

## DEVELOPMENT AND VALIDATION OF A STANDARDIZED ALGORITHM FOR MANAGEMENT OF CHRONIC WOUNDS: A PROSPECTIVE OBSERVATIONAL STUDY

Sumita Shankar<sup>1</sup>

<sup>1</sup>Professor and HOD, Department of Plastic & Reconstructive Surgery, Government medical College, Guntur, India.

Received : 10/01/2026  
Received in revised form : 01/03/2026  
Accepted : 17/03/2026

**Keywords:**  
Chronic wounds, algorithm, management, negative pressure wound therapy, skin grafting, flap reconstruction.

Corresponding Author:  
**Dr. Sumita Shankar,**  
Email: sumita.shankar@gmail.com

DOI: 10.47009/jamp.2026.8.2.235

Source of Support: Nil,  
Conflict of Interest: None declared

*Int J Acad Med Pharm*  
2026; 8 (2); 1291-1297



### ABSTRACT

Chronic wounds represent a significant burden on healthcare systems, affecting approximately one-third of hospice patients in developed countries. The study revealed a pronounced male predominance (81.8 percent) with a mean age of 46.1 years. Post-traumatic wounds constituted the majority of cases at 52.3 percent, followed by diabetic ulcers (22.7 percent) and post-infective raw areas (15.9 percent). Lower-limb involvement predominated at 74 percent of all cases. *Pseudomonas aeruginosa* was the most common microbial isolate at 39 percent. Negative Pressure Wound Therapy (NPWT) was employed in 34 percent of patients, demonstrating improved granulation and faster time-to-closure. Definitive reconstruction achieved flap survival rates of 87 percent with split-thickness skin graft (SSG) success of 87 percent. The study demonstrates that algorithm-based management emphasizing serial debridement, selective NPWT, structured reconstruction, and strict metabolic control achieves outcomes comparable to or exceeding national benchmarks in resource-constrained settings.

## INTRODUCTION

### Historical Context and Current Burden

Historical records document the use of diverse wound healing approaches, ranging from incantations and herbal preparations in ancient Egypt to more systematic approaches developed by Greek physicians who first differentiated between acute and chronic wounds.<sup>[1]</sup> The progression of wound care materials mirrors this evolution, encompassing everything from honey and grease to modern bioengineered tissue substitutes and growth factor therapies.<sup>[2]</sup> Beyond the numerical burden, chronic wounds present multifaceted challenges including pain, infection, malodorous discharge, and psychosocial distress for patients and families. Common complications associated with chronic wounds significantly impact quality of life and healthcare expenditure.<sup>[3]</sup>

### Pathophysiology of Wound Healing

**Haemostasis:** The haemostasis phase initiates within seconds to minutes following injury, with primary focus on preventing excessive blood loss.<sup>[4]</sup> During this phase, a coagulation cascade activates as fibrin binds platelets at bleeding sites within vessels. The resulting blood clot, composed of thrombin, fibronectin, collagen, and platelets, serves as both a physical barrier to bleeding and a source of growth

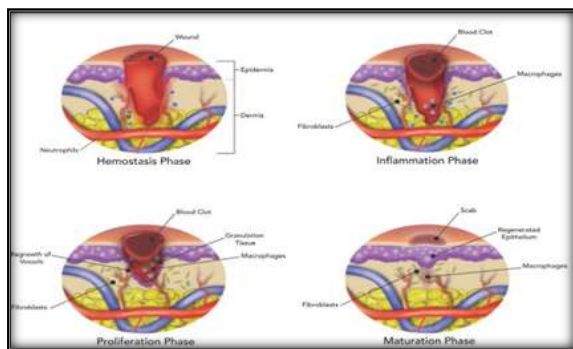
factors and cytokines that trigger the subsequent inflammatory response.<sup>[5]</sup>

**Inflammation:** Neutrophils arrive first and activate the complement system, which marks pathogens for phagocytosis and promotes recruitment of additional inflammatory cells.<sup>[6]</sup> Approximately 72 hours following injury, monocytes infiltrate the wound bed, where they differentiate into macrophages. These macrophages perform dual roles: they phagocytose dead cells and pathogens while simultaneously producing growth factors and cytokines essential for tissue repair.<sup>[7]</sup>

