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ORIGINAL ARTICLE
Reconstructive

Enhanced Closed Incisional Negative Pressure Therapy for Treating Infectious Scars

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Background: Chronic infectious pathological scars, characterized by mutual reinforcement between infection and pathological scarring, pose challenges in reconstructive surgery. We introduce an enhanced closed incisional negative pressure therapy following a 1-stage surgery to simultaneously eradicate infection and alleviate wound tension.

Methods: A total of 25 patients who underwent chronic infectious pathological scar treatment by using this enhanced closed incisional negative pressure therapy were retrospectively reviewed. The outcomes were evaluated by postoperative recurrence frequency of infection and scarring during a 1-year follow-up, as well as the Patient and Observer Scar Assessment Scale and quality-of-life scores.

Results: After treatment, no serious complications, such as incision dehiscence, occurred. The average wound healing time was 12.68 days. Only 1 patient experienced surgical site scarring. Besides, average infection frequency decreased significantly from 6.40 to 0.00 times per year ($P < 0.0001$). The Patient and Observer Scar Assessment Scale score decreased from 81.60 to 25.36 ($P < 0.0001$), whereas the quality-of-life score increased from 2.20 to 4.88 ($P < 0.0001$).

Conclusions: The enhanced closed incisional negative pressure therapy effectively facilitated infectious wound healing in a 1-stage operation and simultaneously prevented infection and scarring recurrence in long-term follow-up, resulting in satisfactory postoperative outcomes. *(Plast Reconstr Surg Glob Open 2025;13:e6776; doi: 10.1097/GOX.0000000000006776; Published online 15 May 2025.)*

INTRODUCTION

Surgical, traumatic, and infectious events leave more than 100 million individuals worldwide with lasting scarring. It is estimated that 15% of scars are hypertrophic scars or keloids.[1,2] These scars are characterized by excessive continuous and histologically localized inflammation, fibrosis, and extracellular matrix deposition in the reticular dermis.[3] Hypertrophic scars do not extend beyond the margins of the original wound, whereas keloids do.[4] Both of them pose significant challenges to reconstructive surgery, particularly when accompanied by infection. Notably, there exists a symbiotic relationship between infection and scars, leading to a vicious cycle that perpetuates scar progression.[5] As scars deepen, sweat, stains, and sebum become entrapped, exacerbating the infection.[6] Repeated or persistent infections are implicated as causative factors in the development of pathological scars. Consequently, these infectious pathological scars tend to exhibit frequent relapses or transform into persistent chronic infectious lesions even after undergoing multiple treatments.[7,8] Infectious scars cause cosmetic disfigurement, physical discomfort, and functional impairment, greatly impacting patients' quality of life (QOL).[1,3] Although various methods, such as surgery,[9,10] laser therapay,[11,12] and radiotherapy,[9,12] have been used for pathological scars, managing infectious scars remains a complex and thorny issue for clinicians.

Negative pressure wound therapy has been widely used to enhance wound healing and prevent complications such as infections and dehiscence in primary closed incisional wounds.[13–15] However, following the removal of infectious and scarring tissues, closed incisions often fail to establish an effective negative pressure environment under the incision, leading to subcutaneous edema and hematoma formation. Furthermore, primary closure of the debrided wound results in an increased subcutaneous tension. These factors pose a high risk for infection and scarring

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Received for publication December 29, 2024; accepted March 27, 2025.

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DOI: 10.1097/GOX.0000000000006776

Disclosure statements are at the end of this article, following the correspondence information.

recurrence. Here, we develop an enhanced closed incisional negative pressure wound therapy (ciNPWT) technique for the treatment of infectious scars. This technique enables simultaneous management of both infection and wound tension within a single-stage operation, addressing 2 critical aspects of infectious scar management effectively; hence, we term this the "enhanced ciNPWT."

Takeaways

Question: Current treatments for infectious scars often require 2 separate operations: one for debridement of necrotic tissue and another for wound closure. After debridement, closed incisions often fail to create effective negative pressure, leading to subcutaneous edema and hematoma formation.

Findings: We developed an enhanced closed incisional negative pressure therapy to treat infectious scars, effectively draining secretions and reducing wound tension in a single-stage surgery, promoting optimal healing and preventing pathological scar recurrence.

Meaning: The enhanced therapy promotes optimal wound healing, reduces the risk of infection, and lowers scarring recurrence, resulting in favorable long-term postoperative outcomes.

