of non-viable tissue and preparation of the wound bed for healing.	Surgical debridement, enzymatic debridement, autolytic debridement (hydrogels), mechanical debridement, larval therapy
I – Inflammation / Infection	Assessment of signs of local infection, biofilm, inflammation, and exudate.	Reduction of inflammation and bacterial burden; disruption of biofilm.	Dressings with silver ions, PHMB, or povidone-iodine, antibiotic therapy only in clinically infected wounds, debridement, specialized anti-biofilm dressings
M – Moisture Balance	Assessment of exudate level: too little / optimal / too much.	Maintenance of an optimally moist wound environment to accelerate healing.	Polyurethane foam dressings, alginates (for heavy exudate), hydrocolloids (for low exudate), hydrogels (for dry wounds), NPWT for excessive exudate
E – Edge	Assessment of epithelialization progress, rolled wound edges, and hyperkeratosis.	Stimulation of epithelialization and advancement of wound edges.	Removal of hyperkeratosis, debridement of rolled wound edges, negative Pressure Wound Therapy (NPWT), biological therapies (PRP, growth factors)

which affect the body's ability to mount an appropriate inflammatory and regenerative response [2],[5]. External factors include mechanical stress acting on the wound, the presence of contaminants or foreign bodies, inappropriate wound temperature, excessive drying or tissue maceration, infection, exposure to chemical substances, and other environmental elements such as tobacco smoking or the use of certain medications [7].

Table 1. Components of the TIME strategy and their clinical significance.

The diversity and coexistence of these factors make the healing process particularly vulnerable to disruption, often resulting in the development of a chronic wound [1]. To facilitate wound assessment and standardize therapeutic management, the TIME framework is used; its individual components are summarized in Table 1. TIME encompasses four key therapeutic domains: T (Tissue) – assessment and debridement of non-viable tissue; I (Inflammation/Infection) – control of inflammation and infection; M (Moisture) – maintenance of moisture balance; and E (Edge) – evaluation of the wound edges and stimulation of epithelialization. This model enables systematic identification of the major barriers to healing

and supports the selection of appropriate therapeutic interventions, which is particularly important in the management of patients with chronic wounds [7], [8],[25].

Introduction to Specific Types of Chronic Wounds

Chronic wounds represent a heterogeneous group of conditions that differ in both pathophysiology and therapeutic requirements [1],[2]. Effective management therefore requires not only an assessment of the wound bed itself, but above all a precise determination of the underlying etiology [7]. Each type of chronic wound - whether venous, diabetic, ischemic, or a pressure injury - develops under distinct biological conditions, which translate into different mechanisms of impaired healing and specific therapeutic goals [3], [5],[13].

Understanding the fundamental differences between these categories is essential for selecting appropriate causal treatment, optimizing local wound therapy, and adequately modifying systemic factors [7]. In clinical practice, this approach enables the planning of management strategies that are consistent with both the TIME framework and current scientific society guidelines, ultimately improving patient prognosis [5],[7].

The following subsections discuss the most common types of chronic wounds, outlining their characteristic clinical features and current therapeutic options, including causal and local treatment as well as adjunctive methods supporting the wound healing process.

Venous Etiology Wounds

Chronic venous insufficiency is one of the most common vascular disorders and may affect nearly half of the adult population over 18 years of age in Poland. Its development is primarily driven by venous valve incompetence, impaired vessel patency, reduced vascular wall tone, and insufficient function of the calf muscle pump. These mechanisms promote venous reflux, blood stasis, and sustained venous hypertension, which constitute the principal pathophysiological factors underlying venous leg ulcers [6]. The severity of chronic venous insufficiency and the characteristics of venous ulcers are assessed using the CEAP classification, which incorporates clinical, etiological, anatomical, and pathophysiological aspects of the disease. Duplex Doppler ultrasonography is considered the diagnostic gold standard for identifying venous insufficiency and assessing reflux, allowing detailed evaluation of valve function and venous patency [14].

