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WOUND CARE IN THE AGING POPULATION: CURRENT MANAGEMENT AND FUTURE DIRECTIONS

Karolina Lach *(Corresponding Author, Email: lach.karolina@outlook.com)*
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0000-0002-0529-1623

Anna Kułach
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0000-6476-8869

Wiktoria Modrzejewska
National Medical Institute of the Ministry of Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0006-9165-7404

Gabriela Krok
Henryk Klimontowicz Hospital, Gorlice, Poland
ORCID ID: 0009-0000-3969-9929

Katarzyna Siwiec
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0001-6411-9727

Maria Chmielewska
Mazovian Brodnowski Hospital, Warsaw, Poland
ORCID ID: 0009-0002-7702-8282

Weronika Basak
Military Institute of Medicine, Warsaw, Poland
ORCID ID: 0009-0007-1788-3259

Zofia Wcisło
Military Institute of Medicine, Warsaw, Poland
ORCID ID: 0009-0009-9058-4882

Łukasz Lamparski
National Medical Institute of the Ministry of Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0005-3143-3583

Patrycja Piekarska
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0007-0085-6617

ABSTRACT

As global demographics shift toward an aging population, wound management in the elderly has become a clinical challenge. Wound management in this demographic is uniquely complex, requiring an understanding of age-specific changes and the multisystemic nature of geriatric health. This article focuses on challenges in geriatric wound care, specifically on how the skin's physical structure changes over time, the diverse range of treatment methods currently available, and the often-overlooked impact of a patient's socioeconomic status on their recovery. One of the assessment tools being used nowadays is the Clinical Frailty Scale (CFS), which, by shifting focus from chronological age to a patient's functional independence and comorbidity load, can better quantify their risk of complications. Current research into novel therapies, particularly the use of specialized wound dressings, advanced drug therapies and the integration of artificial intelligence, shows promising ways of predicting complications and personalizing care for older adults. Effective wound management in the elderly requires a transition from traditional episodic care to a multidisciplinary, technology-enhanced approach.

KEYWORDS

Wound Management, Elderly, Personalized Medicine, Frailty, Dressings

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1. Introduction

Population dynamics indicate a significant shift toward an ageing population, with the number of older adults projected to rise from 1 billion to 2.1 billion in the following years [1]. Despite this collective growth, the chronological classification of the elderly remains regionally dependent. In most developed countries, 65 years is typically recognized as the standard benchmark, whereas in developing nations this line is often set at 60 years [2]. As the number of older adults rises, healthcare systems face new challenges. Treating an older demographic requires navigating a complex web of age-related comorbidities, particularly in specialized wound care, where diminished healing capacity necessitates increasingly sophisticated and patient-centered clinical interventions. Wound healing is a normal physiological response. However, in the elderly, this process is fundamentally undermined by the progressive deterioration of the skin's integrity. Starting around age 60, a progressive flattening of the skin layers severely compromises mechanical stability, predisposing the tissue to shear-induced injury and epidermal stripping. This structural fragility is compounded by impaired neurosensory function and disrupted barrier permeability. Consequently, the regenerative process in ageing skin is markedly protracted and fraught with clinical complications [2,3].

Wounds can present as either open or closed. Open wounds are marked by disrupted skin and exposed underlying tissues, whereas closed wounds involve injury to tissues beneath intact skin and may be acute or chronic in nature. Acute wounds result from sudden damage to the skin, happen in an instant, and typically heal within 8–12 weeks. In contrast, chronic wounds don't progress through the normal stages of healing and timeframe. Chronicity typically occurs during the inflammatory phase, when the body's protective response and initiation of repair are impaired [1,4]. The geriatric population is uniquely susceptible to chronic wounds due to multimorbidity and polypharmacy. Systemic conditions such as Type 2 diabetes and peripheral arterial disease (PAD) fundamentally impair vascular insufficiency, including arterial and venous compromise, creating a state of chronic hypoxia that prevents wounds from progressing past the inflammatory phase and the development of chronic wounds [4,5]. This review explores the challenges that clinicians face when treating wounds in elderly patients.

2. Methodology

Literature for this narrative review was identified through a systematic search of PubMed, Scopus, and Google Scholar. Primary search terms included *elderly wound management, skin changes in the elderly* and *innovative wound care technologies.* To ensure a high standard of clinical relevance, inclusion was restricted to international clinical guidelines, peer-reviewed systematic reviews, meta-analyses, and original research.

3. The Frailty Framework

While chronological age is a common metric, frailty scores have emerged as superior outcome measures in the geriatric population. Frailty is characterised by a systemic reduction in physiological reserve across multiple organ systems, leaving patients vulnerable to functional decline and biological response delays [6]. In the context of wound healing, frailty predicts a protracted healing timeline, as the patient lacks the reserve to overcome the metabolic and microbial hurdles inherent in chronic wounding [7]. Clinical tools like the Clinical Frailty Scale (CFS) allow clinicians to quantify this risk, focusing on independence and comorbidities rather than age alone. The CFS provides a global assessment based on activity levels and functional independence and is a predictor of survivability [8].

