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HEALING OF PERIODONTAL WOUNDS - A REVIEW

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Abstract:

A number of periodontal treatments have been used to regenerate periodontal tissues since periodontal disease is a serious public health concern. Most periodontal operations include invasive techniques such as excision of the gingiva or alveolar bone. The periodontium is a complex system made up of specialised tissues that support the teeth. The healing process of periodontal disease is nearly scar-free, and it differs somewhat from cutaneous wound healing. It also resembles foetal recovery. This review article provides a general overview of periodontal wound healing.

Keywords: Periodontium, Periodontal tissue regeneration, wound healing

Introduction:

The periodontium acts as a supporting apparatus for the teeth and is a complex structureconsisting of soft and hard tissues. The main functions of the periodontium are to ensure thatthe teeth are attached to the bones; to protect the nerves, blood vessels, and teeth from injuries;and to provide a barrier to the underlying structures from the oral microbiome.[1] Soft tissueincludes the gingiva, mucosa, and periodontal ligament (PDL), and hard tissue includes thecementum and the alveolar bone.

Periodontal tissues form a complex dentogingival junction, crucial for bone and ligament preservation. Chronic inflammation in periodontal diseases can lose this junction's structure, necessitating the re-establishment of this junction. Conventional periodontal therapy often involves instrumentation in the inflamed dentogingival complex, causing wounding of inflamed tissues. The outcome depends on wound healing events.Periodontal wounds have a mineralized tissue interface, similar to other body wounds. Following surgical procedures, tissues represent surgically wounded sites, initiating a cascade of cellular and molecular events for wound repair.Advances in periodontal tissue biology have led to surgical procedures for regeneration of lost periodontal tissues.

Wound healing begins with temporary repair, followed by inflammation, fibroblasts, endothelial cells, and epithelial cells. The healing tissue matrix matures, leading to contraction or scarring, with these phases overlapping over time.

Historical Background

Wound healing has evolved from magical incantations to a systematic text from Sushruta, Hippocrates, and Celsus. Ancient Indian, Chinese, Korean, Egyptian, and African healers all focused on wound healing due to frequent battles. Sushruta Samhita contains chapters on healing wounds, over 100 plants, and procedures for clean wounds and healing keloids.[2]

Periodontal wound

According to **Carranza in 1993** The wound margins are not two opposing vascular gingival

margins but comprise the rigid nonvascular mineralized tooth surface, on the one hand, and the connective tissue and epithelium of the gingival flap, on the other. The periodontal wound also includes tissue resources from the alveolar bone and the periodontal ligament. Clot formation at the interface between the tooth and a gingival flap is initiated as blood elements are imposed onto the root surface during surgery and at wound closure in a seemingly random manner.

Phases of wound healing:

Haemostasis

Haemostasis occurs after an injury, where platelets activate, adhere, and aggregate at the injury site. They detect extravascular collagen through integrin receptors, release mediators and adhesive glycoproteins, and aggregate. As platelet aggregation progresses, clotting factors are released, forming a fibrin clot as a provisional matrix.[3]

Inflammation

Inflammation, the next stage of wound healing, occurs within 24 hours after injury and can last up to two weeks in normal wounds and longer in chronic non-healing wounds. Key cells during this phase cleanse the wound, release pro-inflammatory cytokines and growth factors, and recruit fibroblasts and epithelial cells for healing.

Neutrophils

Neutrophils are the first inflammatory cells to respond to the soluble mediators released by platelets and the coagulation cascade. They serve as the first line of defence against infection by phagocytozing and killing bacteria, and by removing foreign materials and devitalized

tissue. During the process of extravasation of inflammatory cells into a wound, important interactions occur between adhesion molecules (selectins, cell adhesion molecules (CAMs) and cadherins) and receptors (integrins) that are associated with the plasma membranes of circulating leucocytes and vascular endothelial cells.[4]

Macrophages

Macrophages begin as circulating monocytesthat are attracted to the wound site beginning about 24 hours after injury. They extravasate bythe mechanism described for neutrophils, and are stimulated to differentiate into activatedtissue macrophages in response to chemokines, cytokines, growth factors and solublefragments of extracellular matrix components produced by proteolytic degradation of collagenand fibronectin.[5]

Proliferative phase

The proliferative phase of healing involves replacing the provisional fibrin matrix with a new collagen-based one, angiogenesis, granulation tissue formation, and epithelialization, with fibroblasts being key cells in this process.

