To compare the efficacy of four conventional and contemporary methods of debridement i.e. surgical, autolytic, enzymatic and mechanical, in the healing of wounds of various etiology

Dr. Arun Kumar Pargi¹ (Assistant Professor), Dr. Virendra Pachole² (Senior Resident), Dr. Rinku Yadav³ (Assistant Professor)

^{1,2,3}Department of Surgery, Nandkumar Singh Chauhan Govt. Medical College, Khandwa (M.P.)

First Author: Dr. Arun Kumar Pargi Corresponding Author: Dr. RinkuYadav

Abstract:

Background & Method: This prospective study is done with an aim to compare the efficacy of four conventional and contemporary methods of debridement i.e. surgical, autolytic, enzymatic and mechanical, in the healing of wounds of various etiology. One hundred patients admitted in Department of Surgery in G.R.M.C. Gwalior, who fit the inclusion criteria were selected with acute ulcers as well as chronic non healing ulcers such as diabetic ulcers, venous ulcers, decubitus ulcers, post traumatic and post-burn wounds, skin & soft tissue infections. Thorough examination of wound was recorded and also a note of the grade of wound, swab culture sensitivity and presence of systemic infection was made at the outset.

Result: This suggests that there is not a signification difference in symptom level at presentation (p-value=0.07). This further reveals that the difference in the improvement of the symptom (pain) becomes statistically significant. This also reveals that the improvement of symptom is consistent within this group throughout the period of follow up.

Conclusion: Even though surgical debridement has by far been considered the gold standard for getting rid of necrotic tissue, it may not essentially be the best. It does remove the source of infection in the fastest way and promotes the phases of healing both proliferative and inflammatory and helps in accurate assessment of the wound but it also destroys the vital new tissue. Also it may not be safe and has complications like bleeding. It requires an extensive set-up for anaesthesia delivery and monitoring and this may be cumbersome if the procedure has to be repeated. Safer alternatives such as autolytic, mechanical and enzymatic debridement optimize the wound environment and promote healing without much technical skill. Equivocal results have been seen in autolytic and mechanical debridement in reduction in discomfort and pain. This study proves that mechanical debridement may hasten wound healing. The biggest advantage lies in the fact that these methods of debridement may be repeated as often as the dressings themselves safely and without causing the patient much discomfort

Keywords: Debridement, Wound healing, Bacterial load, Hemorrhage

Study Designed: Observational Study.

1. INTRODUCTION

Wound healing is an innate mechanism of action that works, most of the times. Optimum healing of a cutaneous wound requires a wellorchestrated integration of the complex biological and molecular events of cell migration and proliferation and of extracellular matrixdeposition and remodeling. Cellular responses to inflammatory growth factors and cytokines and to mechanical forces must be appropriate and precise. However this orderly progression of healing process is impaired in chronic wounds. Several pathogenicab normalities ranging from disease specific intrinsic flaws in blood supply, angiogenesis and material turn over to extrinsic factors due to infection and continued trauma contributes to failure to heal. Yet, despite these obstacles enhanced understanding and correlation of pathogenic factors, combined with strict adherenceto standards of care and with technological breakthroughs in biological agents is giving a new hope to the problems of impaired wound healing.

A large number of patients with skin and soft tissue infection due to road traffic accidents and other traumatic causes, surgical site sepsis are present in surgical wards of our hospital. These patients needs pecialized organized care with multi-displiniary approach. We follow a specific pattern of wound care in our setting in that order: wound cleansing, irrigation, infection drainage, wound debridement, surgical closure of wound or dressings of various types and systemic

administration of antibiotics. Out of these modalities of wound care debridement of wound is the basic necessity to induce functional process of tissue repair. In the last years many different new debridement techniques have been introduced; primarily applying physical principles and forces to promote the development from acute inflammatory phase to there parative condition.[1]

The word debridement comes from a French word, which means to remove a constraint. In clinical medicine this term was first used by Henri Le Dran (1685-1770), in the context of an incision to promote drainage and relieve of tension.[2][3]

Debridement involves removal of necrotic burden from a wound and isan important initial step in wound bed preparation. The term necrotic burden encompass necrotic material or non viable tissue exudate and high levels of bacteria.[4[5]Bacterial colonies present in necrotic tissues produce damaging proteases and collagenases, which break down important constituent of extracellular matrix and have negative effect on formation of granulation tissue and re-epithelization. Process of debridement reduces wound contamination and removes cell debris there by reducing tissue destruction and promotes healing. [6] [7] There are four methods of debridement Sharp or Surgical, Enzymatic, Autolytic and mechanical. In certain cases, use of more than one debridement methodmay be appropriate. [8] [9]

2. MATERIAL & METHOD

In this prospective study, one hundred patients admitted in Department of Surgery in G.R.M.C. Gwalior from October 2013 to September 2014, who fit the inclusion criteria were selected with acute ulcers as well as chronic non healing ulcers such as diabetic ulcers, venous ulcers, decubitus ulcers, post traumatic and post-burn wounds, skin & soft tissue infections. Thorough examination of wound was recorded and also a note of the grade of wound, swab culture sensitivity and presence of systemic infection was made at the outset.