Venous leg ulcers are the most common cause of chronic wounds and occur predominantly in individuals aged between 50 and 80 years. They are characterized by a high recurrence rate, reaching up to 20–50% within the first year after healing, particularly in cases of inadequate compression therapy or absence of causal treatment [7]. Venous ulcers are defined as full-thickness skin defects, usually located in the medial malleolar region, that show no tendency for spontaneous healing. Lesions are typically oval in shape, with a flat wound bed covered by fibrinous tissue and often accompanied by exudate or purulent discharge. Common accompanying features of chronic venous insufficiency include lower limb edema, skin hyperpigmentation, lipodermatosclerosis, and telangiectasias [6],[7].

Compression therapy remains the gold standard of treatment, aiming to reduce venous hypertension, improve venous return, and stimulate microcirculation. Appropriately applied compression accelerates wound healing, reduces edema, and lowers the risk of recurrence. Compression can be delivered using multilayer bandaging systems, ready-made compression systems, or compression stockings individually fitted to the patient [7].

Although compression therapy is the cornerstone and most effective treatment for venous ulcers and chronic venous insufficiency, several contraindications must be considered prior to its initiation. The most important is severe arterial perfusion impairment, defined as an ankle–brachial index (ABI) < 0.5, which constitutes an absolute contraindication due to the risk of worsening ischemia. Caution is also required in patients with ABI values between 0.5 and 0.8, who may require modified compression protocols [16].

Other contraindications include acute inflammatory and infectious conditions of the limb, such as active cellulitis, as well as “florid” venous ulcers characterized by heavy exudation and marked inflammation. Compression should not be applied in patients with metabolic edema (e.g. due to renal failure or liver cirrhosis), in whom compression does not address the underlying cause of swelling [7].

Additional contraindications include heavily exudative dermatoses and severe forms of eczema or dermatitis, where compression may exacerbate skin irritation. Particular caution is required in patients with diabetic macroangiopathy or microangiopathy, who are at increased risk of impaired perfusion and delayed healing [16].

Acute, untreated, or progressive deep vein thrombosis represents a relative contraindication; compression may be initiated only after anticoagulant therapy has commenced and specialist assessment has been performed. Another contraindication is peripheral neuropathy with sensory

impairment, which increases the risk of trauma, pressure injuries, and uncontrolled compression [7]. Compression therapy should also be avoided in patients with active autoimmune or inflammatory diseases, such as systemic lupus erythematosus or acute inflammatory arthritis, when significant inflammation or joint swelling is present.

In parallel with causal treatment, proper wound bed management according to the TIME strategy (Tissue, Inflammation/Infection, Moisture balance, Edge of wound) is of key importance. This model enables systematic assessment of the ulcer bed and identification of factors that impede healing. In practice, it includes debridement of necrotic tissue, control of inflammation and microbial burden, maintenance of optimal wound moisture, and assessment and stimulation of epithelialization at the wound edges. Implementation of the TIME strategy is essential for compression therapy and causal treatment to achieve full clinical effectiveness [7].

An additional crucial aspect of management is care of the periwound skin, which in chronic venous insufficiency is often altered, dry, irritated, or inflamed. Neglecting periwound skin care increases the risk of maceration, skin breakdown, infection, and progression of trophic changes. The use of emollients that restore the hydrolipid barrier is recommended, preferably preparations containing 5–10% urea, ceramides, or other moisturizing agents. In cases prone to maceration, the skin should be protected with barrier products (e.g., zinc oxide creams or polymer-based protective films). Short-term use of mild topical corticosteroids may be considered in the presence of inflammation, whereas low-concentration keratolytic agents containing urea or salicylic acid may be used for excessive hyperkeratosis [7],[8]. Accurate etiological assessment, appropriate application of compression therapy, and proper wound bed management together form the foundation of effective treatment of venous leg ulcers [6].

Arterial Etiology Wounds

Arterial wounds arise as a consequence of chronic lower limb ischemia, most commonly associated with peripheral arterial atherosclerosis [13], [14]. This condition affects approximately 3–10% of the adult population, with prevalence increasing with age and the presence of risk factors such as diabetes mellitus, tobacco smoking, arterial hypertension, dyslipidemia, and chronic kidney disease [2],[17]. Progressive arterial narrowing or occlusion leads to impaired tissue perfusion, hypoxia, and accumulation of metabolic by-products, which

in turn disrupt the wound healing process and promote the development of ischemic ulcers [1],[13].