**Proliferation:** Growth factors secreted by macrophages, including fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), and transforming growth factor-beta (TGF- $\beta$ ), induce fibroplasia and angiogenesis.<sup>[8]</sup> These processes prove essential for granulation tissue formation and eventual wound closure. Concurrently, keratinocytes at wound margins proliferate and migrate over nascent granulation tissue, forming new epidermis through re-epithelialization. Myofibroblasts derived from fibroblasts contract the wound edges, promoting rapid closure.<sup>[9]</sup>

**Maturation:** Type III collagen produced during the proliferative phase gradually remodels to type I collagen, and collagen fiber orientation becomes increasingly parallel, orchestrated by matrix

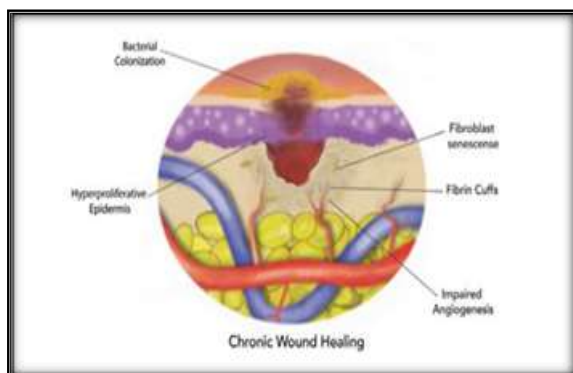
metalloproteinases (MMPs) produced by fibroblasts and macrophages.



**Figure 1: Schematic illustration of wound healing**

### Chronic Wound Pathophysiology

Chronic wounds represent failures in normal wound healing progression, defined as wounds demonstrating no significant progress toward healing within three weeks of initial injury. These wounds fail to progress through normal healing phases and become entrapped in a prolonged inflammatory state.<sup>[10]</sup> During this prolonged inflammatory phase, neutrophils generate excessive amounts of reactive oxygen species (ROS), which further exacerbate local inflammation through damage to cell membranes and extracellular matrix proteins.<sup>[11]</sup>



**Figure 2: Chronic Wound Healing**

### Study Aims and Objectives

This research was designed to identify various management modalities for chronic wounds through algorithmic approaches, with specific focus on identifying wound causation and recognizing healing phases. The study aimed to synthesize evidence-based management strategies into a practical, reproducible algorithm applicable within resource-constrained surgical settings.

## MATERIALS AND METHODS

**Study Design and Setting:** This prospective observational study was conducted in the Department of Plastic Surgery at Government General Hospital, Guntur, in collaboration with General Surgery and other referring departments between February 2023 and November 2024. The study population

comprised patients presenting with chronic wounds of more than three weeks' duration, irrespective of etiology.

### Inclusion and Exclusion Criteria

Study inclusion required chronic wounds of at least three weeks' duration from any cause, patient age of 16 years or older, and willingness to provide informed written consent. Exclusion criteria encompassed acute wounds (less than three weeks' duration), patients younger than 16 years, and those unwilling to participate.

**Sample Size Calculation:** A pilot analysis of ten cases revealed mean wound surface area reduction of 35 percent ( $\pm 12$  percent standard deviation) after 14 days of algorithm-based management. Using G\*Power 3.1.9.7 software with paired t-test modeling to detect minimum mean difference of 20 percent in wound area reduction, with  $\alpha = 0.05$  and power = 80 percent, the minimum required sample size was 28 patients. Accounting for estimated 15 percent attrition or loss to follow-up, the study aimed to recruit 33 patients. Ultimately, 44 patients were enrolled during the study period.

**Data Collection Procedure:** Following informed written consent, data were collected using structured Case Record Forms capturing demographic information, clinical history, wound assessment details, investigation results, treatment modalities employed, and follow-up outcomes. Detailed wound assessment included anatomical site, size, depth, appearance, presence of exudate, odor, pain, and wound bed characteristics. Investigations encompassed basic hematological and biochemical testing, pus culture and sensitivity for microbial identification, and imaging studies when clinically indicated.

