Breaking the cycle of hard-to-heal wounds: the role of autologous whole blood clot therapy

Abstract: Hard-to-heal (chronic) wounds pose significant challenges in healthcare due to their prolonged inflammatory phase and impaired healing processes. These wounds, commonly associated with diabetes, vascular insufficiency or impaired mobility, fail to progress through the orderly phases of haemostasis, inflammation, proliferation and remodelling. Persistent inflammation, driven by M1 macrophages, neutrophils and proteases, disrupts extracellular matrix (ECM) reconstruction and degrades growth factors and cytokines, halting healing. Conventional treatments often fail to address the underlying pathophysiology, prompting interest in advanced autologous therapies. Autologous whole blood clot (AWBC), an innovative autologous therapy, activates the patient's blood to create a biological scaffold rich in growth factors and cytokines. This scaffold promotes wound healing by supporting ECM reconstruction, causing inflammatory M1 macrophages to transition to regenerative M2 macrophages, and enhancing neutrophil clearance. The objective of this review was to summarise the current clinical evidence and elucidate the proposed biological mechanisms by which AWBC promotes healing in hard-to-heal wounds. Clinical studies have demonstrated AWBC's efficacy across multiple wound types, including diabetic foot ulcers, pressure ulcers and surgical wounds. With a robust safety profile, minimal risk of adverse reactions and reported efficacy, AWBC offers a transformative solution for hard-to-heal wound management, addressing unmet clinical needs and improving patient outcomes.

Declaration of interest: RJS is a consultant at RedDress Medical, FL, US, the company that manufactures the device which creates the AWBC reported in this article.

autologous whole blood clot • chronic wounds • extracellular matrix • hard-to-heal wounds • therapeutic • wound • wound care • wound dressing • wound healing

Hard-to-heal (chronic) wounds are a concern in healthcare, especially among individuals with diabetes, vascular insufficiency or impaired mobility.[1] While the normal wound healing process goes through the four healing phases of haemostasis, inflammation, proliferation and remodelling, hard-to-heal wounds fail to follow a normal, orderly and timely sequence of events that lead to wound healing.[2] A key contributor to the chronicity of a wound is a prolonged, stagnant inflammatory phase, which is unable to transition to the remodelling phase. The constant inflammation consists of proinflammatory macrophages,[3,4] neutrophils[5] and proteases,[6] all linked to wound deterioration and ulcer severity.

When a wound first forms, neutrophils infiltrate the wound bed within 24 hours, followed by proinflammatory M1 macrophages, both essential to keep the wound clear from contaminations of bacteria, fungi and yeast.[7] However, in hard-to-heal wounds, macrophages often remain in the M1 state, unable to transition to M2 due to persistent inflammation and factors such as excess iron accumulation, often derived from haemoglobin breakdown in hard-to-heal wounds, which further impairs healing.[4]

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Elevated iron levels promote oxidative stress and the production of reactive oxygen species, which sustain proinflammatory signalling pathways that maintain macrophages in the M1 phenotype.[5] This persistent M1 state inhibits the normal transition to the anti-inflammatory M2 phenotype, thereby prolonging inflammation and impairing effective tissue repair.[8] Neutrophils, on the other hand, are crucial in the early defence against infection; however, their accumulation and delayed clearance in hard-to-heal wounds can exacerbate inflammation, creating a loop from which the wound is unable to exit. These dysregulated processes perpetuate the hard-to-heal wound state.

This chronic condition also affects the reconstruction of the extracellular matrix (ECM), which is the largest structural component of the skin, and plays a critical role in wound healing by facilitating dynamic communication between cells and their microenvironment through a process known as dynamic reciprocity.[9,10] Composed of proteins, such as collagen, elastin and proteoglycans, the ECM provides both structural support and regulatory signals essential for tissue repair. Hard-to-heal wounds disrupt this process, where excessive matrix metalloproteases and neutrophil elastase degrade the ECM and cause the degradation of growth factors (e.g., vascular endothelial growth factor (VEGF),[11] transforming growth factor-beta (TGF-β)[12]) and cytokines (e.g., tumour necrosis factor-alpha (TNF-α)[13]), halting the healing process.[14,15] Both VEGF and TGF-β play crucial roles in the wound

healing process through their regulation of cellular responses and tissue repair mechanisms. TGF-β is broadly involved in coordinating wound healing by modulating immune cell infiltration, ECM production and fibrosis, and it also influences multiple phases of wound repair, from inflammation to remodelling.[16] VEGF is vital for angiogenesis during wound healing, promoting the growth of new blood vessels to supply nutrients and immune cells to the wound site.[17] Conventional wound treatments often fail to address the underlying pathophysiology of hard-to-heal wounds and require the intervention of advanced wound care technology.[18]