4. Changes in the elderly

Ageing exerts a profound influence on the skin's integrity, driven by a synergy of intrinsic and extrinsic factors. Intrinsic ageing, often referred to as chronological ageing, is an unavoidable biochemical degeneration driven primarily by genetic and hormonal factors [2,7]. At the cellular level, DNA damage and telomere shortening lead to a profound defect in cell proliferation. Although the thickness of the stratum corneum remains preserved, the underlying cellular and biochemical architecture undergoes significant degradation. Specifically, the corneocyte population is diminished, and lipid synthesis is impaired. This results in a disorganised lipid bilayer, fundamentally compromising the skin's epidermal barrier function and its ability to maintain homeostasis. Furthermore, the structural integrity of the dermal-epidermal junction (DEJ) is compromised as the basement membrane undergoes significant flattening by over one-third [3,9]. Within the basal layer, keratinocytes exhibit heterogeneity, characterised by increased variability in size and shape, leading to delayed epithelialisation [7,9]. Simultaneously, the dermis undergoes progressive atrophy, marked by a reduction in fibroblasts, while remaining cells exhibit increased cytoplasmic volume. Papillary fibroblasts within the upper dermis undergo a progressive functional shift, leading to a decrease in expression of extracellular matrix and transition toward an adipogenic phenotype. Aged fibroblasts upregulate matrix metalloproteinases (MMPs), which facilitate extensive collagen fragmentation, further destabilising the dermal architecture and manifesting as wrinkles and a reduction in elasticity. The atrophy of the subcutaneous dermal white adipose tissue (dWAT) takes place, as well [6,9]. A reduced interfacial surface area between the dermis and epidermis impairs functional surface area available for microvascular exchange, while restricting the diffusion of oxygen and essential nutrients to the avascular epidermis. This metabolic deficit predisposes the aged skin to dermo-epidermal separation [10]. Consequently, there is a coordinated decline in the skin's protective, regenerative and immune function [9]. Aged skin exhibits diminished vascularity and impaired hemodynamic flow, which collectively induce a state of chronic tissue hypoxia [3,9]. Visually, skin manifests as pale, dry, and finely-wrinkled [8]. The biochemical environment of ageing skin is further destabilised by fluctuating hormone levels. While the stress hormone cortisol sabotages the ECM by breaking down elastin, proteoglycans and collagen, a significant reduction in sex hormones (particularly estrogen) negatively impacts epidermal function and cell regeneration [2,8]. Concurrently, the skin's functional reserves are eroded by immunological shift toward immunosenescence [2,3,8]. This process is an age-related decrease in immune cell functionality, which drives a state of subclinical, chronic inflammation known as "inflammaging". Significant elevation of IL-1β, IL-6, IL-8, and TNF-α defines the chronic inflammatory landscape of ageing, directly correlating with increased mortality and a diminished physiological capacity to resolve injury [3].

In contrast, extrinsic ageing is driven by exogenous environmental, lifestyle, and mechanical factors. This pathway is characterised by deep wrinkles, rough texture, patchy hyperpigmentation, and a significant loss of elasticity. Ultraviolet radiation accounts for approximately 80% of facial skin ageing by triggering prolonged oxidative stress that aggressively accelerates collagen breakdown. The other lifestyle factors influencing ageing are smoking, alcohol, chronic exposure to pollutants, and poor nutritional intake [2,4,8]. For example, tobacco use induces peripheral vasoconstriction and hypoxia, thereby delaying wound closure and elevating the risk of dehiscence, necrosis, and infection. Similarly, ethanol consumption impairs innate immunity by attenuating neutrophil recruitment and phagocytic efficiency. Moreover, systemic glucocorticoid

therapy acts as a major inhibitor of wound resolution, suppressing the expression of hypoxia-inducible factor-1 (HIF-1), thereby predisposing patients to clinical complications [4]. Ultimately, this synergistic impact of environmental toxins and mechanical stressors, like repeated pressure or reduced motility, reduces tissue oxygenation and heightens the susceptibility to skin damage and injury [2,8].