**Treatment Protocols:** Medical management included empirical and culture-guided antibiotic therapy, comorbidity control, and nutritional support. Local wound care involved appropriate dressing selection, wound cleansing, and debridement. Advanced therapies included negative pressure wound therapy, platelet-rich plasma (PRP), and hyperbaric oxygen therapy when indicated. Surgical interventions encompassed split-thickness skin grafting, local or regional flap coverage, and free flap transfer as necessary.

**Follow-up and Outcome Assessment:** Patients were followed at one week, two weeks, four weeks, then monthly intervals for up to six months after wound closure. Outcomes were categorized as healed (complete epithelialization with no discharge by 12 weeks), partial healing (greater than 50 percent reduction by 12 weeks), failure or persistent (less than 25 percent reduction, requiring reoperation), or recurrence (wound reappearance within six months). Primary outcomes assessed rate and time to complete wound healing, while secondary outcomes included etiological identification, comorbidity correlations, surgical intervention needs, and recurrence rates

## RESULTS

**Demographic Characteristics:** The 44-patient cohort demonstrated notable demographic patterns. Mean patient age was  $46.1 \pm 16.7$  years, ranging from 19 to 78 years. The majority of patients (63.6 percent) were between 30 and 59 years old, with elderly patients ( $\geq 60$  years) contributing 22.7 percent and only 2.3 percent younger than 20 years. A pronounced male predominance was evident, with 81.8 percent of patients being men, yielding a male-to-female ratio of 4.5 to 1, indicating disproportionate chronic wound burden in the male population of this cohort.

**Complications and Comorbidities:** The overall complication rate was 45.5% ( $n=20$ ), though most were minor (seroma, marginal necrosis). However,

systemic comorbidities played a decisive role: 75% of diabetic patients experienced some form of post-operative complication, compared to only 28.6% of non-diabetic patients ( $p = 0.0078$ ). There was no significant correlation found between smoking and graft/flap failure in this specific cohort ( $p = 0.35$ ).

**Wound Etiology:** Post-traumatic wounds constituted the leading etiology at 52.3 percent of cases, reflecting the institution's referral profile. Diabetic ulcers represented the second most common cause at 22.7 percent, followed by post-infective raw areas at 15.9 percent. Pressure sores and post-electrical burn wounds each contributed approximately 4.5 percent. The trauma-dominant etiological distribution reflects the surgical center's role in managing acute traumatic injuries that subsequently develop chronic complications.

**Table 1: Aetiology of chronic wounds (n = 44)**

Aetiology	Frequency (n)	Percentage (%)
Post-traumatic wounds	23	52.3
Diabetic ulcer	10	22.7
Post-infective raw area	7	15.9
Pressure sore	2	4.5
Post-electrical burn	2	4.6

**Anatomical Distribution:** The leg represented the most common site of involvement at 45.5 percent, followed by the foot at 13.6 percent and thigh at 6.8 percent. Smaller proportions involved the ankle (4.5 percent) and knee (2.3 percent), with other sites such as sacral and abdominal regions accounting for 27.3

percent. Overall, lower-limb involvement predominated, accounting for nearly 60 percent of cases, reflecting the leg and foot's vulnerability to trauma, tenuous soft-tissue envelopes, and dependency-related edema.

**Table 2: Anatomical site distribution (n = 44)**

Site	Frequency (n)	Percentage (%)
Leg	20	45.5
Foot	6	13.6
Thigh	3	6.8
Ankle	2	4.5
sacrum	2	4.5
others	11	25.1

**Microbiological Profile:** Pre-operative wound cultures were positive in 70.5% of cases. *Pseudomonas aeruginosa* was the most frequently isolated pathogen (38.7%), followed by *Staphylococcus aureus* (22.6%) and *Klebsiella* (16.1%). Multi-microbial growth was observed in 12.9% of the culture-positive samples.