They were randomly divided in four groups each matched for age, sex and other comorbid conditions, refer Appendix III.

Group I

We allotted 25 patients who were suffering from wounds of various etiologies in this group and they were subjected to surgical (sharp) debridement. These wounds were managed with

VOL12,ISSUE06,2021 ISSN:0975-3583,0976-2833

surgicaldebridement under total intravenous anesthesia, regional or local blocks depending on severity of infection, site and co-morbidity status. This was followed by routine conventional betadine-normal saline

PATIENT SELECTION INCLUSION CRITERIA

All acute and chronic ulcers in stage A and Stage B (University of Texas) were considered as a part of the study. These also included long standing ulcers >Imonth duration, which were usually post traumatic (Post RTA, Post Thermal injuries) or as a sequel to necrotizing fascitis. Burn wounds, bed sores, diabetic foot, arterial ulcers, venous ulcers, post operatively dehised wounds and amputation stumps with raw areas were all included in the study group subject to them giving due consent to be a part of the study.

For simplicity of observation, assessment and calculations, solitary ulcers, dehisced abdominal wounds, ulcers on a limb incontinuation, circumferential ulcers on the extremities were preferably selected to be studied.

For chest and abdominal wounds the criteria accepted was size more than 2x2cm and less than 10x10cm. However, for limb wounds and cases of extensive trauma with or without skin loss, the total area may have exceeded this limit and these wounds were followed up in the same fashion for assessing healing and wound contraction.

EXCLUSION CRITERIA

Multiple ulcers

Malignant ulcers

Pregnant women, Paediatric and mentally unstable persons.

People with known allergy to honey/honeybee/pollen.

Ischemic ulcers (Stage C & Stage D according to University of Texas classification of ulcers)

3. RESULTS

Dov	Discomfort	Discomfort	Discomfort	Discomfort
Day	Disconnort	Disconnort	Disconnort	Disconnort
	Scale	Scale	Scale	Scale
	(Mean±SD)	(Mean±SD)	(Mean±SD)	(Mean±SD)
	Group I	Group II	Group III	Group IV
Day -1	9.44±1.1	9.68±0.74	10±0	9.68±0.74
Day -3	7.54±1.19	7.6±1	8.24±1.05	7.76±1.2
Day -7	5.8±1.28	5.76±1.33	7.52±0.87	6.64±1.8
Day -14	3.6±1.49	3.68±1.49	5.6±0.81	4.56±2.48
Day -21	1.36±1.49	1.76±0.87	4±1	3.12±3.00
Day -28	0.04±0.81	0.48±1.04	2.16±0.8	1.68±2.21
Day -35	00	0.08±0.4	0.56±0.91	0.8±1.91

Tabla	No	01.	
таис	LIU.	VI.	

Table No. 02: J	mprovement of S	ymptoms of	pain at day-01
-----------------	------------------------	------------	----------------

Variation	SS	Df	MS	F Statistics
Between Groups	9.401	3	3.134	2.429
Within Groups	122.558	95	1.290	
Total	131.960	98		
P Value	0.070			

Table No. 03. Improvement of Symptoms of pair at day-35				
Variation	SS	Df	MS	F Statistics
Between Groups	15.465	03	5.155	4.082
Within Groups	116.191	92	1.263	
Total	131.656	95		
P Value	0.009			

 Table No. 03: Improvement of Symptoms of pain at day-35

This suggests that there is not a signification difference in symptom level at presentation (p-value=0.07). This further reveals that the difference in the improvement of the symptom (pain) becomes statistically significant. This also reveals that the improvement of symptom is consistent within this group throughout the period of follow up.

4. **DISCUSSION**

The term debridement comes from the French debrides, meaning to unbridle. It was probably first used as a medical term by surgeons working several hundred years ago in war zones, who recognized that grossly contaminated soft tissue wounds had a better chance of healing(and the soldier surviving) if the affected tissue was surgically removed to reveal a healthy bleeding wound surface[12]. When necrotic or foreign material is present in a wound, sharp or surgical debridement can reduce the risk of infection and sepsis and aid wound healing. Several studies have been conducted to compare shar pdebridement with enzymatic/ autolytic/ mechanical debridement as byfar it has been considered gold standard of debridement[13].