5. The Biology of Normal vs. Aged Healing

5.1 Wound healing in normal skin

Wound healing is a dynamic, physiological response that relies on a complex network of signalling molecules and cellular recruitment. The process depends on the recruitment of platelets, neutrophils, macrophages, growth factors, and cytokines, alongside fibroblasts, keratinocytes, and endothelial cells [11]. This physiological repair is a highly coordinated process conventionally divided into a four-phase sequence: hemostasis (starts immediately after injury), inflammation (around 2–4 days), proliferation of mesenchymal cells (begins within 72 hours and lasts for about 14 days), and tissue remodelling(starting at around day 8, and persists for a year or longer). Hemostasis is initiated via microvascular vasoconstriction and activation of the coagulation cascade, culminating in the formation of a provisional fibrin matrix through platelet aggregation [12]. Such immediate, physiological mechanisms serve to stop blood loss, shielding compromised tissue from opportunistic pathogens [11]. Then, during the inflammatory phase, the environment transitions from a neutrophil-dominant to a macrophage-directed state, which acts as the primary orchestrator of the wound environment [3,12]. The recruitment of neutrophils and the polarisation of macrophages are further driven by reactive oxygen species (ROS) - byproducts of aerobic metabolism. The inflammatory cells gain access to the wound via two primary pathways: direct release through microhemorrhages and the targeted mechanism of transendothelial migration [11]. The replacement of the fibrin clot with vascularized granulation tissue marks the subsequent proliferative phase. During this stage, the establishment of vascularized extracellular matrix (ECM), driven by the reciprocal endothelial-fibroblast crosstalk, establishes the structural framework necessary for tissue stability. ECM serves as a potent catalyst for angiogenesis and is essential for restoring the delivery of oxygen and nutrients to the wound. The restoration of the epidermal barrier occurs via re-epithelialization of the dermal bridge, concurrently with wound contraction. Remodelling is initiated by the programmed apoptosis of myofibroblasts and macrophages, transitioning, within months, the Type III collagen matrix into a more permanent, more disorganised Type I collagen network characteristic of mature scarring [13]. However, the successful wound resolution is ultimately governed by four critical variables: the infective load, the virulence of colonising pathogens, the local wound microenvironment, and the patient's physiological reserve [14]. Furthermore, the accumulation of reactive oxygen species (ROS) drives oxidative damage to cellular components, leading to diminished epidermal turnover and compromised tissue function.

5.2 Wound healing in aged skin

In aged skin, the phases of wound healing become fundamentally dysregulated. While the process normally begins with platelet activation and a rapid coagulation cascade, the inflammatory phase in the elderly often devolves into a protracted, self-reinforcing cycle of destruction. Paradoxically, although increased platelet adhesion in aged tissue triggers a surplus of pro-inflammatory cytokines such as PDGF, TGF-β, and TGF-α, the cellular response is severely hindered. Specifically, the recruitment of neutrophils, macrophages, and T-lymphocytes is compromised, and monocyte infiltration is impaired. This leads to a depletion of mature macrophages, which in turn reduces granulation tissue formation and angiogenesis while impairing the synthesis of collagen and essential growth factors. Furthermore, the transition to the proliferative phase is sabotaged by a failure in macrophage phenotype switching. In aged tissue, macrophages often fail to undergo the necessary repolarisation from pro-inflammatory IL-12 production toward the anti-inflammatory IL-10 signalling required to terminate the inflammatory cascade and initiate repair. Instead, the persistent activation of the COX pathway leads to an overproduction of PGE2, which perversely degrades adjacent healthy cells and exerts a direct inhibitory effect on fibroblasts. Consequently, the essential processes of reepithelialisation and remodelling are not merely delayed but are fundamentally undermined by a landscape of chronic inflammation and impaired cellular signalling [2].

Although ROS are essential secondary messengers for angiogenesis and cell motility at low concentrations, their overproduction, exacerbated by persistent hypoxia, induces extensive tissue injury and sustains the inflammatory arrest characteristic of chronic wounds [3]. The chronic oxidative environment triggers a cascade of lipid peroxidation, protein degradation, and DNA damage, ultimately driving the wound toward cellular apoptosis and senescence. This metabolic breakdown significantly stalls the proliferative phase,

a delay further exacerbated by the age-related deterioration of the microvasculature. As blood vessel growth and function falter, the resulting hypoperfusion creates a nutrient deficiency at the injury site, limiting the delivery of the inflammatory cells and chemical mediators required for the healing process. Furthermore, this circulatory failure shifts the wound into a state of prolonged hypoxia. While temporary, acute hypoxia is a necessary trigger for angiogenesis and cell migration, the chronic oxygen deficit found in aged tissue induces necrosis and halts the healing [2].