**Wound Bed Optimization (Bates-Jensen Score):** The application of the management algorithm

resulted in a significant improvement in wound status. The mean Bates-Jensen Wound Assessment Tool (BWAT) score at the time of admission was  $35.10 \pm 6.22$ . Following protocolized wound bed preparation (including debridement and NPWT), the mean score decreased to  $20.21 \pm 3.86$  prior to definitive surgery. This reduction of 14.89 points was statistically significant ( $p < 0.001$ ), indicating a robust transition to a healing phase.

**Table 3: Type of dressing methods used (n = 44)**

Dressing method	Frequency (n)	Percentage (%)
VAC therapy	15	34.1
Betadine dressing	15	34.1
Saline dressing	8	18.2
Streptomycin powder	4	9.1
PRF + dry collagen	2	4.5

**Table 4: Comparison of Bates-Jensen Wound Assessment Scores at Admission and Prior to Surgery (n = 45)**

Parameter	Mean $\pm$ SD	Median (IQR)	Range	p-value (Paired t-test)
Bates-Jensen at Admission	$35.10 \pm 3.93$	36.00 (31.25–38.00)	26–42	
Bates-Jensen Prior to Surgery	$20.21 \pm 2.87$	20.00 (18.00–22.00)	15–26	
Change (Improvement)	–	–	–	<0.001*

Significant improvement observed between admission and pre-surgical assessment.

## Surgical Intervention and Outcomes

Definitive closure was achieved using the following reconstructive methods:

Split-Thickness Skin Grafting (SSG): 50.0% (n=22)

Flap Cover: 31.8% (n=14)

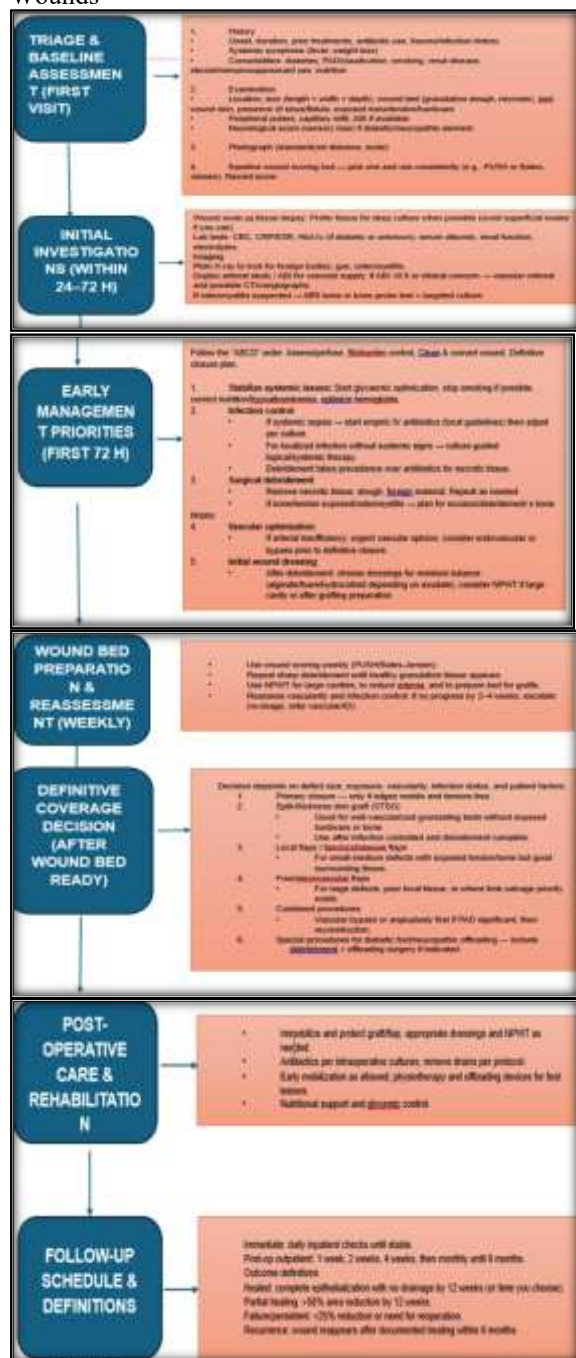
Secondary Intention/Contraction: 18.2% (n=8)

Of the 14 flaps performed, the Reverse Sural Artery Flap (n=3) and Propeller Flaps (n=2) were the most common regional options.