METHODS

Patient Enrollment

This retrospective study aims to evaluate the efficacy of our enhanced ciNPWT for postoperatively managing chronic infectious pathological scars. We defined chronic infectious pathological scars as hypertrophic scars or keloids that meet all 3 of the following criteria: (1) presence of infection for more than 1 month; (2) recurrence of infection 2 or more times; (3) presence of sinus localized in the deep region. These chronic infectious pathological scars will be referred to as infectious scars hereafter. In this study, we included patients with infectious scars presenting abscesses, pus, or sinus localized on the chest, abdomen, and perineum (Fig. 1A). Notably, patients with superficial and large infectious scars or those unable to undergo 1-stage suturing after excision due to oversized sinus tracts were excluded from the study. Moreover, patients with uncontrolled diabetes mellitus (2-h postprandial blood glucose > 8.0 mmol/L), cardiopulmonary disease, autoimmune disease, long-term immunosuppressive therapy, or those unable to comply with follow-up were excluded from the study. A total of 25 patients who underwent surgery followed by postoperative enhanced ciNPWT treatment at our hospital between February 2020 and October 2023 were included. Scar assessments were performed preoperatively as well as at 3-, 6-, and 12-month intervals postoperatively. Our study was approved by the ethics committee of our institute, Shanghai Ninth People's Hospital (SH9H-2023-T258-1).

Treatment Protocol

Patients with severe infections upon presentation were managed by internal medicine with general antibiotics administration first, to optimize the infected scar to a certain extent, making it available for 1-stage surgery.

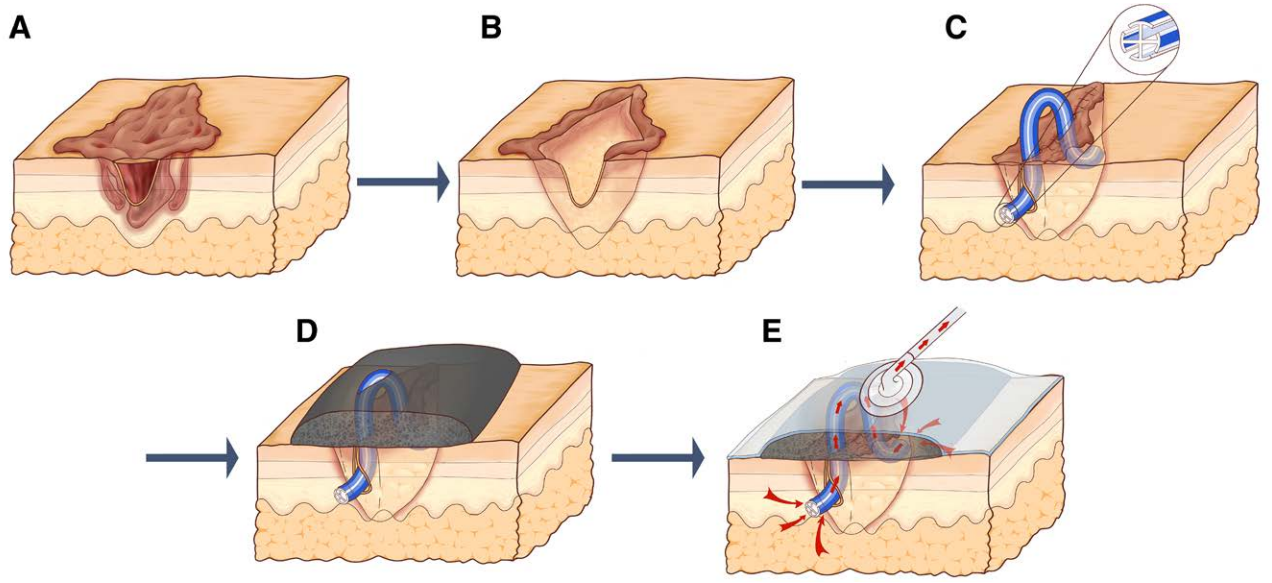

Fig. 1. Schematic drawing of our enhanced ciNPWT. A, A infectious scar wound with abscesses, pus, and sinuses. B, After debridement, the infectious and scarring tissues are removed. C, A fluted drainage tube is placed deep into the wound, with its ends left within the wound and the intermediate portion left outside. The wound is subsequently closed by interrupted sutures. D, A soft open-cell foam is directly placed over the incision, covering the periphery of the incision, and the intermediate portion of the drainage tube is inserted into the foam. E, The foam is sealed with adhesive film. When connected to a negative pressure device, a controlled negative environment forms underneath and above the closed incision, which facilitates the removal of excess fluid and reduces subcutaneous tension. The negative pressure is maintained at a continuous preset pressure of −80 to −100 mmHg.