6. Challenges in the Elderly

The factors that impair wound resolution are significantly more prevalent in the elderly population [15]. Nutritional status is a dual determinant of health, as both macronutrients and micronutrients are essential for inflammation, proliferation, and remodeling [7,16]. In the elderly, the presence of a chronic wound or pressure injury triggers a systemic catabolic state, characterized by a surge in catabolic hormones and a simultaneous downregulation of anabolic hormones. To satisfy the resulting metabolic stress, the body initiates gluconeogenesis, breaking down its own fat and protein stores to meet energy demands. Without nutritional intervention, this process leads to a rapid loss of metabolic cell mass and physical frailty, fundamentally delaying wound closure [16]. This vulnerability is exacerbated by the “anorexia of ageing”, described by Morley and Silver [17], where reduced appetite and physical activity lead to undernourishment. Clinically, malnutrition functions as both a precursor to and a consequence of systemic decline, serving as a primary driver of sarcopenia, osteoporosis, and impaired wound healing by reducing the tensile strength of incisions and halting collagen synthesis [7,16]. Comprehensive reviews focusing on geriatric trauma and wound care emphasize a critical need for nutritional screening in older populations. These studies advocate for the implementation of high-energy, high-protein dietary interventions for at-risk patients to meet the elevated metabolic demands of tissue regeneration [7]. Evidence from a randomized controlled study by Blass et al. [18] indicates that antioxidant and glutamine supplementation accelerates healing in trauma patients by mitigating oxidative stress and meeting increased cellular demands. Their findings suggest that chronic inflammation leads to hypoalbuminemia, which disrupts the transport of essential micronutrients to the wound. Addressing this transport failure through nutritional supplementation is therefore critical to overcoming the oxidative burden that characterizes delayed healing in geriatric trauma. Optimal tissue repair necessitates specific micronutrients, like Vitamin C, which is essential for the procollagen triple helix formation. Vitamin A is necessary for healing and can reverse impairments caused by steroids or radiation. Furthermore, minerals such as Zinc and Copper act as indispensable cofactors for repair, and the amino acid Arginine has been shown to uniquely augment the healing process [15].

The pre-existing conditions, like diabetes mellitus (DM) or heart disease, significantly impact overall well-being and serve as a contributory factor to traumatic injury [7]. This impaired healing trajectory in DM is driven by macrovascular atherosclerosis and microvascular basement membrane thickening, which impede the delivery of oxygen and nutrients. Furthermore, peripheral neuropathy eliminates "protective sensation," allowing minor mechanical or thermal traumas to escalate into full-thickness ulcers unnoticed. This is compounded by immune suppression, as hyperglycemia compromises leukocyte function and thickened capillaries hinder immune cell migration, allowing infections to spread rapidly [15]. Heart diseases, like Peripheral Artery Disease (PAD) is a critical vascular barrier to wound healing, often presenting as intermittent claudication or remaining asymptomatic until significant tissue loss occurs. This disease disrupts the local wound environment through both macrovascular atherosclerosis and microvascular dysfunction. At the cellular level, PAD induces a state of "mitogenic paralysis". Fibroblasts in ischemic tissue exhibit decreased mobility and a blunted response to essential growth factors, which prevents the formation of healthy granulation tissue. This failure is compounded by reduced mobility, which subjects fragile tissues to prolonged pressure and shear forces, further depleting oxygen tension and inducing necrosis [19].

Some chronic wounds are more ubiquitous in the older population [15]. This prevalent clinical spectrum is dominated by pressure injuries, skin tears, and surgical wounds. Furthermore, age-related vascular and neurological decline frequently manifests as venous, arterial, and neuropathic ulcers, alongside atypical presentations of inflammatory or neoplastic origin [8]. Skin tears represent a prevalent acute traumatic wound, typically resulting from friction, shear, or blunt trauma [1]. These traumatic wounds don’t extend beyond the subcutaneous layer and are categorized as uncomplicated if they achieve full closure within four weeks or complicated if healing is delayed by infections or comorbidities. Because they expose the sensory nerve endings of the dermis, skin tears are significantly painful and carry a high risk of becoming chronic if the initial trauma is mismanaged [20]. Moreover, surgical site infections remain a critical postoperative complication,

necessitating dressing selections based on whether a wound is classified as clean, clean-contaminated, contaminated, or dirty. Among chronic conditions, vascular ulcers of the lower limb are the most frequent [1]. While venous ulcers result from fluid congestion and high pressure, arterial ulcers arise from oxygen starvation. The venous ulcers drive oedema and chronic inflammation, typically manifesting above the medial malleolus with characteristic hyperpigmentation. The arterial ulcers are smaller, rounded, and located over bony prominences with shiny skin. Additionally, pressure ulcers are often caused by prolonged pressure or friction and remain a significant burden. The elevated pressure triggers tissue ischemia. These wounds typically concentrate over bony prominences like the sacrum and heels, where age-related muscle loss and moisture-induced shear further compromise skin integrity [1,15].

While the patient and family experience was identified by Dhar et al. [21] through three critical challenges: "expecting and managing the pain", "receiving expert advice and reflecting on previous care", and "managing the cost of care", the specific consequences of these hurdles for individuals who self-treat were further explored by Kapp et al. [22]. Kapp's investigation revealed that individuals who self-treat their wounds face overwhelming challenges, including persistent pain and forced physical inactivity, which profoundly disrupts the physical, emotional, and socioeconomic domains of a patient's Quality of Life (QoL). Furthermore, the economic burden is often disproportionate, consuming most of the patient's income. Moreover, the time commitment required for repeated clinical visits creates a pervasive lifestyle disruption. As identified by Michael et al. [23], critical systemic barriers to wound care include patient educational deficits regarding home care and significant delays in clinical appointments due to staffing and material shortages.