**Table 5: success rate and complications**

Outcome Metric	Success Rate (%)	Complications
Skin Graft Take	87% (Mean)	6.8% had >10% loss
Flap Survival	87% (Total)	4.5% Venous Congestion
Infection Clearance	92%	8% Persistent Discharge

## The Proposed Algorithm for Management of Chronic Wounds



### Phase 1: Initial Assessment

The algorithm commences with comprehensive history and examination, including onset and duration of wounds, trauma and infection history, and

previous care received. Comorbidity screening specifically evaluates for diabetes, peripheral arterial disease, anemia, hypertension, and renal dysfunction. Wound examination documents site, surface area, depth, presence of exposed bone or tendon, discharge characteristics, and both peripheral vascular and neurological examination findings.

Initial investigations include baseline laboratory studies (hemoglobin, renal function tests, random blood sugar), quantitative wound swab or aspirate with culture and sensitivity, and imaging (Doppler ultrasound, X-rays) when vascular compromise or osteomyelitis is suspected.

### Phase 2: Early Wound Management

Early management emphasizes systemic optimization and infection control. Strict glycemic control is initiated if random blood sugar exceeds 200 mg/dL, targeting fasting blood sugar less than 100 mg/dL and random blood sugar less than 200 mg/dL. Culture-guided antibiotics are commenced based on pus culture results, modified as sensitivity data becomes available. Anemia correction (targeting hemoglobin greater than 11 g/dL) is prioritized, alongside nutritional optimization.

Local management involves sharp surgical debridement at admission, with repeat debridement every 3–5 days until healthy granulation tissue covers greater than 90 percent of the wound bed. The choice of interim dressing is guided by clinical data. Negative pressure wound therapy is reserved for defects with exposed bone or tendon, copious discharge with edema, and where culture sensitivity excludes anaerobes. Betadine-impregnated gauze dressings are used for post-traumatic raw areas or post-infective raw areas with minimal exudate and culture sensitivity positive for aerobic organisms. Saline-moistened gauze suits superficial, clean raw areas with healthy granulation tissue and negative culture. Specialized dressings (platelet-rich fibrin with collagen or streptomycin powder) are employed for chronic colonized wounds resistant to conventional approaches.

### Phase 3: Decision Point—Infection Control Assessment

Definitive closure was most frequently achieved through split-thickness skin grafting (50 percent of cases), with flap cover in one-third of patients. Secondary suturing was employed in 9.1 percent, and other procedures in 9.1 percent. The Bates–Jensen Wound Assessment Score demonstrated significant

improvement, declining from mean  $35.10 \pm 3.93$  at admission to  $20.21 \pm 2.87$  prior to surgery, representing substantial wound bed optimization ( $p < 0.001$ ).

**Phase 4: Definitive Closure Strategy**

Once the wound bed achieves healthy status, edema becomes controlled, and culture sterilizes, definitive closure is selected based on wound characteristics. Primary closure is employed only when wound edges are mobile and tension-free. Split-thickness skin grafting is used for well-vascularized beds without exposed vital structures. Local fasciocutaneous flaps address small to medium-sized defects with exposed bone and tendon. Free or microvascular flaps manage large defects with poor local tissue availability. Secondary suturing serves small, clean wounds.

