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A Modern Multimodal Approach to the Treatment of Venous Trophic Ulcers in Outpatient Settings

Volodymyr Goshschynsky[1], Bogdan Migenko[2], Pavlo Hoshchynskyi[3], Volodymyr Dzhyvak[3]*

[1] Department of Surgery, Faculty of Postgraduate Education, I. Horbachevsky Ternopil National Medical University, Ternopil, Ukraine
[2] Department of Therapy and Family Medicine, I. Horbachevsky Ternopil National Medical University, Ternopil, Ukraine
[3] Department of Children's Diseases and Pediatric Surgery, I. Horbachevsky Ternopil National Medical University, Ternopil, Ukraine
* Correspondence: djyvak@tdmu.edu.ua; +380 66 038 3714

Abstract

Background. The treatment of trophic ulcers of the lower extremities is challenging due to their chronic nature, frequent relapses, and significant impact on quality of life.

Aim. This study assessed the effectiveness of a multimodal treatment combining ultrasonic debridement, platelet-rich plasma (PRP), and radiofrequency vein ablation in patients with decompensated varicose veins complicated by trophic ulcers.

Methods. A total of 105 patients with C6-stage varicose veins (CEAP classification) and venous ulcers lasting 2.1 ± 0.6 years (average size – 35 ± 1.7 cm²) were examined. All underwent ultrasound diagnostics to determine phlebohemodynamic disorders. During preoperative preparation, 70 patients received ultrasonic cavitation debridement using the SONOCA 185® device (25 kHz), performed twice at 1–2-day intervals. The control group (35 patients) used chlorhexidine bigluconate for wound cleansing. After debridement, patients were divided into two subgroups: 60 patients received combined PRP and platelet-rich fibrin (PRF) therapy to stimulate tissue regeneration, while 45 were treated with Granuflex® hydrocolloid dressings. Bacteriological and cytological studies were conducted to assess wound regeneration and control infection.

Results. Sequential use of ultrasonic debridement followed by PRP/PRF therapy created optimal conditions for stimulating granulation and epithelialisation in trophic ulcers. This approach provided effective local wound preparation and allowed successful performance of radiofrequency ablation to eliminate the underlying venous reflux responsible for ulcer persistence.

Conclusions. A multimodal strategy combining ultrasonic debridement, PRP/PRF stimulation, and subsequent superficial vein surgery is a highly effective office-based method for treating venous trophic ulcers, promoting faster healing, reducing infection, and enabling timely surgical correction.

Keywords: trophic ulcers; ultrasonic debridement; platelet-rich plasma

1. INTRODUCTION

The number of patients with trophic ulcers of the lower extremities of venous aetiology is constantly increasing (Savoliuk & Dembitskyi, 2023). This requires significant material costs for the treatment and rehabilitation of this category of patients (O'Donnell et al., 2014). The variety of methods of approach to the treatment of trophic ulcers indicates the absence of a single reliable method of treatment that would allow obtaining a stable positive result. A positive effect of treating a patient with a long-term venous ulcer can only be achieved when the chain of pathological changes is broken and the cause of its formation is eliminated (Ren at al., 2020). Thus, to remove the pathophysiological causes of trophic ulcers, correcting vertical and horizontal reflux in the system of the great and small saphenous veins as well as in the perforating veins is the primary step (Jahan et al., 2024). It is beyond dispute that radical treatment of chronic venous insufficiency guarantees healing of trophic ulcers (Chatterjee, 2012; Schul et al., 2023). However, 'infected' trophic ulcers deter surgeons from performing operations due to the many possible purulent-inflammatory complications. In this regard, the primary task is the effective treatment of trophic ulcers at the preoperative stage (Goshchynsky et al., 2022).

The modern strategy for the treatment of chronic wounds, including trophic ulcers (wound bed preparation), represents a comprehensive, well-founded intervention of the wound healing process, concluding with the formation of healthy granulation tissue (Barrett, 2017; Leaper et al., 2012). In the early 2000s, the theory of wound bed preparation was developed with the aim of converting a chronic wound into an acute one and removing both the necrotic component, consisting of necrotic tissue, and phenotypically altered cells of the wound edge and bottom and the exudate produced by them (Moore et al., 2019; Witte et al., 2017). It should be noted that a chronic wound differs from a classic acute wound in biochemical, morphological, physiological, and genetic terms. Thus, excessive production of inflammatory cytokines is the basis of persistent inflammation. Excessive production of proteases leads to degradation and inactivation of growth factors and matrix proteins, impaired collagen synthesis, which, interacting with the phenotypic failure of marginal zone cells, leads to impaired healing.

From these positions, the TIME concept (T – tissue, I – infection, M – moisture balance, E – epithelial edge advancement) was developed, including factors – both systemic and local – that impact tissue regeneration in the ulcer. According to literature data, the main link of the TIME concept at the beginning of trophic ulcer treatment is the restoration of the functions and condition of the tissues filling the bed using debridement, which makes it possible to clean the wound from necrotic and non-viable tissues (Harries et al., 2016). Debridement has long been recognised as the most important component in the treatment of chronic wounds, and numerous scientific publications have evaluated its positive effect (Zhang et al., 2022).

In recent years, clinical interest in therapy with growth factors released from activated platelets during whole blood centrifugation has increased as an alternative to other methods of influencing the regeneration process in trophic ulcers (Goshchynsky et al., 2022). Platelet-rich plasma has been found to improve wound healing by promoting this process through growth factor (GF). These include platelet-derived GFs (αα, ββ, and αβ), fibroblast GFs, vascular endothelial GFs, epidermal GFs, insulin-like GFs, and transforming GFs (Dzhyvak et al., 2024; Verma et al., 2023). A number of studies have confirmed that, in most cases, chronic wounds responded by accelerating the regenerative process in the wound and significantly reducing their area when autologous platelet-rich plasma was used (Duzhiy et al., 2018).