7. Current Management & Future Directions

Wound management in the geriatric population is uniquely challenging, stemming from the intersection of biological senescence and systemic health complexities. The current management prioritizes a holistic, person-centered approach to address age-related physiological changes such as reduced skin elasticity and impaired cellular regeneration [24]. Modern protocols utilize the TIME framework (Tissue, Infection, Moisture, and Edge) to guide wound bed preparation, with a growing emphasis on preventative skincare [25]. Optimized wound resolution has driven substantial collaboration between researchers and clinicians, looking to enhance healing. Traditional wound management is centered on maintaining an isothermic and moist microenvironment while mitigating microbial control through natural and synthetic dressings [26]. The ideal therapeutic dressing must be biocompatible and flexible, highly absorbent, providing a physical defensive barrier while facilitating optimal gas exchange [9, 27].

7.1 Standard dressing

Standard wound dressings are engineered to facilitate exudate, reduce bacterial load, and promote tissue regeneration while remaining cost-effective and non-toxic. Povidone-iodine is frequently used for cleansing and preoperative preparation, whereas enzymatic products containing collagenases or proteases facilitate debridement by degrading necrotic and devitalized tissue. To optimise healing, absorbing dressings maintain a moist wound environment. In contrast, silver dressings provide broad-spectrum antimicrobial activity through the sustained release of ions that disrupt microbial processes, especially in chronic wounds. Similarly, medical-grade honey (MGH) dressings harness natural antimicrobial properties to promote autolytic debridement and enhance re-epithelialization. Beyond these traditional dressings, self-healing injectable hydrogels represent another advanced treatment modality [1].

7.2 Advanced dressing

The development of advanced dressings and drug-delivery systems has provided clinicians with new tools to address the complex biochemical barriers that stall healing in the geriatric population [13]. The therapeutic landscape has been transformed by the emergence of nanofiber-based dressings, which are an alternative to traditional wound care. Unlike conventional materials, nanofibers provide excellent gas exchange and biomimetic hydrophilicity and become a platform for the controlled delivery of bioactive substances and antimicrobial agents. As established by Li et al. [28], by providing enhanced mechanical support and a targeted biochemical release, nanofibers stabilize the wound and mitigate the pathological effects of chronic inflammation of geriatric wounds.

The modern clinical dressings have expanded to include specialised ones such as hydrofibers, sponges, bioactive hydrogels, hydrocolloids, collagen and fucoidan-based scaffolds. These dressings are classified into antiseptic, ionic/nanocrystalline silver, and antibiotic. Antiseptic dressings, specifically, are engineered for the

controlled release of microbicidal agents at concentrations that effectively eradicate diverse microorganisms while remaining below the threshold of cytotoxicity for senescent host tissue. Smart hydrogel wound dressings represent the next generation of care, shifting the focus from simple protection to active, responsive treatment. Categorised into thermo-sensitive, pH-sensitive, and light-responsive, these materials adapt to the specific conditions of the wound. By leveraging the dynamic microenvironment of the wound, such as the alkaline pH shift typical of chronic infection or the localised hyperthermia of inflammation, these hydrogels can provide on-demand pharmacological release and real-time structural adaptation, ensuring that therapeutic delivery is both precise and efficient [27].

7.2.1 Thermo-sensitive hydrogels

Thermo-sensitive hydrogels represent a class of biomaterials defined by their capacity for switching in response to temperature change. These products offer a potential clinical advantage in managing irregular and deep-tissue wounds [29]. By maintaining a low-viscosity fluid state at ambient temperatures, they allow for the precise infiltration targated area. Upon contact with physiological heat, the material undergoes an in-situ sol-to-gel transition, establishing a stable three-dimensional (3D) bioactive matrix. Specifically, polypeptide-based thermogels provide a highly cytocompatible environment that mimics the native extracellular matrix (ECM). Their simplistic encapsulation protocols facilitate the integration of therapeutic cells and growth factors, providing the structural and signalling integrity necessary to catalyse tissue regeneration in compromised host environments [30]. Synergistic therapies combining metallic ions and pharmacological agents may be used for treating chronic, non-healing wounds, often associated with diabetes [31]. Pan et al. [32] created a sprayable photothermal hydrogel, which undergoes rapid NIR-II laser-triggered gelation, providing hemostatic, antibacterial, and photothermal effects, specifically engineered to dismantle the resilient Gram-negative biofilms that characterise geriatric wound failure.