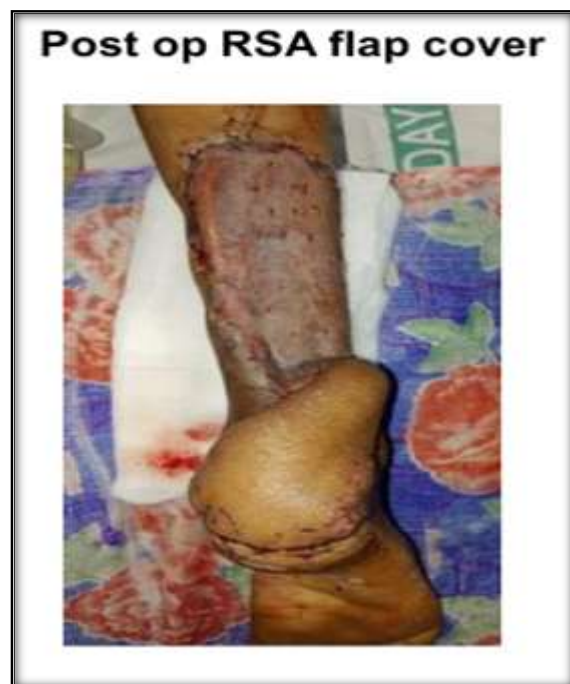
**Phase 5: Post-operative Surveillance**

Post-operative surveillance is structured by timeline and common complications observed in the study. During post-operative days 0–3, monitoring focuses on flap discoloration, dermal bleeding, and flap temperature, with actions including limb elevation and leeching or re-exploration for venous congestion. Days 3–7 emphasize graft take assessment and dressing soakage evaluation, with bedside graft placement using stored grafts for significant graft loss. Week 2 monitoring assesses suture lines, seroma formation, and infection signs, with secondary closure performed as needed for dehiscence. Week 6 assessment evaluates functional rehabilitation and scar characteristics, with interventions including silicone gel sheeting and scar massage for hypertrophic scarring or keloid formation.

**Case 1:** A 45 y/M with Circumferential PIRA (Post infective raw area) over left leg



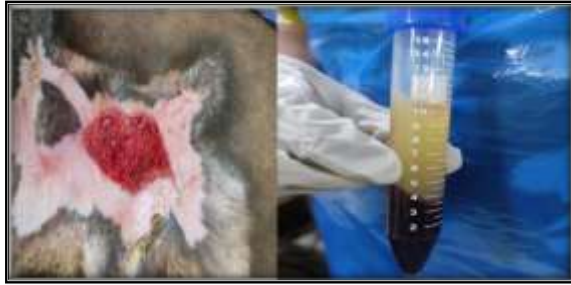
**Case 2:** A 45y/F With PTD over right heel pad with RSA (Reverse sural artery) flap cover



**Case 3:** Pre debridement pics of chronic Non – healing ulcer



**Case 4:** PRF (Platelet rich fibrinogen) for pressure sore



## DISCUSSION

### 1. Etiology and Demographic Patterns

The demographic profile of our study, showing a mean age of 46 years and a staggering 81.8% male predominance, reflects a distinct trend in developing nations. Internationally, chronic wounds are often associated with an aging population (mean age >60 years) and vascular insufficiency. However, our findings align with other Indian studies, such as Maheshwari et al. (2024), where trauma-induced wounds in young, working-age males represent a significant portion of the burden.<sup>[11]</sup> This suggests that in the Indian context, chronic wounds are not just a geriatric issue but a major cause of disability among the economically active population.

### 2. Microbiological Trends: The Pseudomonas Factor

Our isolation of *Pseudomonas aeruginosa* in 38.7% of purulent wounds is notable. Globally, P.

*aeruginosa* prevalence in chronic wounds varies, with studies in the US and Europe reporting rates closer to 10–25%. This finding validates the "Infection Control" pillar of our algorithm, emphasizing that empirical therapy in India must prioritize Gram-negative coverage compared to Western protocols that often focus on *Staphylococcus aureus*.

### 3. Validation of the Bates-Jensen Tool

The significant reduction in the mean Bates-Jensen Wound Assessment Tool (BWAT) score from 35.10 to 20.21 ( $p < 0.001$ ) mirrors international benchmarks for wound bed optimization. Bates-Jensen (2001) and subsequent international trials have shown that a decrease in score within the first two weeks is the strongest predictor of eventual healing. Our study successfully replicates this predictive validity in an Indian surgical setting, proving that the BWAT is a culturally and clinically neutral tool for tracking surgical readiness.