We believe that the introduction into the practice of outpatient surgery of the phased use of ultrasonic debridement to cleanse trophic ulcers of necrotic and non-viable tissues and plasma enriched with growth factors to accelerate the healing process of trophic ulcers can help radically resolve the issue

of surgically eliminating the causes of their occurrence. The aim is to justify the use of a multimodal approach to the treatment of patients with decompensated varicose veins of the lower extremities on the background of trophic ulcers, which consisted of ultrasonic debridement, platelet-rich plasma, and radiofrequency vein ablation surgery.

2. METHODS

2.1. Study design and ethical approval

This was a prospective clinical study conducted at I. Horbachevsky Ternopil National Medical University, Ministry of Health of Ukraine. The study protocol was reviewed and approved by the institutional Bioethics Commission (Approval ID: 66a, November 1, 2023). All participants provided written informed consent prior to enrolment. This study is a follow up study and includes some findings from Goshchynsky et al. (2022).

2.2. Study participants and inclusion / exclusion criteria

A total of 105 patients diagnosed with decompensated varicose veins of the lower extremities (C6 stage according to the CEAP (Clinical-Etiological-Anatomical-Pathophysiological) classification) were included in the study. The cohort consisted of 59 women and 46 men, with a mean age of 55.0 ± 4.6 years. The mean duration of venous ulcers was 2.1 ± 0.6 years, and the average ulcer area measured 35 ± 1.7 cm². Inclusion criteria comprised patients with superficial or partial venous trophic ulcers involving the dermis, subcutaneous fat, and/or fibrous tissue. Exclusion criteria included deep trophic ulcers involving muscle, fascia, or bone; ankle-brachial index ≥0.7; occlusive arterial disease of the lower limbs; diabetes mellitus; uncompensated heart failure; systemic connective tissue disorders; active malignancy; peripheral nerve injury; hormone therapy; lymphoedema; pregnancy; and other decompensated systemic diseases.

2.3. Data collection and assessment procedures

All participants underwent ultrasound examination to assess phlebohemodynamic disturbances in the affected limb. Prior to ultrasonic debridement, 70 patients were evaluated using the MEASURE system, which included measurements of ulcer length, width, depth, and area, as well as assessment of exudate quantity and quality, wound bed appearance, pain intensity, necrosis, and the condition of the wound edges and periwound skin. Dynamic wound healing was monitored using the +WoundDesk® mobile application (Android OS). Ulcer contours were delineated using the '+WD' calibration scale provided by the application's developers, allowing planimetric assessment of wound healing progress. Dynamic bacteriological analysis of ulcer exudate was performed at baseline and after ultrasonic debridement. Biological samples were collected using sterile swabs and transported in Amies medium. Culturing was conducted using the sequestration method on solid nutrient media, and bacterial strains were identified using a semi-automatic Mini API Analyser (*BioMérieux, France*). For cytological evaluation, sterile, degreased slides were applied to the ulcer centre and fixed by air drying. Slides were then stained with Romanowsky azure–eosin for three minutes, differentiated in distilled water, and air dried. Microscopy was performed at ×40 magnification using a biological microscope equipped with a micro web camera. Cellular composition was assessed based on cell count per unit area and morphological characteristics.

2.4. Ultrasonic debridement procedure

During the initial treatment phase (preoperative preparation), all patients underwent ultrasonic cavitation of trophic ulcers using the SONOCA 185® device (*Söring GmbH, Germany*) operating at 25 kHz (Figure 1). The device power was adjusted according to the amount of devitalised tissue or purulent and fibrinous layers. Each patient received two debridement sessions at 1–2-day intervals. A 0.9%

saline solution, supplemented with Prontosan® in a 1:10 ratio, served as the irrigation fluid to enhance decontamination. Ultrasonic treatment was performed under local anaesthesia using 0.75% ropivacaine. Different ultrasonic tips were selected based on ulcer morphology: a 'ball' tip for deep, irregular ulcers and a 'hoof' tip for superficial wounds. The exposure time was maintained at 5–15 seconds per cm² of wound surface. Systemic antibiotic therapy was not administered.

Figure 1. **SONOCA 185® device from Söring GmbH (25 kHz), ultrasonic-assisted wound debridement instrument ('double ball' tip)**

2.5. Treatment groups and regenerative therapy

Following ultrasonic debridement, patients were randomly divided into two treatment groups. The first group (n = 60) received a combination of platelet-rich plasma (PRP) and platelet-rich fibrin (PRF) to stimulate wound regeneration. The second group (n = 45) received Granuflex® hydrocolloid dressings (*ConvaTec, England*) as standard care which served as the control (Figure 2).

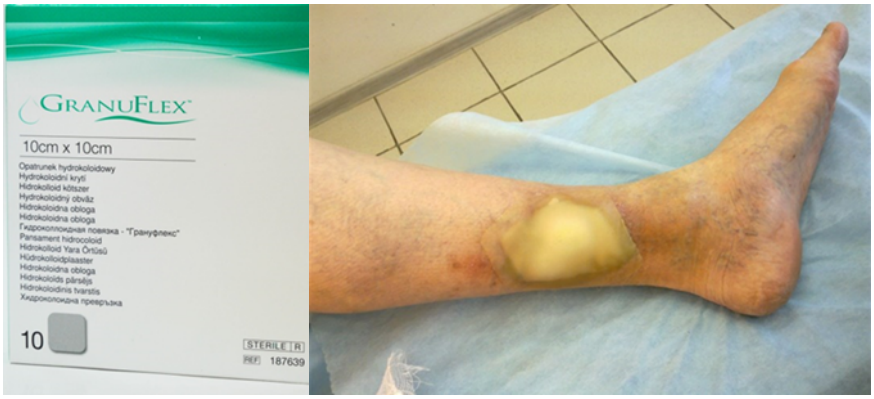


Figure 2. **Use of Granuflex® hydrocolloid dressing (*ConvaTec, England*) in patients with trophic ulcers (control group)**

PRP was prepared following the BTI Biotechnology Institute protocol (*PRGF®–ENDORET®, Spain*) (Figure 3).