7.2.2 pH-sensitive hydrogels

The pH of the wound serves as a critical biomarker for wound status. While healthy skin maintains an acidic pH 4.5–6.5, wounds typically alkalize to pH 7.4 or higher, signalling persistent infection or stalled healing. Red cabbage extract/methacrylate chitosan hydrogels have emerged as diagnostic and therapeutic platforms. Studies by Reshimi et al. [33] demonstrate that nano-chitosan-enriched poly(ε-caprolactone) membranes can achieve the targeted, pH-dependent release of therapeutic payloads like curcumin. Similarly, Ren et al. [35] developed a hydrogel with tannic acid and keratin-graphene oxide quantum dot, which exhibits sustained release of pharmaceutics, effectively neutralising pathogens like E. coli and S. aureus. Research by De Piano et al. [36] suggests that physically combining alginate and Carbopol yields hydrogel patches that make them capable of modulating swelling, erosion, and drug release in a pH-dependent manner. Similarly, Singh et al. [37] demonstrated that the graft copolymerization of locust bean gum with acrylamide and acrylic acid creates effective drug delivery matrices, marking a significant advancement in wound care technology.

7.2.3 Other types of smart dressings

Colourimetric humidity indicators represent an integrated approach to wound monitoring, utilising moisture-sensitive dyes that change colour when exudate levels exceed a specific threshold. More advanced technological methods incorporate impedance or capacitance sensors to exploit the conductivity of ion-rich wound fluid. Specifically, smart dressings may feature conductive tracks printed on transparent films where exudate bridges the circuit to facilitate current flow. One device - WoundSense employs an impedance-based sensor pad beneath a dressing, allowing for the measurement of saturation levels without requiring dressing removal. Flexible capacitive sensors serve as an alternative monitoring tool, utilising parallel-plate technology to measure shifts in capacitance relative to the dielectric constant of the dressing medium. As wound fluid displaces air within the dressing, the capacitance increases proportionally. These flexible patches can be integrated into foam dressings and scanned to quantify fluid absorption through capacitance changes without disturbing the wound site. Pressure-sensing dressings incorporate flexible sensors that map pressure distribution across wounds, making them highly effective for the management of pressure ulcers and diabetic foot complications. However, their commercial availability remains limited. In chronic wounds, natural electrical signalling is frequently diminished or disoriented, which stalls the healing process. Electrical stimulation (ES), typically employing low-intensity currents, is used to combat infection and modulate inflammation. Electroactive dressings, specifically conductive hydrogel dressings enriched with carbon nanotubes, graphene, metal nanoparticles, or conductive polymers, provide a moist environment [34]. A

significant advancement in this field is the self-powered microneedle platform introduced by Li et al. [38], which was specifically engineered to treat infected diabetic wounds through integrated electrochemical interactions. The intelligent wound dressings integrate biosensors to detect biological or physical parameters, often featuring connectivity for data transmission. These systems essentially compact microcontroller to collect sensor data and communicate it wirelessly via Bluetooth or NFC to a smartphone [34]. For instance, a next-generation smart dressing incorporates sensors for pH, temperature, uric acid and strain gauge, all connected to a miniature Bluetooth module at the bandage's edge [39].

Self-healing injectable hydrogels offer another promising therapeutic pathway, integrating antibacterial, antioxidant, and electrically conductive properties to treat wound infections and facilitate tissue regeneration [27]. By enabling the controlled release of bioactive substances and expediting wound closure, they provide targeted drug delivery. Their rapid self-healing ability allows for precise injection into patient-specific tissue defects, where they maintain structural integrity without leakage. When paired with noninvasive imaging, these hydrogels provide a robust framework for personalised and highly specialised interventions [41].

7.2.4 Pharmacological treatment

Pharmacological modulation of the wound microenvironment via antimicrobial products addresses the persistent bioburden and biofilm formation that often stall the healing. By utilizing agents such as ionic silver, nanocrystalline silver, and cadexomer iodine, clinicians can mitigate microbial bio-burden. Historically, the application of silver compounds represented a milestone in acute wound management, significantly reducing mortality associated with wound-induced sepsis. While ionic silver provides a rapid antimicrobial burst, nanocrystalline silver offers a more sustained release of active silver ions, which is particularly effective against antibiotic-resistant strains. Furthermore, cadexomer iodine has emerged as a specialised modality for chronic wounds, specifically engineered to penetrate the protective polymicrobial biofilm matrix.

While systemic and topical antibiotics remain essential in geriatric wound care, their historical overuse, particularly in non-infected or colonised wounds, has significantly contributed to the emerging global antibiotic resistance crisis. In response, the clinicians have shifted toward the use of broad-spectrum antiseptics. Modern guidelines increasingly rely on agents such as Octenidine hydrochloride (OCT), Polyhexamethylene biguanide (PHMB), Povidone-iodine (PVP-I), and Sodium hypochlorite (NaOCl). Unlike traditional antibiotics, these antiseptics exert multi-target antimicrobial effects with high bactericidal potency and significant anti-biofilm activity, making them indispensable for managing the polymicrobial environments [27].