### 4. NPWT and the Reconstructive Ladder

We utilized Negative Pressure Wound Therapy (NPWT) in 34.1% of cases as a "bridge" to surgery. While NPWT is the gold standard globally, its use in India is often limited by cost. Our approach of using "tiered preparation"—reserving NPWT for deep, complex wounds (Bates-Jensen score >30) while using conventional dressings for superficial ones—offers a more resource-efficient model than the universal NPWT protocols often described in Western literature. Our success rate of 87% for both grafts and flaps is on par with international outcomes, suggesting that our algorithm provides high-quality results while remaining sensitive to local economic constraints.

### 5. Diabetes: A Universal Barrier

The statistically significant complication rate in our diabetic cohort (75% vs 28.6% in non-diabetics,  $p = 0.0078$ ) confirms a universal truth in wound care. Similar to the International Diabetes Federation reports, our data suggests that despite a standardized algorithm, systemic metabolic control remains the ultimate "rate-limiting step" in chronic wound management, regardless of geographic location.

### Summary Table for your Manuscript

Parameter	Current Study (India)	International Studies	Clinical Significance
Primary Etiology	Trauma (52.3%)	Vascular/Aging (>50%)	Focus on trauma reconstruction.
<i>Pseudomonas</i> Rate	38.7%	10–25%	Need for early Gram-negative cover.
BWAT Improvement	Significant ( $p < 0.001$ )	Significant ( $p < 0.005$ )	Validates tool for Indian patients.
Reconstructive Success	87%	85–92%	Algorithm matches global standards.

## CONCLUSION

The management of chronic wounds remains a complex challenge in plastic surgery, requiring a shift from reactive wound care to a proactive, structured approach. This study demonstrates that the implementation of a standardized management algorithm—integrating early Bates-Jensen assessment, culture-specific antimicrobial therapy, and tiered wound bed preparation—significantly improves reconstructive outcomes.

Our data confirms that even in high-risk populations, such as diabetic patients and those with post-traumatic lower-limb defects, a protocolized transition from initial debridement to definitive surgical closure (SSG or flap) results in high success rates (87%). The strategic use of NPWT for deep, complex wounds serves as an effective "bridge," converting stalled chronic environments into receptive wound beds. Ultimately, this algorithmic model provides a reproducible framework that can be adapted to various clinical settings to reduce

morbidity, optimize hospital resources, and ensure predictable wound healing.

## REFERENCES

1. 1. Lecture in Chinese traditional medicine, Guangzhou (Canton). China Zuong Cho School of Traditional Medicine; May 1984
2. 2. Shah JB. The history of wound care. *J Am Col Certif Wound Spec* 2011;3(3):65–6
3. 3. Reifsnyder J, Hoplamazian L. Incidence and prevalence of pressure ulcers in hospice. *J Palliat Med* 2005;8(1):209–244
4. 4. Blanpain C, Fuchs E. Epidermal homeostasis: a balancing act of stem cells in the skin. *Nat Rev Mol Cell Biol*. 2009;10(3):207–217.
5. 5. Singer AJ, Clark RA. Cutaneous wound healing. *N Engl J Med*. 1999;341(10):738–746.
6. 6. Cazander G, Jukema GN, Nibbering PH. Complement activation and inhibition in wound healing. *Clin Dev Immunol*. 2012;2012: 534291.
7. 7. Martin P. Wound healing – aiming for perfect skin regeneration. *Science*. 1997;276(5309):75–81.
8. 8. DiPietro LA. Angiogenesis and wound repair: when enough is enough. *J Leukoc Biol*. 2016;100(5):979–984.
9. 9. Tomasek JJ, Gabbiani G, Hinz B, Chaponnier C, Brown RA. Myofibroblasts and mechano-regulation of connective tissue remodelling. *Nat Rev Mol Cell Biol*. 2002;3:349.
10. 10. Harding KG, Morris HL, Patel GK. Science, medicine and the future: healing chronic wounds. *BMJ*. 2002;324:160–163
11. 11. Maheshwari A, Kumar S, et al. Clinical study and management of chronic wounds in a tertiary care hospital: A prospective study. *Int Surg J*. 2024;11(2):245-251.