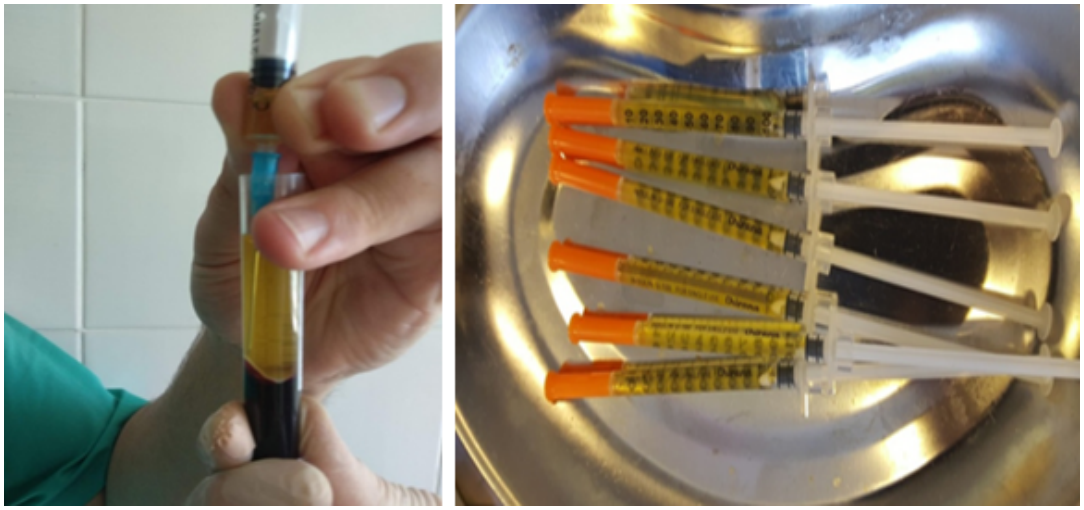


Figure 3. **Collection of platelet-rich plasma (PRP) after centrifugation for injection into trophic ulcer tissue**

Under aseptic conditions, subcutaneous injections were administered along the ulcer periphery, maintaining a distance of 1 cm from the edge and 2 cm between injection points (Figure 4).

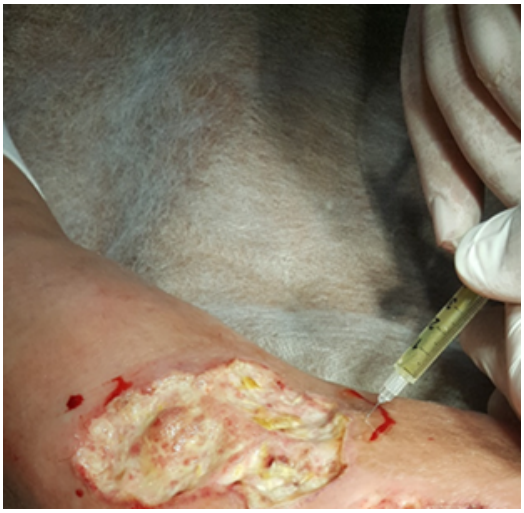


Figure 4. **Plasma injection along the periphery and base of the trophic ulcer**

Simultaneously, the ulcer surface was covered with a plasma membrane enriched with growth factors (Figure 5), providing a biological scaffold to promote epithelialisation and tissue regeneration.

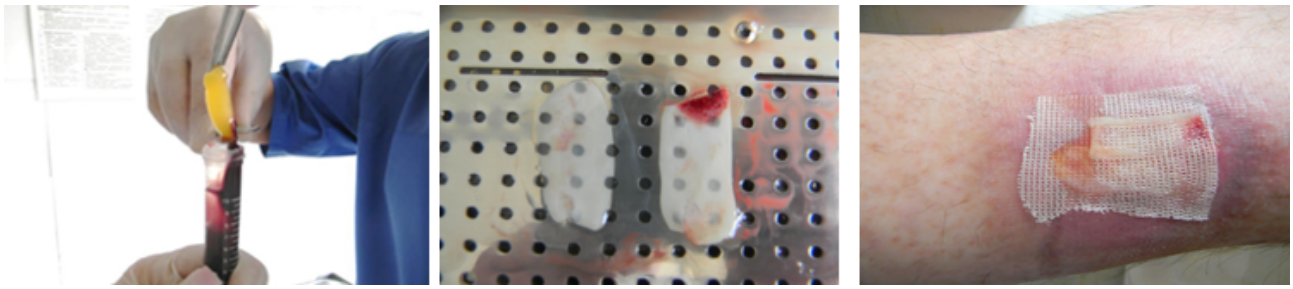


Figure 5. **Application of plasma membrane enriched with growth factors for closure of the trophic ulcer surface**

2.6. Statistical analysis

All statistical analyses were performed using licensed software packages STATISTICA 10.0 (*StatSoft Inc., USA*) and Microsoft Excel (*Microsoft Corp., USA*). Data are presented as mean ± standard deviation (SD). Parametric tests were applied, including Student's *t*-test, to determine the significance of differences between groups. A p-value < 0.05 was considered statistically significant.

3. RESULTS

3.1. Microbiological findings

Microbiological examination of trophic ulcers revealed polymicrobial contamination involving combinations of *Escherichia coli* + *Pseudomonas aeruginosa*, *P. aeruginosa* + *Staphylococcus aureus*, *P. aeruginosa*, *S. aureus*, *Staphylococcus epidermidis*, *Acinetobacter* spp., and *Klebsiella* spp. The initial bacterial load ranged from 10^5 to 10^8 CFU/g of tissue, indicating a high level of infection.