Arterial ulcers are most commonly located on the toes, heel, lateral border of the foot, or around the lateral malleolus. They are characterized by well-demarcated edges, minimal exudate, yellowish or black necrotic tissue, and significant pain, which is often exacerbated at night and in the supine position [18]. The periwound skin is typically cool, pale, or cyanotic, often shiny and hairless. Additionally, diminished or absent peripheral pulses are observed, along with classic symptoms of chronic limb ischemia such as intermittent claudication and, in more advanced stages, rest pain [14], [17].

In the diagnostic evaluation of ischemia, the ankle–brachial index (ABI) serves as the primary screening tool. ABI values below 0.9 indicate the presence of peripheral arterial disease, while values below 0.5 are indicative of severe ischemia and necessitate urgent vascular consultation [13],[14]. In patients with diabetes or advanced medial arterial calcification, the toe–brachial index (TBI) provides greater diagnostic reliability due to non-compressible vessels [5],[18]. Duplex Doppler ultrasonography is the first-line imaging modality for assessing hemodynamic flow, whereas computed tomography angiography, magnetic resonance angiography, or conventional angiography are employed for planning revascularization strategies [14],[17].

The management of arterial wounds is primarily based on causal treatment aimed at improving tissue perfusion through revascularization. Endovascular techniques such as balloon angioplasty, atherectomy, and stent implantation, as well as open surgical procedures including arterial bypass, are commonly employed [13], [14]. In patients who are not candidates for revascularization, supportive pharmacotherapy (e.g., cilostazol) and aggressive modification of cardiovascular risk factors—such as smoking cessation, blood pressure control, and glycemic optimization—are essential [2],[17]. Comprehensive pain management and limb offloading are also of critical importance.

Local wound management must be tailored to the severity of ischemia and the current condition of the wound. Within the TIME framework, priority is given to cautious, selective debridement of necrotic tissue, avoidance of aggressive debridement in patients without prior restoration of blood flow, and control of local inflammation [5],[7]. Due to typically low levels of exudate, dressings that maintain a moist wound environment—such as hydrogels or thin hydrocolloids—are commonly used. In painful wounds, analgesic or silicone-based dressings may be beneficial [7]. In contrast to venous ulcers, compression therapy is absolutely contraindicated in patients

with significant ischemia, as it may further impair perfusion and precipitate tissue necrosis [13], [14]. An important component of treatment is the care of the periwound skin, which in ischemic patients is often thin, fragile, and prone to injury. Gentle cleansing, the use of light-textured emollients, and avoidance of products that may cause maceration are recommended [7]. Injury prevention, protection against friction, and patient education regarding appropriate footwear are key measures to prevent further complications.

Arterial wounds result from chronic ischemia and are characterized by impaired healing due to reduced tissue perfusion. Early diagnosis of vascular impairment and prompt initiation of causal treatment—particularly revascularization—are crucial for improving outcomes [13],[14]. Local wound care must be cautious and adapted to the degree of ischemia, while meticulous skin care and injury prevention remain essential elements of comprehensive patient management [7].

Diabetic Foot Ulcer

Diabetes affects approximately 500 million people worldwide, and up to 25% of patients will develop a chronic, hard-to-heal foot ulcer during their lifetime [3],[4]. Diabetic foot disease is defined as a condition affecting the feet of a person with diabetes in whom at least one of the following disorders is present: peripheral arterial disease, peripheral neuropathy, infection, ulceration, neuro-osteoarthropathy, gangrene, or a history of amputation [5],[15],[19] In some patients, a diabetic foot ulcer develops and is associated with exceptionally high mortality, reaching up to 50% within five years after ulcer onset [3]. From an etiopathogenetic perspective, diabetic foot ulcers are classified as neuropathic, ischemic, or neuro-ischemic [18], [20]. Differentiation between these forms is presented in Table 2.