Antimicrobial Peptides (AMPs) represent an emerging class of therapeutics for managing traumatic infections. These peptides exert their microbicidal effects through electrostatic interactions with negatively charged microbial membranes, leading to pore formation, loss of cytoplasmic integrity, and the inhibition of essential cell-wall and protein synthesis [42]. This multi-target mechanism effectively suppresses microbial proliferation while minimising the likelihood of developing cross-resistance. Although thousands of AMPs have been identified, only a few are currently available for clinical use [24]. Several peptides and glycopeptides are currently utilised in clinical practice. Bacitracin remains a standard for topical management of Gram-positive pathogens, including Staphylococcus, Streptococcus, and Clostridium species. For more systemic or deep-seated infections, Daptomycin is widely employed for Staphylococcus and Enterococcus species, while Teicoplanin is used in Europe, Asia and South America for Methicillin-resistant Staphylococcus aureus (MRSA). The rise of Vancomycin-resistant strains has further generated the use of Dalbavancin, Oritavancin, and Telavancin. Additionally, agents like Gramicidin are effective against a broad spectrum of Gram-positive and select Gram-negative organisms and frequently incorporated into multi-agent topical formulations. The continuous evolution of microbial resistance ensures that many novel candidates remain under active investigation in clinical trials [43].

Biological therapies accelerate the repair of complex wounds by fostering microenvironments that stimulate stromal and endothelial cell proliferation, thereby facilitating the formation of essential new vascular networks. As explored by Mullin et al. [44], growth factor (GF) therapies target to overcome the healing of chronic wounds by delivering essential signalling proteins like PDGF, VEGF, and FGF. However, the clinical efficacy is often compromised by the hostile, protease-rich wound microenvironment, which rapidly degrades these molecules before they can take effect. Currently, rPDGF-BB (Regranex) remains the only FDA-approved growth factor treatment specifically for lower-extremity diabetic neuropathic ulcers [13]. To solve this, recent advancements have shifted toward GF gene therapies, which utilise viral or non-viral vectors to enable sustained, localised production of growth factors directly by the host cells. By integrating these genetic

instructions with protective biomaterial scaffolds, researchers are developing a more durable delivery system that bypasses the limitations of short half-lives and frequent dosing [44].

Beyond the delivery of isolated signalling proteins, a more comprehensive regenerative strategy was identified through the application of stem cell therapies. Mesenchymal Stem Cells (MSCs), which are derived from bone marrow, adipose tissue, and the umbilical cord, act as a potent intervention for accelerating the healing of chronic wounds. These cells function primarily through paracrine signalling and immunomodulation, releasing essential growth factors that stimulate angiogenesis, promote collagen deposition, and resolve chronic inflammation. In a systematic review, Farabi et al. [45] conclude that stem cell applications significantly improve wound closure rates and reduce ulcer size in complex cases like diabetic foot ulcers and venous insufficiency, although it still requires standardised protocols for delivery and dosage.

Parallel to these biological approaches, small-molecule therapies offer a more stable and cost-effective alternative, with simplified regulatory pathways. By modulating key signalling axes, these agents stimulate neovascularisation and tissue regeneration while resisting the rapid enzymatic degradation common in protein-based treatments. RNA interference (RNAi) therapies offer a highly selective method for silencing gene expression at the post-transcriptional level. By targeting specific mRNA molecules for destruction, RNAi can modulate pathways, such as the TGF-β/Smad2 axis for scar reduction or the suppression of Connective Tissue Growth Factor (CTGF), that are often difficult to regulate via traditional proteins or small molecules [13]. Recent advancements by Huang et al. [46] highlight the potential of engineered miR-31 exosomes as a specialised delivery system designed to overcome the inherent fragility of genetic material. By utilising these highly biocompatible exosomes to transport microRNA-31 directly into the wound bed, this therapy successfully drives angiogenesis and re-epithelialization in diabetic ulcers. This shift toward genetic and exosomal modulation opens the door for the development of precision therapeutics that can fundamentally change the management of chronic wounds resistant to traditional pharmacological interventions.

While pharmacological treatments represent a critical aspect of care, the immediate management of complex wounds often necessitates more direct, mechanical intervention. Surgical debridement remains the gold standard for managing wounds, effectively reducing microbial diversity and eradicating necrotic debris, apoptotic cells, and resilient biofilms. By clearing this 'biological load,' debridement restores the microenvironment required for epithelialization and optimises the local vascular supply. However, failure to address high microbial loads can lead to progressive tissue deterioration, osteomyelitis, and life-threatening sepsis. When conservative management fails to mitigate deep-tissue infection involving the musculature or adipose layers, amputation becomes a clinical necessity [27].

Looking ahead, the future of geriatric wound care is shifting toward a multimodal and personalised paradigm that seamlessly integrates the molecular, cellular, and surgical strategies. The development of technologies points toward the design of "smart bandages", integrating real-time monitoring of pH, temperature, and infection markers with on-demand drug delivery and electro-mechanical stimulation. As biosensor-equipped dressings generate data, Artificial Intelligence will be pivotal for interpretation. By processing complex healing trajectories in real-time, AI-driven algorithms can predict complications before they manifest clinically, moving the field toward a highly personalised model of care that optimises the reparative environment for each specific patient [34].