Gram-positive microorganisms accounted for 55% of isolates, with *S. aureus* being the most prevalent species (76%). Gram-negative bacteria were cultured in 34% of samples, predominantly *E. coli* (38%). Seven days following ultrasonic cavitation, the predominant isolates were *P. aeruginosa*, *S. aureus*, and *S. epidermidis*, while non-fermenting Gram-negative bacilli and enterobacteria were detected in 29% and 21% of cases, respectively. The overall bacterial load significantly decreased to 10^2–10^3 CFU/g of tissue, confirming the pronounced antimicrobial efficacy of ultrasonic debridement.

3.2. Cytological findings

Baseline cytological analysis demonstrated inflammatory–regenerative cytograms characterised by a predominance of neutrophils, mainly segmented forms. Phagocytic neutrophils comprised 10.5 ± 1.1%, while degenerative neutrophils accounted for 7.2 ± 0.6%. Degenerative forms appeared swollen, with indistinct contours, altered nuclear morphology, and a reddish cytoplasmic hue. Neutrophilic phagocytes were large, containing cytoplasmic vacuoles and inclusions. A small number of macrophages (1.4 ± 0.5%) and fibroblasts (0.8 ± 0.3%) were also observed.

After the first session of ultrasonic debridement, the cytological pattern shifted towards a regenerative–inflammatory type. A marked reduction in segmented neutrophils (21.0 ± 1.4%) and degenerative neutrophils (6.5 ± 0.6%) was observed, with total neutrophil content decreasing to below 53.1%. Concurrently, the number of connective tissue cells increased, including fibroblasts (8.5 ± 0.4%), histiocytes (5.0 ± 1.2%), and macrophages (5.2 ± 0.3%), indicating active wound cleansing and phagocytic activity.

Following the second debridement session (Goshchynsky et al., 2022), the cytograms demonstrated a predominance of fibrocytes and fibroblasts, with the appearance of endothelial and squamous epithelial cells, signifying progression to the reparative phase of healing (Table 1).

Table 1. **Cytogram characteristics of trophic ulcers before and after ultrasonic debridement (Goshchynsky et al., 2022, p. 11)**

Cell type	**Cell content in trophic ulcer in % (mean ± SD)**		
	Before ultrasonic debridement	After the first ultrasonic debridement session	After the second ultrasonic debridement session
Segmented neutrophils	46.0 ± 2.9	19.3 ± 1.2	0.8 ± 0.3
Phagocytic neutrophils	4.6 ± 0.1	3.3 ± 0.2	2.4 ± 0.3
Degenerative neutrophils	7.2 ± 0.6	6.5 ± 0.6	7.0 ± 0.7

Cell type	Cell content in trophic ulcer in % (mean ± SD)		
	Before ultrasonic debridement	After the first ultrasonic debridement session	After the second ultrasonic debridement session
Eosinophils	2.2 ± 0.3	7.0 ± 0.7	0.25 ± 0.1
Lymphocytes	21.7 ± 0.6	16.2 ± 0.3	16.7 ± 0.4
Monocytes	2.0 ± 0.3	3.0 ± 0.3	3.0 ± 0.6
Histiocytes	3.9 ± 0.3	5.0 ± 1.2	3.5 ± 0.9
Macrophages	1.4 ± 0.5	5.2 ± 0.3	7.0 ± 0.4
Fibroblasts	0.8 ± 0.3	8.5 ± 0.4	10.0 ± 0.7
Fibrocytes	–	21.0 ± 2.0	24.0 ± 2.9
Endothelium	–	+	+
Epithelium	–	2.0 ± 0.2	0.25 ± 0.1
Type of cytograms	Inflammatory–regenerative	Regenerative–inflammatory	Regenerative

3.3. Morphological findings

Histological examination prior to ultrasonic debridement revealed pronounced intercellular oedema, a disorganised extracellular matrix (ECM), scarce fibroblast-like cells, and dense inflammatory infiltration (Figure 6). These findings confirm that the prolonged presence of chronic trophic ulcers disrupts normal healing processes.

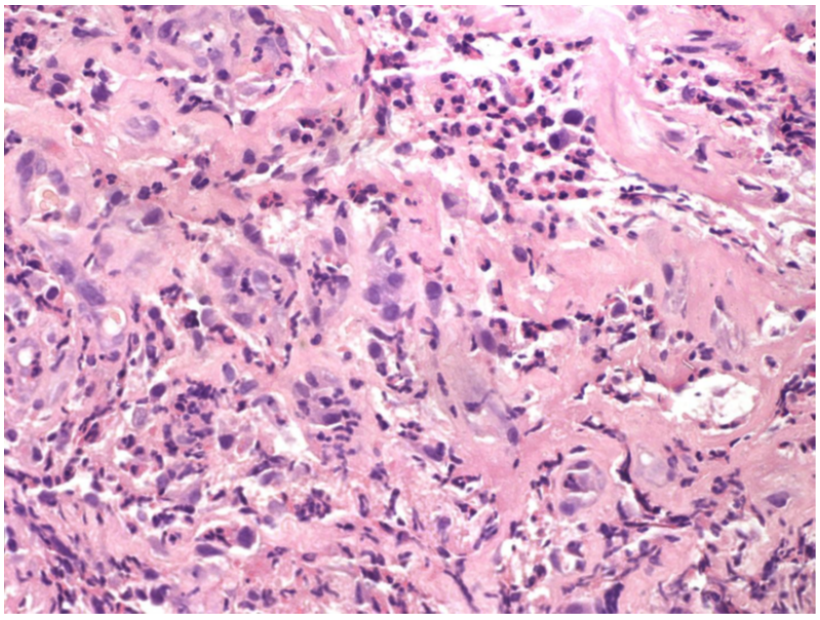


Figure 6. **Typical histological features of a trophic ulcer before ultrasonic debridement (inflammatory phase). A superficial leukocyte–necrotic layer composed of leukocytes and lymphoid**

Following ultrasonic debridement, a significant reduction in intercellular oedema was observed. Although intergroup differences were not statistically significant, ultrasound treatment accelerated the

transition of granulation tissue from ‘young’ to ‘mature’ forms. No substantial reorganisation of ECM structure was detected, likely due to haematoxylin–eosin staining limitations (Figures 7–8).

