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## Clinical profile of patients with non-healing ulcer at a tertiary care hospital

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### Abstract

The Greeks belief of dry healing came from Hippocrates, at a time when the only function of dressings was thought to be the protection of the wound from injury. During the fourteenth century, with the widespread use of gunpowder and the increasing frequency of bullet wounds, there was an increased need for surgeons assuming an aggressive posture, which was often done at the expense of aseptic precautions. The study was estimated to include 50 patients who present with non-healing ulcers at hospital with purposive sampling technique. The main etiology in both groups was traumatic ulcers followed post infective raw areas.

**Keywords:** Non-healing ulcer, traumatic ulcers, post infective raw areas

### Introduction

Historical aspects of wound healing: The history of wound healing is as old as the history of mankind <sup>[1]</sup>. The earliest medical writings deal extensively with wound care. Seven of the 48 case reports included in the Edwin Smith Papyrus (1700 BC) describe wounds and their management. Empirically, the ancient physicians of Egypt, Greece, India and Europe developed gentle methods of treating wounds by removing foreign bodies, suturing, covering wounds with clean materials, and protecting injured tissue from corrosive agents <sup>[1]</sup>.

The Greeks belief of dry healing came from Hippocrates, at a time when the only function of dressings was thought to be the protection of the wound from injury. During the fourteenth century, with the widespread use of gunpowder and the increasing frequency of bullet wounds, there was an increased need for surgeons assuming an aggressive posture, which was often done at the expense of aseptic precautions. Examples included applications of burning oil, scalding water, wine, turpentine, feathers, sugar, clay, bismuth, milk of magnesia to wounds. However, none of these have proven efficacy based on sound scientific studies. The modern era of gentle wound care started in the mid-sixteenth century, when Ambroise Pare, the great French army surgeon, who during the Battle of Villaine, applied milder agents like digestive solution of egg yolk, rose oil, honey and turpentine to amputation stumps with dramatic results. John Hunter, William Stewart Halsted, Alexis Carrel and many other great clinical biologists demonstrated that minimizing tissue injury produces rapid and effective healing leading to the “minimal interference” concept of wound care <sup>[2, 3]</sup>. If the surgeon can remove all impediments, normal wound healing processes will produce the best possible result. The work of Joseph Lister and Louis Pasteur established a sound basis for the management of infection by identifying the cause and developing methods for preventing it. Joseph Lister advocated cleanliness in the hospital, the frequent use of soap and water on wounds and carbolic acid dressings of contaminated wounds <sup>[4]</sup>. Later Semmelweis, Ehrlich, Fleming, and Florey also realized that bacteria were pathogens. Control of bacteria by asepsis, antiseptics and antimicrobials heralded a new era in wound management. World War I resulted in rapid discoveries surrounding the care of wounds, the foremost among these being the use of extensive debridement. Antoine Depage, a Belgian Surgeon, was largely responsible for the development and proper use of debridement. Depage’s philosophy was that all war wounds were most likely to be infected and therefore should be debrided <sup>[5]</sup>.

In recent years, a large amount of research has been conducted on the pathophysiology of leg ulcers. Although the immediate cause of an ulcer is often traumatic, the underlying cause of the condition in people from developed countries tends to be pathological changes in the vascular system of the leg, which are often complex and difficult to characterize. In developing countries,

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the condition is more commonly associated with trauma and infection [6].

**Methodology**

The study was estimated to include 50 patients who present with non-healing ulcers at hospital with purposive sampling technique.

- A complete detailed history and physical evaluation, relevant blood investigations, radiological investigations will be done.
- Age of the patient. Symptoms and their duration, Past history, Complete physical examination, Laboratory investigations (including specific investigations like Pus cultures will be done at regular intervals), Radiological investigations wherever necessary, Treatment given.
- Data was analysed by using following parameters- Discharge, granulation tissue formation, slough, microbiological evaluation on day0, day5, day10.

**Inclusion criteria**

All patients above 20 yrs who are diagnosed to have non-healing ulcers

**Exclusion criteria**

Patients receiving corticosteroids, immunosuppressive agents, radiation, or chemotherapy within one month prior to entry into study were excluded. Malignant ulcers and X-rays showing features of osteomyelitis.