Autologous therapies, which use the patient's own biological materials, are gaining attention due to their ability to promote healing without introducing foreign agents or synthetic materials. Autologous whole blood clot (AWBC) therapy is designed to provide a scaffold rich in cytokines and growth factors, thus enabling a conducive environment for tissue regeneration and wound closure.[7]

Despite increasing clinical data and encouraging outcomes, the biological mechanisms underlying AWBC therapy have not been comprehensively synthesised in the context of hard-to-heal wound pathophysiology. The purpose of this review was to integrate current knowledge on the mechanistic and clinical evidence of AWBC as a treatment in hard-to-heal wounds. In particular, this review focused on the roles of platelets, macrophage plasticity, neutrophil clearance, and the provisional ECM scaffold in converting hard-to-heal wounds into an acute, pro-regenerative healing environment.

Literature review and search strategy

This narrative review was conducted by searching PubMed for publications up to September 2024, using the search terms: 'autologous whole blood clot', 'AWBC', 'ActiGraft', 'wound healing' and 'chronic wounds'. Included studies comprised randomised controlled trials (RCTs), prospective and retrospective observational studies, registry data, pilot studies, and mechanistic reviews reporting on AWBC therapy or its biological components in the context of wound healing. Studies were selected based on relevance to the therapeutic mechanisms or clinical outcomes of AWBC.

Autologous whole blood clot as a treatment in wound healing

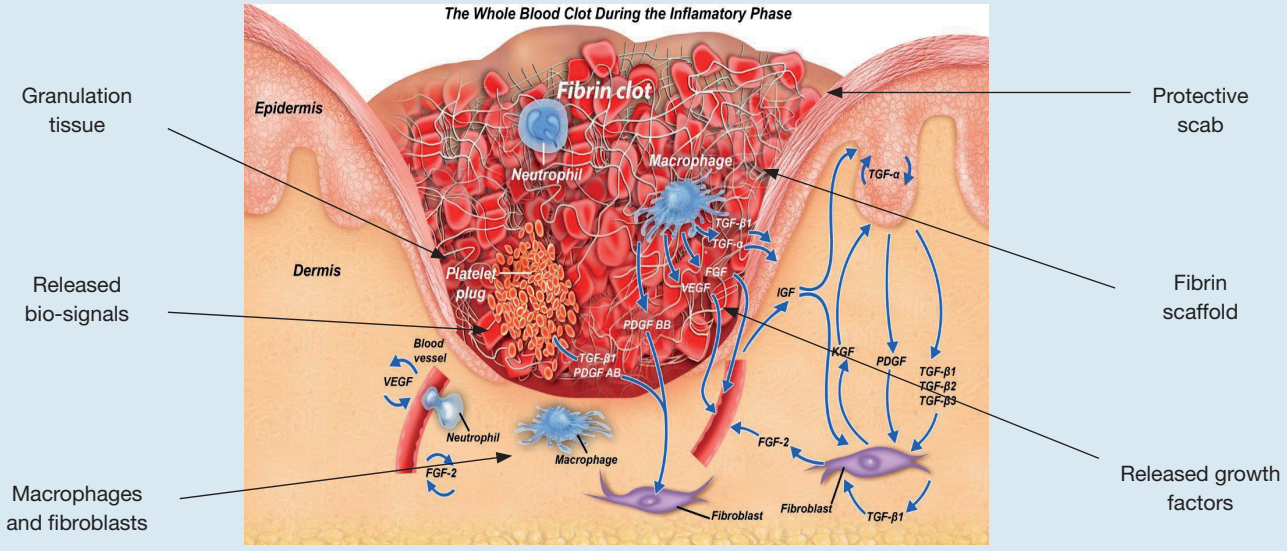
AWBC is a biological material derived from a patient's own blood, processed into a clot that acts as a therapeutic scaffold for wound healing. This clot contains all the native blood components, which collectively aid in the wound repair process.[7] To obtain AWBC, a small volume of blood (typically 15ml) is drawn from the patient. This blood is then processed in a proprietary device (ActiGraft; RedDress Medical Ltd, Israel, the only commercially used device), designed to induce coagulation, resulting in a stable clot. Once formed, the clot is applied topically to the wound site.[7] The AWBC clot functions as a dynamic ECM scaffold that regulates cellular activity within the wound environment.[7] It releases platelets, growth factors and cytokines essential for the various phases of wound healing. Platelets play a central role by mediating haemostasis, thrombosis and vascular patency, while also contributing to immune responses and inflammation.[10,19,20]