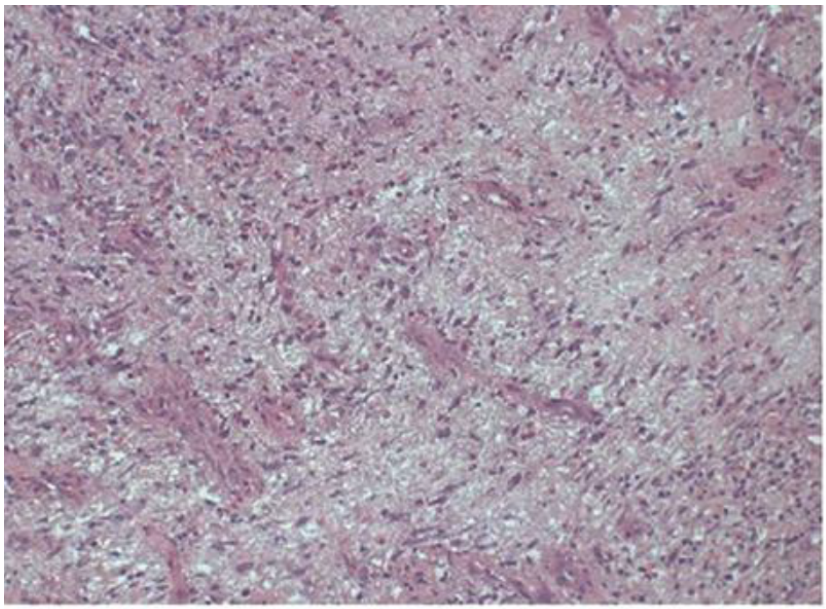


Figure 7. **Wound biopsy after ultrasonic debridement (haematoxylin–eosin staining, ×200). Reduced inflammatory infiltrate and oedema, with organised ECM and formation of mature granulation tissue**

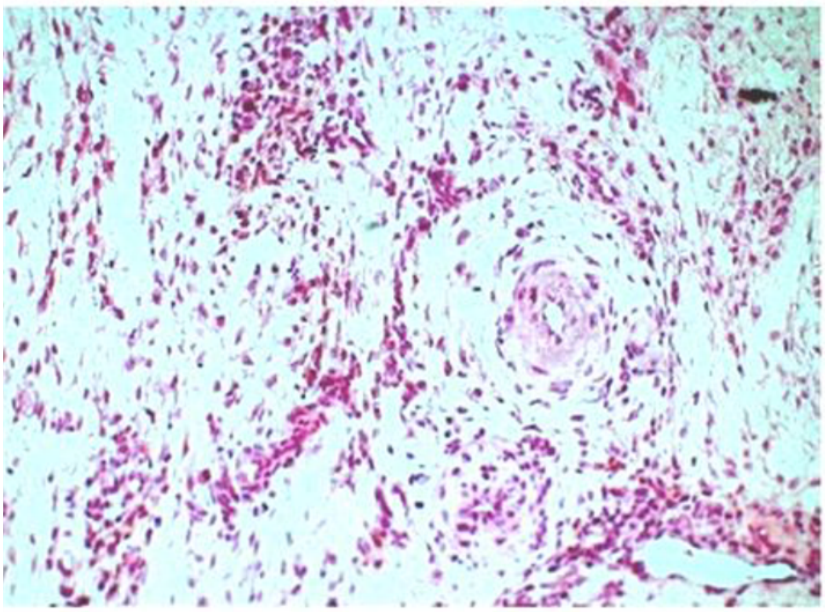


Figure 8. **Morphological image of a healing trophic ulcer in control patients (Granuflex® hydrocolloid dressing, *ConvaTec*). Persistent intercellular oedema and inflammatory cells remain (haematoxylin–eosin staining, ×200)**

3.4. Clinical outcomes after PRP and PRF therapy

Implementation of the TIME concept provided an optimal environment for subsequent stimulation of regeneration using PRP and PRF therapy. After the first PRP + PRF session, patients reported a significant reduction in pain intensity from 5.0 ± 0.2 to 1.4 ± 0.6 points, along with improved sleep, appetite, and normalisation of body temperature.

Compared with the control group, patients receiving PRP + PRF therapy demonstrated more rapid wound healing. The area and depth of ulcers decreased by 23.1%, 37.4%, and 79.9% at days 5–6, 8–9, and 12–14, respectively, relative to baseline values. Perifocal oedema and hyperaemia diminished by the third day in most cases. Granulation tissue formation occurred at 6.74 ± 1.65 days, and marginal epithelialisation at 6.2 ± 1.44 days, significantly earlier than in the control group (9.73 ± 1.4 days and 11.25 ± 1.7 days, respectively; $p < 0.05$).

Cytological evaluation revealed a reduction in inflammatory cells (neutrophils, lymphocytes, monocytes) and a corresponding increase in reparative cells (macrophages, fibroblasts). By days 2–3, neutrophil and lymphocyte numbers decreased markedly, and by days 4–5, all acute inflammatory cells had subsided. Dendritic cells and microbial bodies disappeared, while epithelialisation became evident, confirming the transition from a regenerative–inflammatory to a reparative healing phase (Figures 9–11).