The debridement and excision of the infectious and scarred tissues were consistently performed by the same operator group (Fig. 1B). A fluted drainage tube (Silicone Drainage Tubes, Fluted; Besmed, China) was then placed deep into the wound, with its ends left within the wound and the intermediate portion left outside. Commonly, the size of the drainage tube (8–15 Fr) was selected based on the depth and size of the wound postdebridement. Tubes were trimmed to 1.5–2 times the length of the incision for better placement in the wound. Subsequently, tension-relieving sutures were used for subcutaneous layers using 0 barbed suture (Quill; Medline),[16–19] followed by routine closure of the skin with a 4-0 suture (Prolene; J&J MedTech) (Fig. 1C). A soft open-cell foam (V.A.C. Granufoam; 3M), which should cover the periphery of the incision, was directly placed over the incision; the intermediate portion of the drainage tube was inserted into the foam (Fig. 1D). The foam was sealed with adhesive film (SensaT.R.A.C.; 3M). When connected to a negative pressure device (V.A.C. ULTA; 3M), a controlled negative environment formed underneath and above the closed incision, which facilitated the removal of excess fluid and reduced subcutaneous tension. The negative pressure was maintained at a continuous preset pressure of −80 to −100 mm Hg (Fig. 1E). During our enhanced ciNPWT treatment, the negative pressure device underwent daily checks for 3 days. Then, patients were discharged with the enhanced ciNPWT treatment continued. Following surgical principles and considering wound healing progress, stitches and the negative pressure therapy device were removed on days 9–15, followed by dressing applications to cover the wound.

Outcome Assessment

To assess the efficacy and safety of our enhanced ciNPWT approach for infectious pathological scars, we conducted preoperative assessments as well as follow-up evaluations at 3, 6, and 12 months postoperatively. Primary evaluation criteria included the recurrence rate of scarring and the infection frequency. Scarring recurrence was defined as a Patient and Observer Scar Assessment Scale (POSAS) thickness score being greater than 5 or thickness exceeding 5 mm.[20] The infection frequency 1 year before and after our management was recorded by medical history inquiry and follow-up assessment.[11] Secondary evaluation criteria included POSAS and QOL scores. POSAS consists of 2 scales: the Patient Scar Assessment Scale (PSAS) and the Observer Scar Assessment Scale (OSAS). Both PSAS and OSAS consist of 6 items rated on a scale from 1 to 10; thus, the total POSAS scores ranged from 10 to 120. For each item, 1 represents a state similar to normal skin, and 10 represents the worst case imaginable. Specifically, OSAS is objectively assessed by the observer on 6 dimensions: vascularity, pigmentation, thickness, irregularity, pliability, and surface.[10] We invited 3 experienced associate chief physicians in our department who were unknown to our study to independently rate these dimensions and took the mean as the final scores. On the other hand, patients assessed PSAS by subjectively evaluating 6 dimensions: pain, pruritus, color, stiffness, thickness, and irregularity.[10] Subjective QOL score, ranging from 0 (extremely affecting life) to 5 (no impact), was assessed. Additionally, wound healing time, defined as the earliest point when there is no wound dehiscence, clinical local infection symptoms, or purulent exudate after suture removal,[21] was recorded. Meanwhile, adverse effects such as blisters and hyperpigmentation were also closely monitored and recorded.[22,23]

Statistical Analysis

Categorical variables were represented as numerical values and percentages, whereas continuous data were expressed as mean ± SD. The ordinary one-way analysis of variance test was applied to analyze each outcome (POSAS score, OSAS score, PSAS score, and QOL score) at different time points. If significant, the post hoc pairwise Tukey test was conducted to compare differences between time points. We constructed 4 generalized linear models with random-effect intercepts for each subject to estimate the effects of our postoperative enhanced ciNPWT approach on each outcome, considering the repeated measures for each patient. The paired *t* test was used to compare the infection frequency before and after the treatment for 1

Table 1. Patient Characteristics

Patient Characteristics	Value
No. patients	25 (from Shanghai Ninth People's Hospital)
Age, y	
Mean	47
Range	34–69
Sex	
Male	5
Female	20
Scar type	
Hypertrophic scar	17
Keloid	8
Etiology	
Surgery	15
Furuncle	5
Folliculitis	3
Burn	2
Scar position	
Thoracic area	12
Abdominal area	11
Perineal area	2
Scar dimension, cm	
Range	1 × 1 to 15 × 5
Period with pathological scar, y	
Mean ± SD	10.60 ± 1.26
Period with infectious pathological scar, y	
Mean ± SD	3.72 ± 0.90
Scar relapse	
Hypertrophic scar	0
Keloid	1
Healing time, d	
Mean ± SD	12.68 ± 0.44
Pretreated infection frequency, per year	
Mean ± SD	6.40 ± 1.29
Posttreated infection frequency, per year	
Mean ± SD	0 ± 0

year. All analyses were performed using GraphPad Prism version 10.0.3 with a statistical significance set at a *P* value less than 0.0001.

RESULTS

Patient Characteristics

We applied our enhanced ciNPWT technique to a cohort of 25 patients, between 34 and 69 years of age, with an average age of 47 years (Table 1). Among them, 17 patients were diagnosed with hypertrophic scars and 8 with keloids based on clinical features and history. The etiology of infectious pathological scars included surgery (15 cases), furuncle (5 cases), folliculitis (3 cases), and burn (2 cases). The involved positions included the mediastinal area (6 cases), paramedian area (6 cases), umbilicus (8 cases), lower abdomen (3 cases), and perineal area (2 cases). The dimensions of infectious pathological scars ranged from 1 × 1 to 15 × 5 cm. Patients had been afflicted by these pathological scars for an average duration of 10.60 ± 1.26 years, with infection complicating the scar condition for approximately 3.72 ± 0.90 years.