Upon activation, platelets release growth factors, such as platelet-derived growth factor (PDGF) and epidermal growth factor (EGF), which promote fibroblast proliferation and ECM synthesis during the proliferative phase of healing.[10,21,22] VEGF, which supports re-epithelialisation, cytoprotection and angiogenesis, is also secreted by the activated platelets, making it another important growth factor in the wound healing process.[10] In addition to growth factor release, platelets participate in immune defence by releasing proinflammatory mediators, facilitating neutrophil extracellular trap formation (NETosis), and promoting leukocyte migration, all of which enhance bacterial clearance.[10,19] The clot's fibrin matrix acts as a mechanical barrier against bacterial infiltration, reducing infection risk and preventing haemorrhage.[7] This matrix facilitates leukocyte adhesion and supports endothelial cell migration, and promotes fibroblast proliferation and angiogenesis (Fig 1).[22] Furthermore, the AWBC clot modulates macrophage phenotypes, promoting the transition from proinflammatory M1 macrophages to anti-inflammatory M2 macrophages. This shift is supported by the clot's enriched microenvironment of growth factors (e.g., PDGF, VEGF) and fibrin scaffolding, which also reduces iron availability, a factor that otherwise sustains M1 dominance in hard-to-heal wounds.[10,23,24] The M2 macrophages foster tissue regeneration, angiogenesis and ECM remodelling, critical for resolving inflammation and restoring tissue integrity.[24,25] By enhancing neutrophil clearance and promoting anti-inflammatory cytokine production, the blood clot fosters a shift to M2 macrophages, which contribute to tissue regeneration, angiogenesis and ECM remodelling, highlighting the therapeutic potential of blood clot tissue in managing hard-to-heal wounds (Table 1).[4,24,26]

Procedure

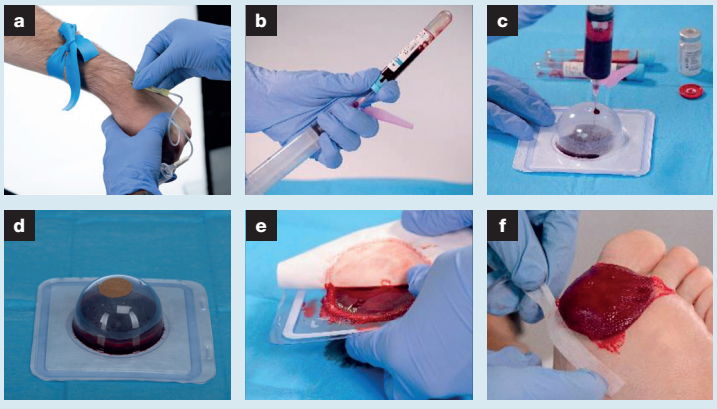
AWBC preparation is a minimally invasive process that can be prepared and administered at the point of care. Preparing the wound bed is a crucial step in optimising the wound conditions for healing. Effective preparation includes proper debridement, maintaining appropriate moisture levels, implementing an offloading regimen, and ensuring robust infection/inflammation control measures to enhance treatment success.[27] The treatment guideline was extensively addressed in a review that introduced the development of consensus guidelines for treating complex wounds with AWBC.[27,28] AWBC benefits in facilitating autolytic debridement, infection

Fig 1. Autologous whole blood clot interactions in the wound bed (image created by RedDress Medical, US). FGF—fibroblast growth factor; IGF—insulin-like growth factor; KGF—keratinocyte growth factor; PDGF—platelet-derived growth factor; TGF—transforming growth factor; VEGF—vascular endothelial growth factor



control and the creation of a moist healing environment, by stimulating the complete array of blood components, were outlined. The AWBC kit contains all the necessary equipment and materials for the creation and application of the blood clot onto the wound, divided into three kit components: a phlebotomy tray, a clot preparation kit and dressing materials.