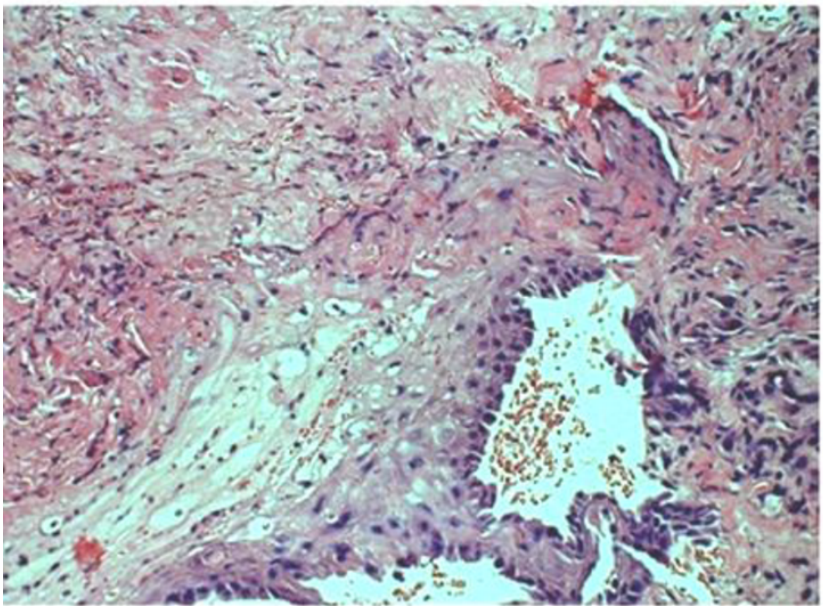
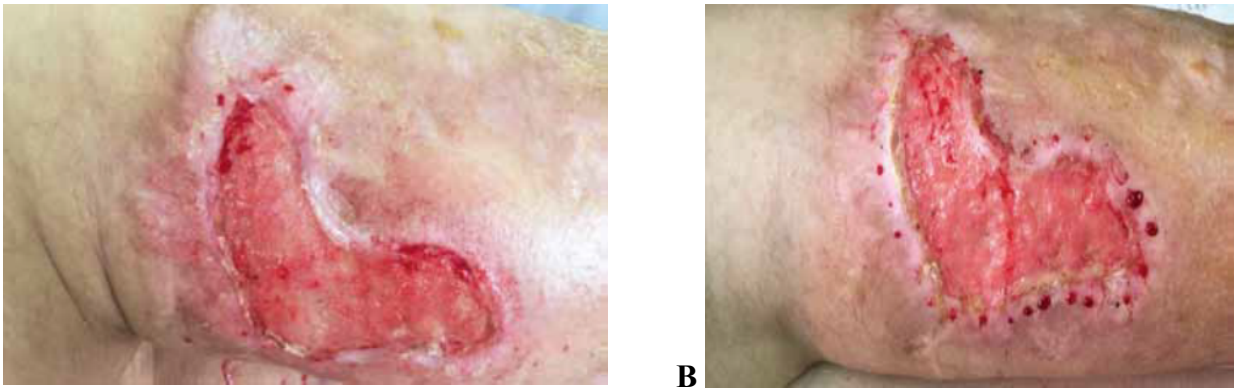
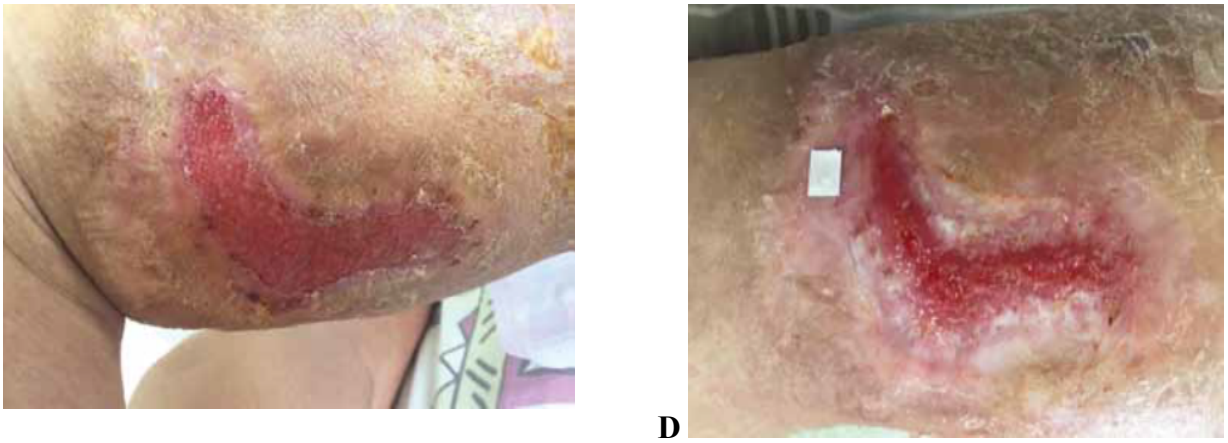


Figure 9. **Light microscopy of a trophic ulcer after PRP + PRF therapy showing active epithelialisation and mesenchymal–epithelial transition**

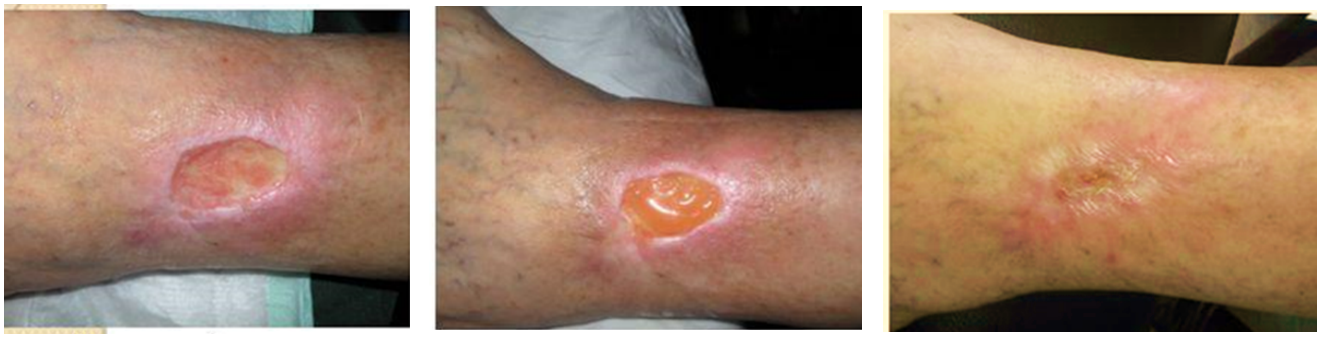


A **B**



C D

Figure 10. **A – trophic ulcer before PRP therapy; B – PRP injections along the ulcer periphery; C – ulcer appearance seven days after the second PRP session showing marginal epithelialisation; D – healed ulcer (day 14) prior to radiofrequency ablation**