Results of the Primary Criteria

In our study, the average healing time for infectious pathological scars was 12.68 ± 0.44 days. Among the patients, only 1 patient experienced keloid relapse. The patient was a 44-year-old woman who had experienced keloid scarring for 18 years, accompanied by infection for 2 years after a left accessory mastectomy (removal of ectopic breast tissue in the axillary region). The scar was broad (12 × 2 × 1 cm) and protruding from the skin surface, with a central ulceration. The patient had not received any pathological scar treatment previously. The keloid relapse occurred 6 months after surgery, but no recurrence of infection was observed during the 1-year follow-up. The mean infection frequency of the infectious pathological scars was 6.40 ± 1.29 and 0.00 ± 0.00 times per year, 1 year before and after the enhanced ciNPWT treatment, respectively. The results showed significant differences ($P < 0.0001$), indicating that our enhanced ciNPWT significantly inhibited the recurrence of infection during the 12-month follow-up.

A representative case is illustrated in Figure 2. The 50-year-old woman developed a 10 × 6 cm infectious "herringbone" keloid on her chest, protruding 0.8 cm above the skin surface. The patient developed keloids after scratching an itchy mass and has had keloids for 23 years, accompanied by infections for 15 years. The patient underwent surgery followed by radiation therapy in 2004, after which the keloid recurred. In 2008, the patient underwent implant surgery, after which the keloid recurred with ulceration. Before our intervention, the scar exhibited a frequency of infection 6 times per year. A sinus tract of about 2 mm in diameter was observed at the center of the keloid. When pressed, it exhibited cystic characteristics with fluid discharge from the sinus tract. Small ulcers were also seen on both sides of the keloid, accompanied by localized redness and swelling, along with yellowish discharges (Fig. 2A). Due to the large size, we excised all infectious scar tissue and surrounding unhealthy tissue, leaving the edge of the scar intact instead of completely removing the entire keloid tissue to avoid excessive tissue tension during wound closure. However, through the combination of surgery and our enhanced ciNPWT treatment, no recurrence of infection or relapse of keloid was found, and the patient was very satisfied (Fig. 2B). Therefore, our approach effectively inhibits infection recurrence and scar progression.

Results of the Secondary Criteria

The total mean POSAS score decreased from 81.60 ± 1.95 preoperatively to 35.72 ± 1.36, 28.88 ± 1.16, and 25.36 ± 1.48 at the 3-, 6- and 12-month follow-up, respectively, which indicates significant improvement after performing our enhanced ciNPWT ($P < 0.0001$). The mean OSAS and PSAS scores were 40.44 ± 1.03, 18.44 ± 0.74, 14.88 ± 0.68, 13.32 ± 0.74 and 41.16 ± 1.45, 17.28 ± 0.80, 14.00 ± 0.64, 12.04 ± 0.83 preoperatively and at the 3-, 6- and 12-month follow-up, respectively, both indicating significant differences ($P < 0.0001$) (Table 2 and Fig. 3). Meanwhile, a significant improvement was observed in OSAS subgroups, including vascularity,

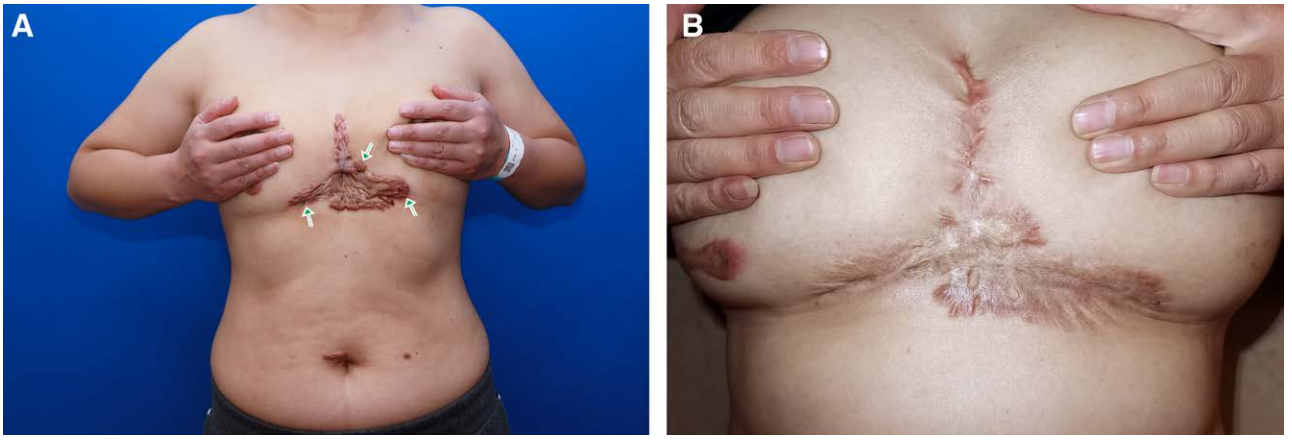

Fig. 2. Photographs of a 50-year-old woman with a thoracic infectious pathological scar. A, Preoperative, with arrows indicating the central sinus tract and the ulcers on both sides. B, Two years postoperative.