Fig 2. Preparation of an autologous whole blood clot. The patient's blood was first withdrawn into an acid citrate dextrose solution A tube **(a)**. The blood was then collected into a syringe using a safety needle **(b)**. The blood was injected into a coagulation mould containing calcium gluconate and kaolin in powder form **(c)**. The mould was then sealed and the blood mixed with the coagulation components for 20 seconds and allowed to coagulate inside the mould for ~5 minutes **(d)**. Once the blood was coagulated, the clot was removed from the mould **(e)**. The clot was placed on top of the wound and secured with Steri-strips; a non-adherent dressing and absorbent foam was placed on top of the clot and covered with gauze **(f)** (images created by RedDress Medical, US)



The preparation of the blood clot therapy begins with the collection of a small sample of blood (~15ml) drawn from the patient into an acid citrate dextrose solution A (ACD-A) tube followed by three steps (Fig 2):

1. **Coagulation:** the blood is transferred to an activation mould that contains calcium gluconate and kaolin, which accelerate clot formation within five minutes
2. **Application:** once the clot forms, it can be easily applied to the wound bed using the Steri-strips incorporated within the ActiGraft commercial kit. A non-adherent dressing is placed on the clot and covered by a hydrophilic, secondary dressing foam
3. **Maintenance:** a weekly re-application is encouraged, depending on the clinician's assessment and the wound's healing response. Weekly applications continue until the wound shows significant signs of closure or re-epithelialisation.

This simple procedure requires no specialised surgical skills and can be completed within minutes, making it a cost-effective option for a variety of healthcare settings, from outpatient clinics to specialised wound care centres.

Efficacy

The efficacy of AWBC has been demonstrated in multiple clinical studies and case series. An RCT revealed that AWBC was significantly more effective than standard of care (SoC) for treating diabetic foot ulcers (DFUs), achieving a 2.73-fold increase in healing rates compared with the control group.[29] Additionally, registry studies involving patients with DFUs,[30] pressure ulcers (PUs)[31] and surgical wounds[32] consistently showed high efficacy in achieving complete wound closure. Notably, these real-world data include patients

with diverse and severe comorbidities and complex wound histories, who are often excluded from controlled trials, providing a more accurate representation of AWBC's practical benefits in varied clinical settings.

The efficacy of AWBC was assessed in >250 wounds of several aetiologies, from both inpatient and outpatient settings in the US, Europe, Israel and South Africa.[29–32] The treated wounds included cases with exposed structures and patients who were candidates for amputation. AWBC not only advanced wound healing and achieved complete closure, but also served as a critical limb-salvage option in complex cases, effectively preventing amputation of the affected limb.[33–35]

Safety profile

AWBC was thoroughly evaluated both pre- and post-licensing, demonstrating a high safety profile. In an RCT involving 119 patients with DFUs, no device-related adverse events (AEs) were observed. Furthermore, wound-related AEs were comparable between the AWBC and SoC groups, with a slight but not statistically significant advantage in favour of AWBC.[29] These data are consistent with the findings from real-world cases, where no device-related AEs were reported, further supporting the high safety profile of AWBC treatment.[29–31,35]

The autologous approach also eliminates risks associated with synthetic or allogeneic materials, such as immunogenic reactions or graft-versus-host disease. The AWBC is biocompatible, minimising risks, such as disease transmission, associated with allografts and significantly reducing the risk of rejection.[7]

Therapeutic applications

AWBC has shown versatility in managing various wound types, including DFUs, PUs, venous leg ulcers (VLUs) and trauma injuries.