Why debride?

An ulcer or open wound cannot be properly assessed until all thede-vitalized tissue is removed. Dead or foreign material in a woundalso adds to the risk of infection and sepsis and inhibits wound healing.

A number of mechanisms are involved: Dead tissue acts as a medium for bacterial growth, particularlyanaerobes such as Bacteroides species and gas gangrene caused by Clostridium perfringens in military surgical practice[14].

Excessive inflammatory response, which results from the presence of necrotic or foreign material, adds to the systemic release of cytokines such as tumour necrosis factor and interleukins, which promote aseptic response[15].

Necrotict issues retard wound contraction, the principle contribution to wound closure when wounds are left to heal by secondary intention.

It can sometimes be difficult to determine whether the tissue covering a wound is physiological, such as a scab, or a pathologicaleschar, which is having a negative impact on healing[16]. Attempts to aid clinical recognition have included the injection of supravital dyes, tissue oximetry, Doppler techniques, and even biopsy. Gangrenous, necrotic, is chaemic and devitalized tissue all need to be removed by debridement.

Evidence for debridernent

Although it is widely accepted that wound debridement is necessary for optimal wound healing, evidence for the effectiveness of different methods of debridement from randomized

controlled trials is lacking and methods of measurement are poorly developed. If dressings or enzymatic agents are to be compared with surgical and

Surgical (Sharp) Debridement:

It is the fastest and efficacious way to remove debris and necrotictissue from the wound bed. Sharp surgical debridement of the hyperkeratotic rim and ulcer base to bleeding is the optimal method and converts chronic non-healing ulcer into the acute healing wound[10].

Surgical debridement converts a chronic wound into an acute wound and gets rid of the necrotic tissue source of infection in the most rapid way but may not always be the safest.

Enzymatic Debridement

Enzymatic debridement is the most selective method of debridement employing the use of manufactured proteolyticenzymes[17].

When these are applied directly onto the wound surface, they worktogether with naturally occurring enzymes to degrade necrotic tissue. Phagocytic cells (such as macrophages) and proteolytic enzymes in the wound bed[18], liquefy and separate necrotic tissue and eschar from healthy tissue. Wound dressings, which maintain a moist wound bed, can provide an optimal environment for debridement, as they allow the phagocytic cells to liquefy necrotic

Autolytic debridement:

Autolytic debridement is a process, which to some extent occursnaturally in all wounds. Moist interactive dressings (Hydrogels, alginates, transparent films hydrocolloids) maintains moist wound bed[11]. Scoring of eschar with a scalpel in a grid pattern helps eschar removal Autolytic DebridementAutolytic debridement is a process, which to some extent, occursnaturally in all wounds.

Mechanical Debridement

Mechanical debridement is a nonselective, physical method of removing necrotic tissue and debris from a wound using mechanical force. This debridement method is generally easy to perform and is more rapid than autolytic and enzymatic debridement. However, this nonselective method can damage healthy granulation tissue both in the wound bed and at the margins of the wound thus causing significant discomfort to the patient. Despite these disadvantages, there are a number of mechanical debridement methods that are in use.

Wet-to-dry dressings are the simplest method of mechanical debridement, but due to the frequent dressing changes, it can require considerable nursing time and hence is costly. Wet gauze dressings are placed onto the wound bed and allowed to dry, trapping the necrotic debris within the gauze.

Pressurized irrigation involves applying streams of water, delivered at either high or low pressure, to wash away bacteria, foreign matter, and necrotic tissue from the wound. However, if the pressure is too great, there may be a risk of forcing bacteria and debris deeper into the wound or damaging viable tissue. Whirlpool therapy uses powered irrigation and can be very effective at loosening and removing surface wound debris, bacteria, necrotic tissue, and exudate from the wound.

Ultrasound treatment has been used to remove necrotic tissue and has been shown to effectively debride wounds and reduce infection caused by bacteria. Vacuum-assisted closure is a noninvasive form of mechanical or physical debridement that exposes the wound bed to negative pressure by way of a closed system. It helps healing of chronic wounds by minimizing exudate and slough in the wound bed, reducing tissue edema, increasing peripheral blood flow, improving local oxygenation and promoting angiogenesis.