8. Discussion

The management of wounds in the elderly is complicated by altered skin, slowed repair and frequent comorbid conditions that reduce healing capacity and increase adverse outcomes. A significant challenge is the inherent fragility of aged skin. The flattening of the dermal-epidermal junction drastically reduces the skin’s ability to resist shear. Essentially, routine movements of the patient can lead to a drastic outcome with tissue separation. This mechanical vulnerability is compounded by microvascular dysfunction, where necessary nutrients can’t be delivered to the site of the injury and the repair process is delayed. Malnutrition is markedly prevalent in this demographic, creating a significant challenge.

Moreover, there is a transition from a neutrophil-dominant acute phase to a macrophage-directed repair phase in a healthy individual. While in the frail population, this transition frequently fails, leading to the delay of reepithelialisation and remodeling. The environment of the wound remains trapped in a pro-inflammatory cycle, where neutrophils continue to degrade the newly forming tissue matrix. To address this, the usage of the Clinical Frailty Scale (CSF) may become a validated assessment tool. Assessing a patient’s functional baseline and comorbidity load provides a more accurate “biological age”, allowing clinicians to anticipate complications before they occur.

The management of patients is complicated by the multifactorial complexity of preexisting conditions. Comorbidities such as diabetes mellitus and cardiovascular disease do not just exist alongside a wound. They actively impair its repair. Diabetes, for instance, severely impairs peripheral circulation and immune response, while heart disease limits oxygen delivery to distal tissues. Another challenge in this population is the prevalence of pressure ulcers. The mobility in older adults is often restricted by frailty or cognitive decline, leading to prolonged tissue compression.

Furthermore, as determined by Dhar et al. and Kapp et al., managing these wounds is not just a structural endeavor but a quality-of-life crisis. Chronic pain and limited physical activity create a vicious cycle that hinders recovery.

Wound management is moving toward a technology-enhanced, multidisciplinary approach in the elderly. The development of multifunctional hydrogels represents a major shift from passive to active care. These matrices, capable of sensing pH, mechanical strain, electrical current, or localized heat, act as surveillance systems for wounds. When paired with Artificial Intelligence, these data points allow for a level of personalization, making them an important tool for future management. In addition to physical material, the rapid evolution of pharmacology is fundamentally expanding our possibilities of treatment. The strategy of treatment is now moving far beyond traditional antimicrobial therapy, which is particularly critical given the rising threat of multidrug-resistant organisms. Antimicrobial Peptides have emerged as a powerful alternative, offering a robust defense against resistant species by directly disrupting microbial membranes. Wound care increasingly involves biological therapies that deliver essential signaling proteins to reactivate the stalled healing process. For instance, gene therapies allow for the localized production of growth factors directly within the host cells, supplying the wound with the proteins needed for repair.

In addition, the application of Mesenchymal Stem Cells has shown remarkable efficacy, particularly in treating diabetic foot ulcers. These cells release growth factors that stimulate angiogenesis, promote collagen deposition, and actively resolve chronic inflammation. The treatment options are complemented by RNA interference, which can drive re-epithelialization in otherwise non-responsive diabetic ulcers.

Ultimately, these advancements represent a shift from merely protecting the wound to actively re-engineering the biological environment. By integrating these advanced pharmacological tools with monitoring systems, we can finally try to overcome the systemic barriers that have historically made geriatric wound healing so difficult to manage.

9. Conclusions

The management of geriatric wounds exceeds the boundaries of local tissue repair, representing a complex intersection of medical treatment and psychosocial reality. A non-healing wound is rarely an isolated pathology, but rather a localised manifestation of systemic frailty and physiological exhaustion. Healing can’t occur and the clinical success of even the most sophisticated molecular therapy is ultimately blocked by patients’ underlying conditions. To treat the wounds effectively, it is necessary to stabilise the organism first, as no regenerative signalling pathway can function if the host lacks the vascular delivery and metabolic reserves to support new tissue. Beyond the biological complexities, the fragility of geriatric independence introduces a profound human challenge. Social isolation and cognitive decline often result in neglect of care, ranging from complex medication to the maintenance of adequate caloric intake. This reality shifts the burden of care from the clinic to home, demanding a multidisciplinary strategy. Consequently, the future of wound care must not only be technologically advanced but also personalized, bridging the gap between sophisticated interventions and the reality of the aging person.

Author Contributions

Conceptualization: Karolina Lach

Methodology: Patrycja Piekarska, Łukasz Lamparski

Formal analysis: Zofia Wcisło, Weronika Basak

Writing-original draft preparation: Anna Kułach, Maria Chmielewska

Writing-review and editing: Wiktoria Modrzejewska, Gabriela Krok, Katarzyna Siwiec, Karolina Lach, Anna Kułach, Łukasz Lamparski

Supervision: Karolina Lach

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