Diabetic foot ulcers

DFUs are a growing problem, with a prevalence of 6.3% worldwide.[36] AWBC efficacy in DFUs has been

Table 1. Blood components in the clot and their function in wound healing

Component	Mechanism/function
Platelets	• Mediate haemostasis, thrombosis, vascular patency[17,19,20] • Release growth factors, i.e., PDGF, EGF, VEGF[17,19,20] • Promote fibroblast proliferation and ECM synthesis[17,21,22] • Support re-epithelialisation and angiogenesis[17] • Participate in immune defence: proinflammatory mediators, NET formation, leukocyte migration[17,19]
Fibrin	• Prevent haemorrhage[7] • Acts as provisional ECM scaffold[7] • Mechanical barrier against bacteria[7] • Supports cell migration, angiogenesis and tissue regeneration[44]
Macrophages	• AWBC promotes M1-to-M2 transition via growth factors and reduced iron levels[10,23,24] • M2: anti-inflammatory, promote ECM remodelling, angiogenesis, tissue regeneration[24,25]
Neutrophils	• Early bacterial clearance[17,19]
Growth factors	• PDGF, EGF: stimulate fibroblast proliferation, ECM deposition[21,22] • VEGF: angiogenesis, re-epithelialisation[10,17] • TGF-β: immune cell modulation, ECM production, fibrosis[16]

AWBC—autologous whole blood clot; ECM—extracellular matrix; EGF—epidermal growth factor; NET—neutrophil extracellular trap; PDGF—platelet-derived growth factor; TGF-transforming growth factor; VEGF—vascular endothelial growth factor

Table 2. Autologous whole blood clot studies

Study	Study design	Sample size, n	Wound type	Comparator	Main outcomes	Study limitations
Snyder et al. (2018)[37]	Prospective, open-label pilot	20	DFU	None	Healing rate: 65% (ITT), 72.2% (PP); PAR: 61.6% (ITT), 60.3% (PP) at 4 weeks; 67.1% (ITT), 76.2% (PP) at 12 weeks	Small sample size; no control group
Williams et al. (2022)[30]	Observational	29	DFU	Published control	Mean PAR: 75.48±27.99% at 4 weeks; 95% complete closure at 12 weeks	Observational design; no control group
Snyder et al. (2024)[29]	RCT	119 randomised (59 AWBC+SoC; 60 SoC)	DFU	SoC	Healing: 41% vs. 15% (ITT; p=0.002); 51% vs. 18% (PP; p=0.0075)	Moderate sample size
Landau et al. (2023)[31]	Observational	22	PU	Published control	Mean PAR: 77.9±24.8% at 4 weeks; 86.3% achieved PAR>40% at 12 weeks; 45.5% complete closure at 12 weeks	Observational design; no control group
Gurevich et al. (2023)[32]	Case series	14	Surgical wounds	None	PAR: 72.33% at 4 weeks; 78.54% healed by 12 weeks	Small sample; no control group
Ram et al. (2024)[44]	Open label	51	Pilonidal sinus	None	82.4% healed at 3 months; 90.2% at 6 months; 82% durable healing at 1 year	No control group

AWBC—autologous whole blood clot; DFU—diabetic foot ulcer; ITT—intention-to-treat; PAR—percentage area reduction; PP—per protocol; PU—pressure ulcer; RCT—randomised controlled trial; SoC—standard of care; vs.—versus

extensively studied. In a pre-licensing study, evaluating AWBC efficacy and safety in Texas grade 1A and 2A DFUs in 20 patients, AWBC weekly treatment achieved 65% and 72.2% healing rates in the intent-to-treat (ITT) and per-protocol (PP) populations, respectively. The percentage area reductions (PAR) were 61.6% and 60.3%, respectively, at four weeks and 67.1% and 76.2% at 12 weeks, respectively, for the ITT and PP populations.[37] Those results were supported in two post-marketing studies. In a large RCT,[29] 199 patients with hard-to-heal DFUs of at least 30 days' duration were screened for the study. Patients underwent a two-week run-in phase, during which they were treated with alginate or hydrogel wound dressing, including offloading, debridement, moisture and infection control, and an adequate dressing regimen, to ensure the inclusion of only hard-to-heal wounds. Of the 199 patients screened, 119 met the eligibility criteria and were randomised to receive either AWBC+SoC (n=59) or SoC alone (n=60). AWBC treatment demonstrated significantly higher healing rates, with 41% of the wounds in the AWBC+SoC group healed versus 15% in the SoC-alone group (p=0.002) in the ITT population, and 51% in the AWBC+SoC group versus 18% in the SoC-alone group (p=0.0075) in the PP population. AWBC showed clear superiority over SoC, with an odds ratio of 2.73 for healing hard-to-heal DFUs.[29]