Table 2. Effects of Our Enhanced ciNPWT on Each Outcome Based on a Generalized Linear Model

Outcomes	Effect	*F*	*P*	Comparison		Adjusted *P*
				Time, mo	Time, mo	
POSAS	Time	397.2	<0.0001	0	3	<0.0001
				0	6	<0.0001
				0	12	<0.0001
				3	6	0.0030
				3	12	0.0003
				6	12	0.0004
OSAS	Time	343.1	<0.0001	0	3	<0.0001
				0	6	<0.0001
				0	12	<0.0001
				3	6	0.0014
				3	12	0.0001
				6	12	0.0032
PSAS	Time	271.6	<0.0001	0	3	<0.0001
				0	6	<0.0001
				0	12	<0.0001
				3	6	0.0080
				3	12	0.0010
				6	12	0.0010
Scar width (cm)	Time	25.96	<0.0001	0	3	0.0002
				0	6	0.0001
				0	12	<0.0001
				3	6	0.2385
				3	12	0.3006
				6	12	0.5602
QOL score	Time	107.0	<0.0001	0	3	<0.0001
				0	6	<0.0001
				0	12	<0.0001
				3	6	0.1220
				3	12	0.1088
				6	12	0.7508

The *F* value and *P* value represent the result of model construction. When $P < 0.05$, the model is deemed to be meaningful. The right column is the adjusted *P* value. The adjusted $P < 0.05$ suggests that the difference between different time points is statistically significant.

pigmentation, thickness, irregularity, pliability, and surface, as well as PSAS subgroups, including pain, pruritus, color, stiffness, thickness, and irregularity at the 12-month follow-up when compared with the baseline. The mean QOL scores were 2.20 ± 0.23, 4.52 ± 0.13, 4.84 ± 0.09, and 4.88 ± 0.09 at baseline and at 3-, 6-, and 12-month follow-up, respectively, showing significant differences between 0 and 3, 0 and 6, and 0 and 12 months ($P < 0.0001$) (Fig. 4). Indeed, for scars with small size and complete resection, aesthetically pleasing outcomes were successfully achieved (Fig. 5). Notably, based on the POSAS, OSAS, PSAS, and QOL scores, improvements in scar appearance and patient satisfaction were achieved within 3 months, whereas no significant changes were observed at subsequent follow-ups.

Monitoring of Adverse Effects

None of the patients exhibited common surgical site complications, including dehiscence, hematoma, seroma, and edema, indicating that our enhanced ciNPWT maintained the inhibitory effects on the surgical site occurrences. Besides, no instance of blister was observed during the follow-up. Notably, 19 patients experienced sponge-induced hyperpigmentation as early as day 10, which almost disappeared within the first month.

DISCUSSION

For infectious pathological scars, the interplay between infections and pathological scars characterized by excessive dermal inflammation can be likened to twin sisters, mutually reinforcing each other and perpetuating a vicious cycle in the progression of pathological scarring.[5] As long as the infection persists, relapse of pathological scars will inevitably occur frequently. When pathological scars progress to invade the surrounding tissues, particularly those abundant in sebum and follicles, the infection is further exacerbated. In this scenario, sweat and stains are entrapped in scars, whereas sebum provides a favorable breeding ground for microbial infection. The infection leads to unfavorably prolonged inflammation in the reticular dermis.[5] Such infectious pathological scars often end up as persistent chronic infectious scars.[8]

Previously, the treatment of infectious pathological scars has been reported to involve intralesional injection of fluorouracil (5-FU)[24] or laser.[11] Fitzpatrick[24] utilized intralesional injection of 5-FU for treating infectious pathological scars; however, a combination of dye laser and intralesional injection of corticosteroid is often required. In addition, intralesional injection of 5-FU can cause pain or discomfort during administration, purpura at the injection site, and in some cases, local superficial tissue loss.