Table 2. Differentiation between neuropathic and ischemic component

Foot / Features	Neuropathic component	Ischemic component

Skin	Pink/red, warm, dry	Pale, bluish, cold, trophic changes
Skin appendages	+	–
Pulse	++	–
Pain at rest	++ (often precedes ulceration; burning, stabbing, tingling)	– / +
Pain during movement	– / +	+ (may be absent)
Pain during wound care	–	+++
Lesion location	Plantar surface, pressure points	Distal parts of the foot, dorsal surface
Pain, temperature, touch, vibration sensation	Impaired / absent	Initially normal, later hypersensitivity
Wound characteristics	Callus, ulceration, moist necrosis	Ulceration, dry or moist necrosis if infected
Foot X-ray	Osteolysis often present	Osteolysis rarely present

Prevention plays a key role and includes regular screening examinations such as assessment of protective sensation using a 10-g monofilament and palpation of pulses of the dorsalis pedis and posterior tibial arteries [15]. The frequency of preventive assessments depends on the individual risk category for ulcer development: very low risk—once yearly; low risk—every 6–12 months; moderate risk—every 3–6 months; high risk—every 1–3 months [5],[19]. Additional preventive measures include daily self-inspection of the feet, podiatric care, use of appropriate footwear with individually fitted insoles, foot-strengthening exercises, avoidance of walking barefoot, adequate glycemic control, and immediate treatment of even minor skin injuries [3], [15].

At the primary care level, the first step should be patient assessment using the SINBAD classification (Site, Ischaemia, Neuropathy, Bacterial infection, Area, Depth) and the WIfI classification (Wound, Ischaemia, Foot Infection) [5],[20]. For most patients, the therapeutic target is an HbA1c level below 7%; however, in elderly patients or those with multiple

comorbidities, values of 8–8.5% may be acceptable [15], [19]. In cases of infection exacerbation or ulcer deterioration, a temporary switch from oral antidiabetic agents to insulin therapy should be considered [15]. Screening assessment of lower limb arteries (ankle–brachial index, hand-held Doppler examination, duplex Doppler ultrasonography) is recommended in all patients with diabetes over 50 years of age [5],[18].

Infection is a common complication of diabetic foot ulcers. Patients with severe infection (fever, tachycardia, tachypnea, markedly elevated C-reactive protein) or moderate infection in the presence of comorbid conditions should be hospitalized. Microbiological samples should be obtained prior to initiating antibiotic therapy. Empirical treatment options include amoxicillin 1 g two to three times daily, cloxacillin 0.5 g four times daily, or clindamycin 0.6 g three times daily. In cases with a risk of Gram-negative infections—particularly in patients with prior antibiotic exposure or hospitalization—trimethoprim–sulfamethoxazole 960 mg twice daily or levofloxacin 500 mg twice daily may be considered. Antibiotic therapy should be adjusted according to culture results, and antibiotics are not recommended for ulcers without clinical signs of infection [21]. Limb offloading is one of the most important components of treatment. Reducing pressure at the ulcer site accelerates healing and decreases the risk of recurrence [3],[22]. Various offloading methods are used, including total contact casts, removable walker-type orthoses, specialized therapeutic footwear, or individually customized insoles designed to reduce pressure on the ulcer [22]. Local wound care requires systematic monitoring and appropriate wound management. Basic principles include wound cleansing and irrigation, removal of contaminants, selective debridement of necrotic tissue, mechanical disruption of biofilm, drainage of superficial fluid collections, and removal of surrounding hyperkeratosis [5]. Dressings should be changed every 2–3 days and selected individually based on the amount of exudate and tissue condition [7]. Hospitalization is required in patients with severe infection, rapidly progressing tissue destruction, suspected involvement of bone or deep structures, critical limb ischemia, inability to achieve effective offloading, or when outpatient care is insufficient [19],[21].

Diabetic foot disease is a complex condition resulting from the interaction of neuropathy, ischemia, and infection, and its consequences significantly worsen patient prognosis [1],[3]. Prevention and early identification of risk factors are of paramount importance, while effective treatment requires simultaneous limb offloading, infection control, optimization of metabolic

control, and appropriate local wound management [5],[15]. An integrated, multidisciplinary approach forms the cornerstone of improved outcomes and reduced risk of amputation [3].