In a real-world data registry study[30] involving 29 patients with DFUs, AWBC treatment demonstrated high efficacy, despite the presence of multiple comorbidities, including type 2 diabetes, neuropathy, hypertension, hyperlipidaemia and peripheral arterial disease. The baseline wound size averaged 9.36cm^2. AWBC treatment resulted in a mean PAR of 75.48±27.99% (range: 22–100%) by week 4. By week 12, 95% of the treated wounds achieved complete closure.[30] Data from both RCTs and real-world cases strongly support the high efficacy and significant benefits of AWBC in treating DFUs.

Pressure ulcers

PUs are the third most costly disease after cancer and cardiovascular conditions, contributing significantly to the healthcare burden.[38] The mortality associated with PUs is 2–6 times higher than other diseases, resulting in approximately 60,000 deaths annually.[39] These wounds are most commonly observed in inpatient settings, particularly affecting the sacrum, heels and other bony extensions of the extremities.[40]

The efficacy of AWBC in treating PUs was first demonstrated in a pilot study conducted at a nursing facility, which examined four hard-to-heal PUs located on the sacrum and heel, with wound areas ranging from 3.2–6.6cm². The wounds were treated weekly with AWBC applications, resulting in three wounds achieving complete closure after 5–7 applications, while one wound demonstrated an 82% reduction in wound area after five applications.[41]

The efficacy of AWBC was demonstrated using data from an observational study, analysing 22 PUs treated with AWBC. The mean wound area at baseline was 20.7±30.3cm^2 (range: 0.2–129cm^2) and the mean wound duration was 13.4±17.1 months (range: 1–60 months), with 50% of the ulcers classified as stage 2–4 PUs. The results showed a mean PAR of 77.9±24.8% by week 4 with 86.3% of the patients exceeding at least 40% PAR at this timepoint. By week 12, all 22 patients achieved a PAR >40%, and 45.5% of patients achieved complete wound closure.[31]

These results show a promising healing effect, especially when comparing them to published data from Palese et al.,[42] which found that patients with a mean PU area of 5.14cm^2 healed with SoC at a rate of 56.7% at 10 weeks. An observational study by Guest et al.[43] found that within a year, 50% of the treated PUs were healed. When comparing AWBC results to the published data, AWBC shows superiority in healing PUs ($p \leq 0.001$). In both comparisons, AWBC was identified as the statistically superior treatment, achieving a higher proportion of 40% PAR at four weeks compared with the current SoC models analysed in this study, and complete wound closure of 45% by week 12. Furthermore, patients in the observational studies had smaller wounds and shorter wound durations, significantly exceeding the benchmarks identified in epidemiological studies as less likely to heal.[42,43]

Surgical wounds

The challenge in surgical wounds is to prevent wound infection and dehiscence, caused by many factors, and mainly attributed to the patient's comorbidities, which can negatively impact recovery time, treatment cost and quality of life. Gurevich et al.[32] explored the effect of AWBC in healing surgical wounds, examining 14 cases of hard-to-heal surgical wounds. The results showed a PAR of 72.33% by week 4, with one-third of patients achieving complete wound closure by that time. By week 12, the number of wounds healed reached 78.54%.[32]

AWBC efficacy in surgical wounds was also demonstrated when combined with minimally invasive procedures, as demonstrated in a large open-label study[44] involving 51 patients with pilonidal sinus (PNS) disease. Patients underwent a minimal trephine procedure where a punch tool was used to create a tunnel in the proximal part of the cyst, which was then cleaned using haemostat forceps and/or a curette, removing all the hair and mucopurulent material before being treated with AWBC in a flowable liquid form. This technique enables the blood to completely fill the cavity and clot within it, taking the shape and structure of the cavity. The patient population included individuals with challenging cases, such as those with multiple prior excision attempts, sinus discharge, and pain associated with the PNS. Results showed that 82.4% of patients achieved complete healing at three months, increasing to 90.2% at six months. Furthermore, 82.0% of patients maintained durable healing at one

year, demonstrating the long-term effect of incorporating AWBC into a surgical procedure.[44]