5. CONCLUSION

Even though surgical debridement has by far been considered the gold standard for getting rid of necrotic tissue, it may not essentially be the best. It does remove the source of infection in the fastest way and promotes the phases of healing both proliferative and inflammatory and helps in accurate assessment of the wound but it also destroys the vital new tissue. Also it may not be safe and has complications like bleeding. It requires an extensive set-up for anaesthesia delivery and monitoring and this may be cumbersome if the procedure has to be repeated. Also a patient's comorbid status maynot allow this.

Safer alternatives such as autolytic, mechanical and enzymaticdebridement optimize the wound environment and promote healing without much technical skill. Equivocal results have been seen inautolytic and mechanical debridement in reduction in discomfort and pain. This study proves that mechanical debridement may hasten wound healing. The biggest advantage lies in the fact that these methods of debridement may be repeated as often as the dressings themselves safely and without causing the patient much discomfort

6. REFERENCES

- [1] Vowden KR, Vowden P. Wound debridement, Part 2: Sharptechniques. J Wound Care. 1999; 8(6): 291-294.
- [2] Vowden KR, Vowden P. Wound debridement, Part 1: Non-sharptechniques. J Wound Care. 1999: 8(5): 237-240.
- [3] Bennett NT, Schultz GS. Growth factors and wound healing:Biochemical properties of growth factors and their receptors. AmJ Surg. 1993: 165:728737.
- [4] Bennett NT, Schultz GS. Growth factors and wound healing:Part 11. Role in normal and chronic wound healing, Am J Surg.1993; 166:74-81.
- [5] Martin P, Hopkinson-Woolley J, McCluskey J. Growth factorsand cutaneous wound repair. Prog Growth Factor Res. 1992;4:25-44.
- [6] Yager DR, Nwomeh BC. The proteolytic environment of chronicwounds. Wound Repair Regen. 1999; 7:433-41.
- [7] Abbas AK, Lichtman AH, Pober JS. Effector mechanisms ofcell-mediated immunity. In: Abbas AK, Lichtman AH, Pober JS(eds). Cellular and Molecular Immunology, Fourth Edition.Philadelphia, PA: WB Saunders Company. 2000;(13):291-308.
- [8] Overall CM. Regulation of tissue inhibitor of matrix metalloproteinase expression. In: Greenwald RA, Golub LM (eds).Inhibition of matrix metal loproteinases: Therapeutic potential.Ann NY Acad Sci. 1994; 732:5164.
- [9] Lazarus GS, Cooper DM, Knighton DR, et al. Definitions and guidelines for assessment of wounds and evaluation of healing. Arch Dermatol. 1994;130(4):489-493.
- [10] BucaloB. Eaglstein WH, FalangaV. Inhibitionof cellproliferation by chronic wound fluid. Wound Repair Regen.1993; 1: 181-186.

- [11] Harris IR, Yee KC, Walters CE, et al. Cytokine and proteaselevels in healing and nonhealing chronic venous leg ulcers.ExperDermatol. 1995; 4:342-349.
- [12] SalimAS. The role of oxygen-derived free radicals in themanagement of venous (varicose) ulceration: A new approach.World J Surg. 1991;15(2):264-269.
- [13] 13.Yager DR, Zhang LY, Liang HX, et al. Wound fluids fromhuman pressure ulcers contain elevated matrix metal lo proteinase levels and activity compared to surgical wound fluids. JInvest Dermatol. 1996; 107(5):743748.
- [14] Wlaschek M, Peus D, Achterberg V, et al. Protease inhibitorsprotect growth factor activity in chronic wounds.Br J Dermatol.1997; 137(4):646.
- [15] Agren MS, Steenfos HH, Dabelsteen S, et al. Proliferation andmitogenic response to PDGF-BB of fibroblasts isolated fromchronic leg ulcers is ulcer-age dependent. J Invest Dermatol.1999; 112:463-469.
- [16] Sibbald RG, Williamson D, Orsted HL, et al. Preparing thewound beddebridement, bacterial balance, and moisturebalance. Ost Wound Manag. 2000;46:14-35.
- [17] Falanga V, Classifications for wound bed preparation and stimulation of chronic wounds. Wound Repair Regen. 2000a;8:347-352
- [18] Thomas S. Sterile maggots and the preparation of the woundbed. In: Cherry GW, Harding KG, Ryan TJ (eds). Wound bed preparation. Proceedings of a symposium sponsored by the European Tissue Repair Society; 2000 Nov 24-25; Oxford UK. International Congress and Symposium Series 250. RoyalSociety of Medicine Press Limited. 2001:59-66.