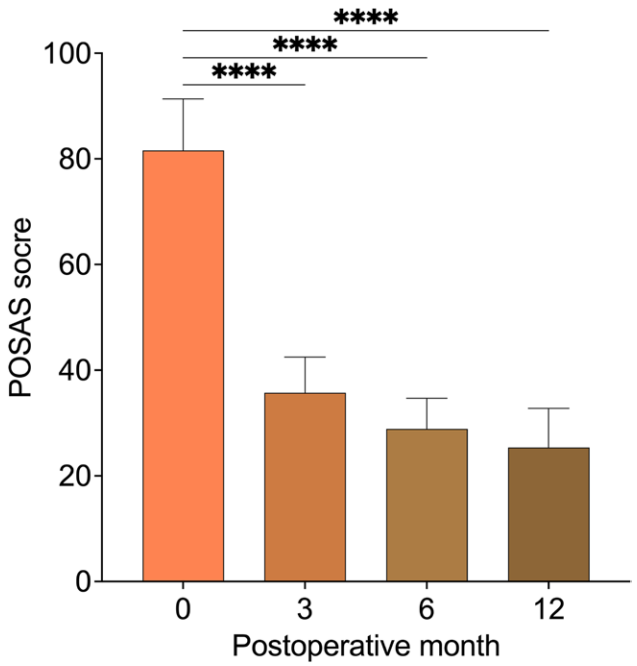

Fig. 3. Evaluation of POSAS score. POSAS score was evaluated before surgery and at the 3-, 6-, and 12-month follow-up after surgery; ****$P < 0.0001$. Repeated measures one-way analysis of variance with post hoc pairwise Tukey test and generalized linear models were applied.

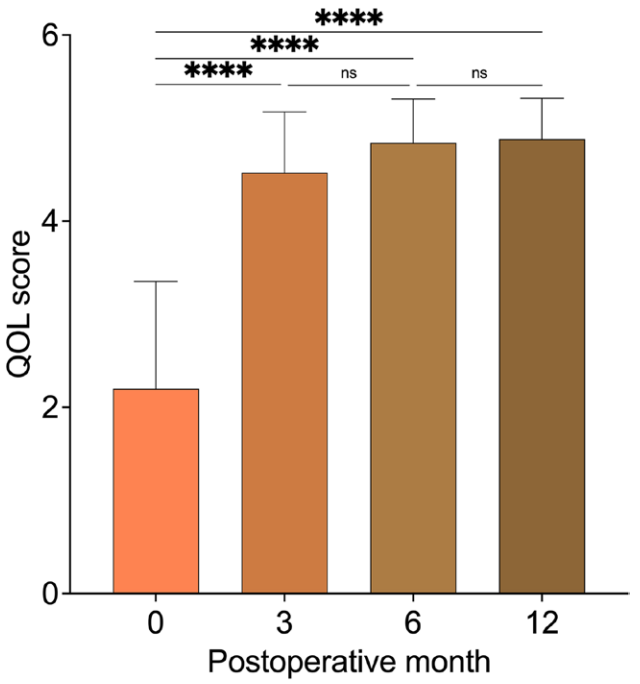

Fig. 4. QOL scores evaluated before surgery and at the 3-, 6-, and 12-month follow-up after surgery. ****$P < 0.0001$, and ns indicates no significance. Repeated measures one-way analysis of variance with post hoc pairwise Tukey test and generalized linear models were applied.

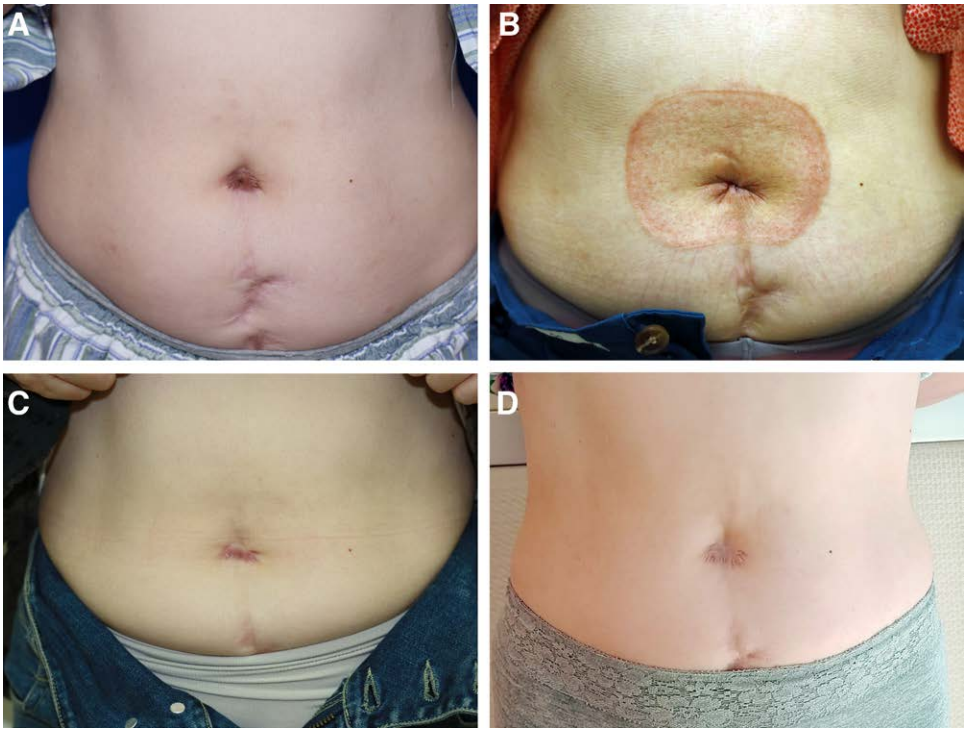

Fig. 5. Photographs of a 52-year-old woman with an umbilical infected hypertrophic scar: preoperative (A), immediately after the suture removal at day 10 (B), 1 month postoperative (C), and 30 months postoperative (D).