Pressure Injuries

Pressure injuries constitute a serious clinical problem both in hospital settings and in long-term and home care. They may develop as early as within two weeks of immobilization, and the risk of their occurrence is further increased by factors such as malnutrition, loss of subcutaneous tissue, advanced age, chronic diseases, and limited mobility. Their pathogenesis is associated with prolonged pressure on soft tissues, leading to impaired perfusion, tissue hypoxia, cellular damage, and ultimately necrosis [7],[23].

Since 2019, the classification of pressure injuries has been updated by the European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Injury Advisory Panel (NPIAP) and includes four stages: skin injury with non-blanchable erythema, partial-thickness loss of dermis, full-thickness loss of skin and subcutaneous tissue, and full-thickness tissue loss with exposed muscle, tendon, or bone. In addition, two categories of unclassifiable pressure injuries are distinguished: unstageable pressure injury (with necrosis preventing assessment of wound depth) and deep tissue pressure injury, characterized by damage to deep tissues with intact, often unbroken, overlying skin.

Prevention remains the most effective strategy for reducing the incidence of pressure injuries. It includes identification of risk factors, regular skin monitoring, assessment using standardized tools (e.g., the Braden Scale), maintenance of appropriate skin hydration, and prevention of excessive moisture and maceration. Pressure-relieving interventions also play a crucial role, including the use of alternating-pressure mattresses, offloading pads, specialized prophylactic dressings (e.g., heel protectors), and frequent repositioning of the patient [7],[23].

Management of pressure injuries depends on the extent of tissue damage, the patient's nutritional status, the presence of infection, and overall prognosis. Appropriate local wound care in accordance with the principles of the TIME strategy is essential and includes tissue assessment, control of inflammation, maintenance of optimal moisture balance, and support of epithelialization [7]. A moist wound environment is not always desirable: in the presence of necrosis and deep tissue destruction, hydrocolloid or hydrogel dressings are not recommended, as they may promote infection and delay healing [2],[7]. Surgical, sharp, or enzymatic debridement, as well as larval therapy, should be considered once necrosis has demarcated.

Only after effective removal of necrotic tissue can autolytic methods be safely implemented [7],[11].

In infected pressure injuries, appropriate microbiological assessment is required, and antibiotic therapy should be used only when clinical signs of infection are present. Equally important are pressure offloading at the injury site and modification of systemic factors, such as optimization of nutritional status, metabolic control, and management of comorbid conditions [7], [23].

Larval Therapy

Larval therapy (LT), also known as maggot debridement therapy (MDT), involves the controlled application of sterile larvae of the fly Lucilia sericata to the wound bed of a chronic wound. The larvae selectively digest necrotic tissue while sparing viable tissue, and through their secretions and excretions they exert proteolytic, antibacterial, anti-inflammatory, and pro-healing effects [11],[12].

Larval-derived products effectively inhibit the formation of bacterial biofilms and degrade existing biofilms, particularly those formed by Staphylococcus aureus and Pseudomonas aeruginosa. In addition, these products increase the susceptibility of biofilms to certain antibiotics [11]. This suggests a potential role for larval therapy not only as a method of wound debridement but also as a tool for biofilm management, which represents one of the major barriers to the healing of chronic wounds. Larval therapy is at least as effective as standard methods (e.g., surgical debridement, hydrogel dressings) in achieving complete wound debridement, with a tendency toward faster and more thorough removal of necrotic tissue, although the differences did not always reach statistical significance.

In summary, available evidence indicates that larval therapy is a valuable adjunctive method in the management of chronic wounds, especially in the context of necrotic tissue removal and biofilm modulation. Its use may be considered in patients with infected, non-healing ulcers, particularly when conventional debridement methods are insufficient or contraindicated [11],[12].

Negative Pressure Wound Therapy

Negative pressure wound therapy (NPWT) involves the application of an airtight dressing connected to a pump that generates negative pressure, enabling controlled removal of exudate, reduction of edema, and mechanical stimulation of the wound bed. The mechanisms of action

of NPWT include macrodeformation (approximation of wound edges), microdeformation at the cellular level (stimulation of cell proliferation and granulation tissue formation), removal of inflammatory fluids, and stabilization of the wound environment, which promotes healing, particularly in exudative wounds and those with tissue loss [9].