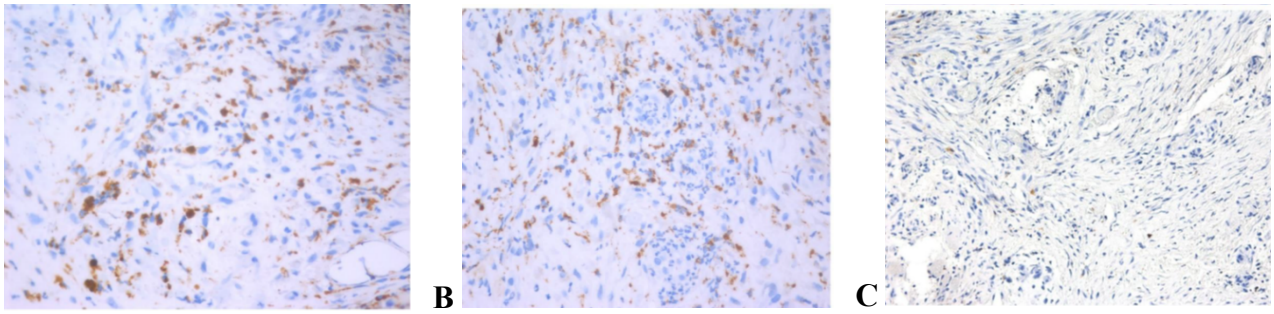


A B C

Figure 11. **Clinical outcome of venous trophic ulcer after PRP + PRF therapy (A – before treatment, B – closure with PRF following PRP, C – complete epithelial healing)**

3.5. Immunohistochemical findings

Immunohistochemical (IHC) analysis demonstrated a pronounced increase in $CD68^+$ macrophage expression following PRP + PRF therapy compared with both the Granuflex® and chlorhexidine-treated groups (Figure 12). This finding confirms the stimulatory effect of platelet-enriched plasma on macrophage-mediated tissue repair.



A B C

Figure 12. **IHC staining for $CD68^+$ (×400). A – marked macrophage infiltration after PRP + PRF therapy; B – moderate increase after Granuflex® dressing; C – isolated macrophages after chlorhexidine dressing**

Collectively, these results confirm that the combined use of ultrasonic debridement followed by PRP + PRF therapy significantly enhances wound regeneration, reduces inflammation, and accelerates epithelialisation in patients with venous trophic ulcers. The described therapeutic approach proved to be an effective preoperative strategy, enabling successful radiofrequency ablation in 49 patients and combined radiofrequency ablation with ultrasound-guided sclerotherapy and autodermoplasty in an additional 21 patients.

4. DISCUSSION

Effective management of long-standing venous trophic ulcers requires interruption of the pathological cascade and targeted correction of the underlying causes. Chronic venous insufficiency remains the primary aetiological factor in these wounds, and radical surgical treatment is the most reliable approach to achieve complete ulcer healing (Colenci & Abbade, 2018). However, before surgical interventions such as phlebectomy or radiofrequency ablation, precise assessment of the superficial and deep venous systems using high-resolution ultrasound is essential to identify the sources of venous reflux and define the surgical plan. Ultrasound examination is widely recognised as the gold standard in diagnosing venous pathology and forms a cornerstone of the multimodal approach to trophic ulcer management.

A major challenge in treating trophic ulcers is the risk of infection associated with the chronic ulcer surface. The presence of persistent bacterial biofilms not only delays wound healing but also increases the risk of purulent-inflammatory complications during surgery. Therefore, effective preoperative decontamination and wound preparation are critical steps to reduce microbial burden and optimise conditions for both conservative and surgical interventions (Matei et al., 2024).

Our study demonstrates that ultrasonic debridement provides a highly effective method for preparing chronic wounds. Microbiological analysis revealed that pre-treatment ulcers were polymicrobially contaminated, with a predominance of Gram-positive bacteria (55%), particularly *Staphylococcus aureus* (76%), and Gram-negative bacteria in 34% of cases, mainly *Escherichia coli*. Following ultrasonic cavitation, bacterial load decreased dramatically to 10^2–10^3 CFU/g tissue, with the microbial spectrum shifting toward non-fermenting Gram-negative bacilli and enterobacteria. This finding confirms the significant antimicrobial efficacy of ultrasonic debridement and its ability to disrupt biofilms that hinder healing.

Cytological analysis corroborated these microbiological findings, showing that prior to debridement, ulcers were in an inflammatory–regenerative state dominated by neutrophils, including phagocytic and degenerative forms. After two sessions of ultrasonic treatment, there was a marked reduction in neutrophils and an increase in reparative cells, including fibroblasts, fibrocytes, macrophages, and endothelial cells, indicating transition to the regenerative phase. Morphological evaluation supported these results, revealing reduced intercellular oedema, improved organisation of the extracellular matrix, and formation of mature granulation tissue. These findings demonstrate that ultrasonic debridement effectively transforms chronic wounds into an environment conducive to tissue repair and regeneration.