Additionally, addressing the recurrence of pathological scars, especially the larger ones, remains challenging and requires long-term periodic treatment that may not be feasible due to issues related to compliance, accessibility, and associated pain caused by injections.[24] Zhang et al[10] demonstrated that noninvasive treatment against infectious keloids with a 1470-nm fiber laser reduced inflammatory response and relatively stabilized the collagen components. However, more than 1 session of this laser treatment is required to achieve more desirable results. Furthermore, although this treatment demonstrates efficacy in preventing infection recurrence, it does not effectively inhibit relapse of pathological scars.

Surgical intervention is commonly considered the most effective method for treating infectious scars. However, conventional 2-stage surgical intervention for infectious scars, consisting of a debridement operation and subsequent wound closure operation, can be challenging and lead to complications, such as wound dehiscence and overinfection.[10,20] To overcome these limitations, we developed an enhanced ciNPWT technique following 1-stage operation. Our retrospective study demonstrated that it is a simple and effective method for promoting wound healing and preventing complications.

Mechanistically, infection and wound tension are 2 critical factors for promoting unfavorable scarring outcomes. It is noteworthy that the majority of undesirable wound tension occurs within the initial 3 months.[10] Therefore, achieving effective tension relief during the early postoperative period is imperative for optimizing the outcome of pathological scars. Conventional ciNPWT often fails to address subcutaneous tensions due to the limited negative pressure environment formed underneath the closed incision.[10,25] To address this limitation, we inserted a fluted drainage tube underneath the closed incision and foam dressing. This enables a controlled negative pressure environment to reach the wound underneath the closed incision, which not only facilitates the removal of excess fluid and promotes blood flow but is also beneficial for reducing subcutaneous tension. The 1-year follow-up results demonstrated that our enhanced ciNPWT technique reduced infection recurrence frequency and achieved a relatively lower scarring recurrence rate, compared with that of traditional surgery of 45%–100%.[10,20] These may be attributed to the radical eradication of infection and relief in subcutaneous tension during the early postoperative stage, which effectively inhibited excessive fibroproliferation and inflammation.[8,26,27]

In this study, we have successfully applied the enhanced ciNPWT following the 1-stage operation to infectious pathological scars in the thoracic, abdominal, and perineal regions. Notably, aesthetically pleasing outcomes were observed in scars with small size and complete resection, whereas larger scars that underwent palliative resection showed slightly less satisfactory aesthetic results. Additionally, possible transient hyperpigmentation and unsightly appearance may limit further application in facial regions. Furthermore, validation is needed for other areas such as extremities. Additionally, an expanded sample size, extended follow-up period, and even future prospective randomized controlled trials should be realized, if possible, to better explore the effectiveness of our enhanced ciNPWT technique.

CONCLUSIONS

The monocenter retrospective clinical study demonstrates that our enhanced ciNPWT technique following the 1-stage operation is an effective and safe method for treating patients with infectious scars that are amenable to primary closure. It promotes optimal wound healing, reduces the risk of postoperative infection, and lowers the incidence of scarring recurrence. Notably, we have not included patients with cardiopulmonary disease, immunosuppression, or several other risk factors. Whether our enhanced ciNPWT technique is safe and effective for those high-risk patients needs further validation.

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DISCLOSURES

The authors have no financial interest to declare in relation to the content of this article. This study was supported by grants from the National Natural Science Foundation of China (82372535), the Shanghai Clinical Research Center of Plastic and Reconstructive Surgery supported by the Science and Technology Commission of Shanghai Municipality (22MC1940300), and the Shanghai Municipal Key Clinical Specialty (shslczdzk00901).

ETHICAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. This study was approved by the ethics committee of our institute, Shanghai Ninth People's Hospital (SH9H-2023-T258-1).