**Results**

The 50 patients admitted for the study were divided into two equal and comparable groups. Patients subjected to Silver alginate interactive dressings were classified under Group I and those who underwent conventional Betadine wound dressings were classified as Group II. The patient’s characteristics of the two groups were well matched as given in the table below

**Table 1:** Sex distribution

	Group I	Group II
No. of patients	25	25
Range of age (years)	20-82	20-85
Male-Female ratio (M:F)	18:7	16:9

**Table 2:** Age wise distribution of patients

Age	Group I	Group II
21-40	8	5
41-60	8	11
61-80	8	8
>80	1	1

The main etiology in both groups was traumatic ulcers followed post infective raw areas

**Table 3:** Etiology of ulcers

	PIRA	Traumatic ulcers	Diabetic ulcers	Venous ulcers
Group I	10	12	2	1
Group II	11	13	1	0

**Table 4:** Wound at day 0

Wound at day 0	Slough		Type of discharge		Granulation tissue	
	Present	Absent	Serous	Purulent	Present	Absent
Group 1	14	11	13	12	10	15
Group 2	17	8	11	14	12	13

**Discussion**

Antiseptics are agents that kill or inhibit microorganisms on living tissue. Providone- iodine and hydrogen peroxide are still very commonly used today. But have been found to have only limited value in wound care today.

Povidone-iodine is very widely used in wound care. *In vitro* studies have shown that povidone- iodine, unless highly diluted, is toxic to most cell types implicated in the healing process. Because povidone-iodine is water soluble, diluting it actually releases free iodine into the tissue. In certain patients in which the primary goal is not wound closure (palliative wound care), povidone-iodine can be very effective at drying the eschar thus inhibiting the development of wet gangrene [7].

Hydrogen peroxide 3% solution is also commonly used today. It cleanses the wound through its release of oxygen. It has been shown to delay wound healing by 8% compared with untreated. However, in patients who require at home wound care and hygiene is a concern, it may be worthwhile to give in to the slight delay and use 3% hydrogen peroxide to cleanse the wound before dressing application. If hydrogen peroxide is diluted to 0.3% its effectiveness against microorganisms is reduced.

Chronic wound fluid is biochemically distinct from acute wound fluid: it slows down, or even blocks, the proliferation of cells such as keratinocytes, fibroblasts, and endothelial cells and has a detrimental effect on wound healing [34]. Pappas *et al.* Demonstrated that dermal fibroblasts cultured from the edges of chronic venous leg ulcers grew more slowly than fibroblasts from healthy skin in the same patient. Cells at the wound margin appeared senescent, that is, with loss of proliferative capacity,

and were larger and less responsive to growth factors. Dermal fibroblasts produce matrix proteins such as fibronectin, integrins, collagen, and vitronectin to form a basal lamina over which keratinocytes migrate. Laminin, on the other hand, a component of the basement membrane, inhibits keratinocyte migration. While a moist environment is conducive to wound healing, chronic wound fluid from leg ulcers contains extensively degraded vitronectin and fibronectin, which may prevent cell adhesion. Other studies have shown that chronic wound fluid may inhibit proliferation of fibroblasts. Chronic wound fluid contains lower amounts of platelet- derived growth factor-like peptides, interleukin-6 (IL-6) and TGF- $\alpha$  and TGF- $\beta$  that induce cell proliferation. The growth inhibitory effect of chronic wound fluid clearly must be overcome to stimulate wound healing and tissue regeneration. In chronic wound fluid there is a very low level of glucose and heightened proteolytic activity, both important factors in impaired epithelialization and healing. The higher concentration of MMPs and serine proteinases in chronic wound fluid result in chronic tissue turnover, leading to the breakdown or corruption of matrix material essential for reepithelialization, and hence to failed wound closure. It is also known that macromolecules in the wound fluid can bind growth factors, making them unavailable to the regeneration process [8]. Proteases can also degrade growth factors and cytokines essential for wound healing. Recently, measurements of MMPs and their natural inhibitors, tissue inhibitor of metalloproteinases (TIMPs) in fluid from chronic wounds showed there was a close correlation between high ratios of TIMP/MMP-9 and healing of pressure ulcers. The

elevated levels of inflammatory cytokines and proteases, along with low levels of mitogenic activity and poor response to cells in chronic wounds, led to the concept that the molecular environment of chronic wounds must be rebalanced to levels seen in acute healing wounds. Silver alginates, which form a gel upon contact, promoting moist interactive healing, are ideal for exudative and infected wounds. Otterlei *et al.* Compared the ability of different alginates to stimulate macrophage cells to produce TNF- $\alpha$ , interleukin-1, and interleukin-6. They reported that high M alginates were approximately 10 times more potent in inducing cytokine production than high G alginates and therefore proposed that mannuronic acid residues are the active cytokine- inducers in alginates, which gives a high gelling property for autolytic debridement, and others have a high galuronic acid content, which provides good fiber integrity for packing sinuses. A treatment of high M alginate with C-5 epimerase, which converts b-D- mannuronic acid into a-L- guluronic acid, results in a loss of TNF-inducingability<sup>[9]</sup>. Postdebridement, they can donate calcium, facilitating hemostasis, and accept sodium, converting the calcium alginate fiber to a sodium alginate hydrogel. No crust is formed and the wound can progress from the inflammatory to the proliferative stage<sup>[10]</sup>.

### Conclusion

The main etiology in both groups was traumatic ulcers followed post infective raw areas.

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