Discussion

AWBC represents a novel approach to wound management, with strong evidence supporting its efficacy and safety across a variety of hard-to-heal and complex wound types. Its innovative use provides several distinct advantages that address key challenges in wound healing. By providing an autologous therapy, the treatment eliminates the risks associated with synthetic allogeneic therapies, i.e., immunogenic reaction, disease transmission or graft rejection. The autologous nature of the therapy makes it highly biocompatible and suitable for a broad range of patients, including those with significant comorbidities. The formed clot is suggested to mimic the ECM properties, creating an optimal healing environment in the wound by providing a dynamic system that regulates the activity between the different cells in the wound area.[7] The clot also supports the release of growth factors and cytokines, which stimulate the ECM reconstruction.[3] Furthermore, the mechanical barrier created by the clot is another protective layer, preventing bacterial infiltration and infection, adding to the accumulated benefits of AWBC therapy in promoting wound healing in hard-to-heal wounds.

Among the benefits of AWBC is the simplicity of its procedure. Designed as a point-of-care treatment, AWBC does not require specialised surgical skills, making it easily accessible in both outpatient and inpatient settings. The kit is organised into three sections: a phlebotomy tray; a clot preparation kit; and dressing materials. This comprehensive design ensures that clinicians have all the necessary components to prepare and apply the treatment efficiently, significantly reducing procedural complexity and time.

The growing body of evidence supporting the efficacy of AWBC in treating hard-to-heal wounds highlights its strong safety profile and potential for treating cutaneous wounds. With its combination of biological activity, patient-centred design and simplicity, AWBC has the potential to become a cornerstone therapy in the evolving field of regenerative wound care.

Limitations

Several limitations of this review should be acknowledged. The selection of included studies was guided primarily by clinical relevance and thematic focus, which introduced the possibility of selection bias in the evidence presented. The available clinical evidence for AWBC is largely derived from registry studies and observational data rather than RCTs, which limits the strength of conclusions that can be drawn regarding efficacy. While the single large RCT provided the most robust level of evidence, the remaining studies were predominantly registry-based, with relatively small sample sizes that may not have fully represented the broader patient population encountered in routine clinical practice. Importantly, however, the registry-based nature of much of the data also means that patients were enrolled without restrictive inclusion or exclusion criteria, reflecting a broad population with diverse comorbidities and wound characteristics. This adds a degree of real-world variability to the findings, suggesting that the observed outcomes may be generalisable beyond the highly selected populations typically enrolled in controlled trials.

Conclusion

AWBC represents a significant advancement in wound care by stimulating the body's natural healing mechanisms to treat hard-to-heal wounds. Its autologous nature eliminates risks associated with synthetic and allogeneic therapies, enhancing patient safety and biocompatibility. The evidence supporting AWBC demonstrates its efficacy in treating various wound types, including DFUs, PUs and surgical wounds. The ability of the treatment to improve healing rates, prevent amputations, and offer a minimally invasive, cost-effective option underscores its potential as a cornerstone therapy in regenerative wound care. Furthermore, its simplicity and accessibility make it a practical solution for a wide range of healthcare settings, from specialised wound centres to outpatient clinics.

As hard-to-heal wounds continue to challenge healthcare, AWBC emerges as an innovative solution that targets the underlying pathophysiology of wound chronicity. By improving patient outcomes and easing the burden on healthcare systems, it adds a valuable treatment to the physician's toolbox for managing hard-to-heal wounds. **JWC**

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Reflective questions

- In what ways does the nature of autologous whole blood clot (AWBC) therapy offer advantages over synthetic or allogeneic therapies, both clinically and practically?
- What is the advantage of AWBC in addressing the underlying pathophysiology of hard-to-heal wounds compared to other wound care products?
- Given the simplicity and flexibility of AWBC application, how could its adoption reshape the delivery of advanced wound care across different healthcare settings (inpatient outpatient, home care)?

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