Building upon this foundation, the application of plasma enriched with growth factors (PRP and PRF) further accelerated the reparative process. Patients receiving PRP and PRF exhibited rapid reduction in pain, normalisation of systemic parameters such as body temperature, sleep, and appetite, and significant decreases in ulcer area and depth by 23.1%, 37.4%, and 79.9% at days 5–6, 8–9, and 12–14, respectively. Early reduction in perifocal oedema and hyperaemia was observed as soon as the third day of treatment. Granulation tissue formation and marginal epithelialisation occurred earlier in the PRP/PRF group (6.74 ± 1.65 and 6.2 ± 1.44 days) compared to controls (9.73 ± 1.4 and 11.25 ± 1.7 days; $p <$

0.05). Cytological evaluation indicated a rapid decrease in inflammatory cells and an increase in reparative elements, confirming a shift from inflammatory–regenerative to regenerative–inflammatory healing.

Immunohistochemical analysis further validated these findings, demonstrating a significant increase in $CD68^+$ macrophages in the PRP/PRF group compared to patients treated with hydrocolloid dressings. This enhanced macrophage activity likely contributed to the accelerated clearance of necrotic tissue, modulation of inflammation, and stimulation of tissue repair, underscoring the regenerative potential of platelet-enriched plasma.

Overall, this multimodal approach – combining ultrasonic debridement with PRP/PRF therapy – creates a favourable environment for wound healing and optimises preoperative preparation. The improved tissue quality allowed for safe and effective definitive surgical interventions, including radiofrequency ablation of superficial veins and, where indicated, sclerotherapy of incompetent perforating veins or autodermoplasty. In 49 patients, full radiofrequency ablation was performed, while 21 patients underwent simultaneous ulcer repair with autodermoplasty, demonstrating the clinical applicability and translational value of this treatment strategy.

In conclusion, our findings highlight that integration of ultrasonic debridement and platelet-enriched plasma therapy provides a scientifically supported, effective, and reproducible method for managing chronic venous trophic ulcers. By reducing microbial burden, modulating the inflammatory response, and stimulating regenerative processes, this multimodal strategy enhances wound healing, optimises patient preparation for surgery, and ultimately improves clinical outcomes and quality of life for patients with complex venous ulcers.

5. CONCLUSIONS AND PERSPECTIVES

The results of our study indicate that in an outpatient setting, a multimodal approach to the treatment of venous trophic ulcers, combining ultrasonic debridement with stimulation of the wound healing process using PRP and PRF, is a promising strategy. This approach not only promotes effective wound regeneration but also has the potential to reduce material costs and shorten the rehabilitation period for patients with decompensated chronic venous insufficiency.

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Šiuolaikinis požiūris į veninės etiologijos trofinių opų gydymą ambulatorinės chirurgijos sąlygomis

Volodymyr Goshschynsky[1], Bogdan Migenko[2], Pavlo Hoshchynskyi[3], Volodymyr Dzhyvak[3]*

[1] Chirurgijos katedra, Aukštojo mokslo fakultetas, Ternopilio nacionalinis medicinos universitetas, Ternopilis, Ukraina
[2] Terapijos ir šeimos medicinos katedra, Ternopilio nacionalinis medicinos universitetas, Ternopilis, Ukraina
[3] Vaikų ligų ir vaikų chirurgijos katedra, Ternopilio nacionalinis medicinos universitetas, Ternopilis, Ukraina
* Susirašinėjimui: djyvak@tdmu.edu.ua; +380 66 038 3714

Santrauka

Tyrimo pagrindimas. Apatinių galūnių trofinių opų gydymas yra sudėtingas dėl jų lėtinio pobūdžio, dažnų atsinaujinimų ir didelio poveikio gyvenimo kokybei.

Tikslas. Šiame tyrime buvo vertinamas kombinuotosios terapijos – ultragarsinio audinių valymo, trombocitų prisotintos kraujo plazmos (PRP) ir radijo dažnio venų abliacijos – veiksmingumas pacientams, turintiems lėtinę venų varikozę su trofinės opos komplikacija.

Metodai. Iš viso buvo ištirti 105 pacientai su nustatyta C6 stadijos venų varikoze (CEAP klasifikacija) ir turintys veninių opų maždaug 2,1 ± 0,6 metus (vidutinis opos dydis – 35 ± 1,7 cm²). Visiems dalyviams buvo atlikta ultragarsinė diagnostika, siekiant ištirti flebologinius kraujotakos sutrikimus. Ruošiantis procedūrai, 70-čiai pacientų buvo atliktas (po du kartus per 1–2 dienas) ultragarsinis audinių valymas naudojant SONOCA 185® prietaisą (25 kHz). Kontrolinėje grupėje (35 pacientai) žaizdoms išvalyti buvo naudojamas chlorheksidino biglukonatas. Po valymo pacientai buvo suskirstyti į du pogrupius: 60-čiai pacientų buvo taikoma kombinuota PRP ir trombocitų prisotinto fibrino (PRF) terapija audinių regeneracijai stimuliuoti, o 45 pacientai buvo gydomi *Granuflex*® hidrokoloidiniais tvarsčiais. Buvo atlikti bakteriologiniai ir citologiniai tyrimai, siekiant įvertinti žaizdos regeneraciją ir kontroliuoti infekciją.

Rezultatai. Ultragarsinis audinių valymas, po kurio buvo taikoma PRP/PRF terapija, sukūrė optimalias sąlygas granuliacijai ir epitelizacijai trofinių opų atveju. Šis metodas užtikrino veiksmingą vietinį žaizdos paruošimą ir leido sėkmingai atlikti radijo dažnio abliacijos procedūrą, kad būtų pašalintas veninis refliuksas, atsakingas už opų išlikimą.

Išvados. Kombinuotoji terapija, apimanti ultragarsinį audinių valymą, PRP/PRF stimuliaciją ir tolesnę paviršinių venų operaciją, yra itin veiksmingas ambulatorinis veninių trofinių opų gydymo metodas, skatinantis greitesnį gijimą, mažinantis infekciją ir leidžiantis laiku atlikti chirurginę korekciją.

Reikšminiai žodžiai: trofinės opos; ultragarsinis audinių valymas; trombocitų prisotinta kraujo plazma

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