In internal medicine practice, NPWT should be considered in patients with deep, exudative, chronic wounds or wounds following surgical debridement, especially when standard treatment fails to achieve the desired results. NPWT is used, among others, in stage III and IV pressure injuries, diabetic foot ulcers after adequate wound bed preparation, postoperative wounds with tissue defects, and traumatic wounds [9]. A prerequisite for safe implementation of NPWT is prior effective wound debridement, control of infection, and exclusion of dry necrosis or uncontrolled bleeding. There are also important contraindications to NPWT, including untreated osteomyelitis, the presence of non-demarcated necrosis, fistulas of unknown origin, malignancy within the wound, and active bleeding. In patients with coagulation disorders or those receiving anticoagulant therapy, NPWT should be applied with particular caution and under close clinical supervision [10]. From the perspective of the internist, proper patient selection for NPWT, monitoring treatment tolerance, and early identification of potential complications such as pain, bleeding, or signs of infection are essential. The internist also plays a significant role in coordinating multidisciplinary care, referring patients to wound care teams, surgeons, or long-term care specialists when NPWT requires continuation in outpatient or home-care settings [9].

Role of the Internist

The internist plays a pivotal role in the care of patients with chronic wounds, as in clinical practice they are often the first point of contact within the healthcare system [1],[2]. Their primary responsibilities include initial wound assessment, identification of wound etiology, and recognition of risk factors that impair healing, such as diabetes mellitus, chronic venous insufficiency, peripheral arterial disease, neuropathy, malnutrition, and coexisting inflammatory conditions [5], [6]. The internist initiates diagnostic work-up in accordance with

current standards by assessing limb perfusion (pulse examination, ankle–brachial index [ABI], toe–brachial index [TBI]), performing neurological evaluation, analyzing signs of infection, and referring the patient for imaging studies, including Doppler ultrasonography [13], [17],[18]. Concurrently, the internist is responsible for optimizing the management of comorbidities, including glycemic control, blood pressure regulation, treatment of dyslipidemia, and interventions related to nutritional status and body weight, as systemic factors significantly influence the wound healing process [1],[15]. In local wound management, the internist applies the principles of the TIME strategy, which encompass wound bed preparation, control of inflammation and microbial burden, maintenance of moisture balance, and assessment and support of epithelialization [7]. Their role also includes qualifying patients for advanced therapies (e.g., negative pressure wound therapy or larval therapy), monitoring treatment tolerance, and early detection of complications such as infection progression, pain, or bleeding. [9], [10], [11]. Patient and caregiver education regarding skin care, injury prevention, offloading principles, and the necessity of regular follow-up visits remains a crucial component of care [3], [5].

Furthermore, the internist acts as a coordinator of multidisciplinary care, referring patients to appropriate specialists (e.g., vascular surgeons, diabetologists, general surgeons, podiatrists), particularly in cases of ischemic wounds, deep infections, suspected bone involvement, or the need for surgical intervention [13],[21]. Holistic and coordinated approach—encompassing causal and local treatment, complication prevention, and evaluation of therapeutic outcomes—contributes to improved prognosis in patients with chronic wounds and reduces the risk of hospitalization and amputation [3], [5].

Conclusion

Chronic wounds represent a complex and heterogeneous clinical problem that requires an individualized diagnostic and therapeutic approach based on precise determination of wound etiology. Effective management depends on the simultaneous optimization of systemic factors, appropriate local treatment in accordance with the TIME strategy, and implementation of causal therapy. The internist plays a key role in early patient assessment, initiation of diagnostic

procedures, management of comorbidities, and coordination of multidisciplinary care. In selected cases, advanced modalities such as negative pressure wound therapy or larval therapy may provide valuable adjuncts to standard treatment. A holistic, evidence-based approach improves healing outcomes, limits complications, and reduces the risk of hospitalization and amputation in patients with chronic wounds.

Disclosure

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Conflicts of Interest

The authors declare no conflict of interest.

In preparing this work, the authors used ChatGPT for the purpose of improving translating into academic English language of the manuscript, ensuring clarity, consistency and adherence to scientific writing standard, text formating and verification of bibliographic style. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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