REFERENCES

1. Sheng M, Chen Y, Li H, et al. The application of corticosteroids for pathological scar prevention and treatment: current review and update. *Burns Trauma.* 2023;11:tkad009.
2. Elsaie ML. Update on management of keloid and hypertrophic scars: a systemic review. *J Cosmet Dermatol.* 2021;20:2729–2738.
3. Li S, Ding H, Yang Y, et al. Global research status of pathological scar reported over the period 2001–2021: a 20-year bibliometric analysis. *Int Wound J.* 2023;20:1725–1738.
4. Huang C, Wu Z, Du Y, Ogawa R. The epidemiology of keloids. In: Téot L, Mustoe TA, Middelkoop E, Gauglitz GG, eds. *Textbook*

on Scar Management: State of the Art Management and Emerging Technologies. Springer; 2020;Chapter 4.
5. Ogawa R. Keloid and hypertrophic scars are the result of chronic inflammation in the reticular dermis. *Int J Mol Sci*. 2017;18:606.
6. Zhao L, Gao L, Chen X, et al. A massive mandibular keloid with severe infection: what is your treatment? *Photodiagnosis Photodyn Ther*. 2021;33:102200.
7. Nguyen TA, Feldstein SI, Shumaker PR, et al. A review of scar assessment scales. *Semin Cutan Med Surg*. 2015;34:28–36.
8. Wang ZC, Zhao WY, Cao Y, et al. The roles of inflammation in keloid and hypertrophic scars. *Front Immunol*. 2020;11:603187.
9. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids: a 2020 update of the algorithms published 10 years ago. *Plast Reconstr Surg*. 2022;149:79e–94e.
10. Min P, Zhang S, Sinaki DG, et al. Using Zhang's supertension-relieving suture technique with slowly-absorbable barbed sutures in the management of pathological scars: a multicenter retrospective study. *Burns Trauma*. 2023;11:tkad026.
11. Li K, Nicoli F, Xi WJ, et al. The 1470 nm diode laser with an intralesional fiber device: a proposed solution for the treatment of inflamed and infected keloids. *Burns Trauma*. 2019;7:5.
12. Ma QY, Yang YT, Chen ZA, et al. Laser combined with radiotherapy for keloid treatment: a novel and efficient comprehensive therapy with a lower recurrence rate. *Plast Reconstr Surg*. 2023;152:1022e–1029e.
13. Liu Y, Xu M, Wang Z, et al. The effect of incisional negative pressure wound therapy on the improvement of postoperative cosmetic suture wounds and scar hyperplasia. *Int Wound J*. 2023;20:3081–3087.
14. Groenen H, Jalalzadeh H, Buis DR, et al. Incisional negative pressure wound therapy for the prevention of surgical site infection: an up-to-date meta-analysis and trial sequential analysis. *EClinicalMedicine*. 2023;62:102105.
15. Quatman CE, Villarreal ME, Cochran A. Incisional negative pressure wound therapy following surgical repair of lower extremity fractures. *JAMA*. 2020;323:513–514.
16. Byrne M, Aly A. The surgical suture. *Aesthet Surg J*. 2019;39:S67–S72.
17. Nambi Gowri K, King MW. A review of barbed sutures—evolution, applications and clinical significance. *Bioengineering (Basel, Switzerland)*. 2023;10:419.
18. Li L, Shao Q, He W, et al. Close orthopedic surgery skin incision with combination of barbed sutures and running subcuticular suturing technique for less dermal tension concentration: a finite element analysis. *J Orthop Surg Res*. 2023;18:333.
19. Matarasso A, Ruff GL. The history of barbed sutures. *Aesthet Surg J*. 2013;33:12S–16S.
20. Burusapat C, Wanichjaroen N, Wongprakob N, et al. The effectiveness of immediate triamcinolone acetonide injection after auricular keloid surgery: a prospective randomized controlled trial. *Plast Reconstr Surg Glob Open*. 2021;9:e3729.
21. Zhao X, Shen Y. Island perforator muscle flaps for chronic osteomyelitis of the lower extremities: a retrospective analysis of 21 consecutive cases. *Plast Reconstr Surg*. 2022;150:677–687.
22. Gabriel A, Gupta S, Orgill DP. Challenges and management of surgical site occurrences. *Plast Reconstr Surg*. 2019;143:7S–10S.
23. Dadras M, Ufton D, Sogorski A, et al. Closed-incision negative-pressure wound therapy after resection of soft-tissue tumors reduces wound complications: results of a randomized trial. *Plast Reconstr Surg*. 2022;149:972e–980e.
24. Fitzpatrick RE. Treatment of inflamed hypertrophic scars using intralesional 5-FU. *Dermatol Surg*. 1999;25:224–232.
25. Mehdorn M, Jansen-Winkeln B. Modified incisional negative pressure wound therapy increases seroma evacuation: an ex vivo model. *Biomed Res Int*. 2021;2021:5846724.
26. Mascharak S, desJardins-Park HE, Davitt MF, et al. Preventing engrailed-1 activation in fibroblasts yields wound regeneration without scarring. *Science*. 2021;372:eaba2374.
27. Hosseini M, Brown J, Khosrotehrani K, et al. Skin biomechanics: a potential therapeutic intervention target to reduce scarring. *Burns Trauma*. 2022;10:tkac036.