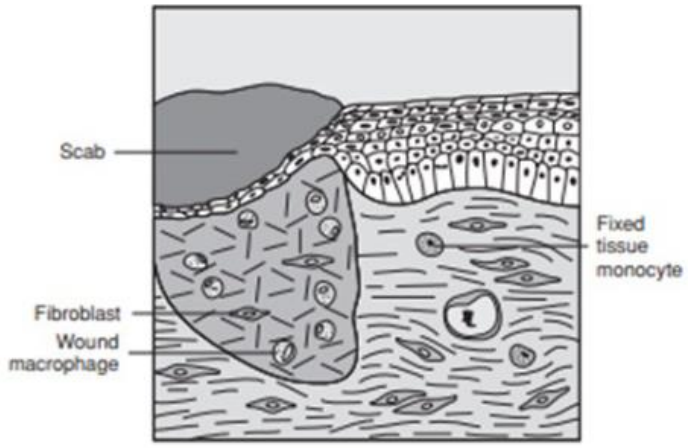

Figure 1. Proliferation Phase

Angiogenesis

The process of angiogenesis is stimulated by local factors in the microenvironment, includinglow oxygen tension, low pH and high lactate levels.[6] Also, certain soluble mediators are potentangiogenic signals for endothelial cells.

Epithelialization

Epithelialization is the process where epithelial cells around the margin of the wound or inresidual skin appendages such as hair follicles and sebaceous glands lose contact

inhibition.Then by the process of epiboly begin to migrate into the wound area. Epithelialization is a multi-step process that involves epithelial cell detachment, and change intheir internal structure, migration, proliferation and differentiation.[7]

Remodelling

Remodelling is the final phase of the healing process in which the granulation tissue maturesinto scar, and tissue tensile strength is increased (Figure.2) The maturation of granulation tissuealso involves a reduction in the number of capillaries via aggregation into larger vessels.Remodelling of the extracellular matrix proteins occurs through the actions of several differentclasses of proteolytic enzymes produced by cells in the wound bed at different times during thehealing process.

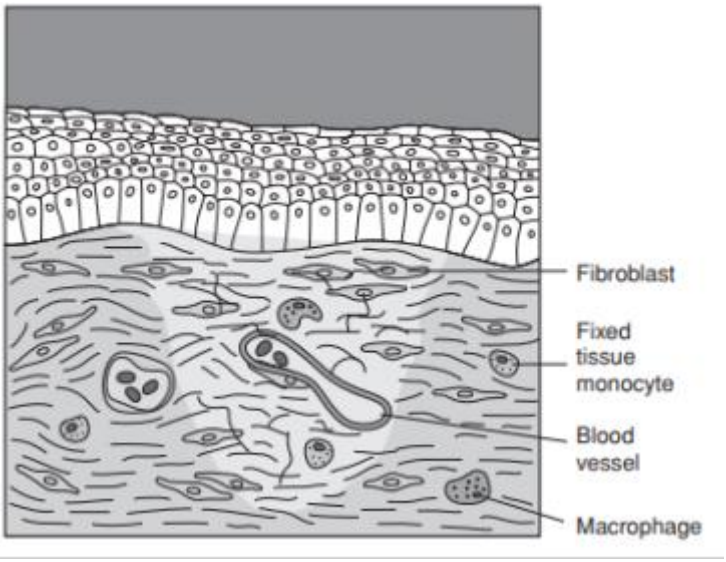

Figure. 2 Remodelling phase.

Types of cells involved in wound Healing

Inflammation in physiological wound repair: cell lineages, functions, and Mediators

Tissue injury causes the immediate onset of acute inflammation. It has long been consideredthat the inflammatory response is instrumental to supplying growth factor and cytokine signalsthat orchestrate the cell and tissue movements necessary for repair. In various experimentalanimal models and human skin wounds, it has been demonstrated that the inflammatoryresponse during normal healing is characterized by spatially and temporally changing patternsof various leukocyte subsets. The well-defined chronology of these events is essential foroptimal repair.

PMNs.- Adhesion molecules which are crucial for neutrophil diapedesis include endothelialP- and E-selectins as well as the ICAM1, -2. These adhesins interact with integrins present at the cells surface of neutrophils including CD11a/CD18 (LFA-1), CD11b/CD18 (MAC-1), CD11c/CD18 (gp150, 95), and CD11d/CD8.Chemokines like IL-8, growth-related oncogene-a, and MCP-1 are crucial for neutrophil recruitment during wound repair, while bacterial products like lipopolysaccharides can accelerate neutrophil locomotion.

Monocytes/macrophages:-

Whereas theextravasation of blood PMN is primarily regulated by the CD11/CD18 complex and ICAMs,emigration of blood monocytes into the wound is in addition regulated by the interaction of thevery late antigen-4(a4b1 integrin) and endothelial vascular cell adhesion molecule-1 (Issekutz.Macrophage depletion using antisera resulted in a significant delay of healing More recent studies have supported and extended these observations. Double P- and E-selectin-deficient mice deficient in b-1,4-galactosyltransferase, which glycosylates the P- and E-selectins.[8]

Mast cells:- Mast cells are an additional leukocyte subset present in the skin and they are animportant source of a variety of proinflammatory mediators and cytokines that can promoteinflammation and vascular changes. Therefore, they are considered to be involved in tissuerepair. Following injury residential mast cells degranulate within hours and thus may becomeless apparent. Mast cell levels return to normal around 48 hours post-injury, and then increasein number as tissue repair proceeds Analysis on the impact of mast cell deficiency in mice has been contradictory..

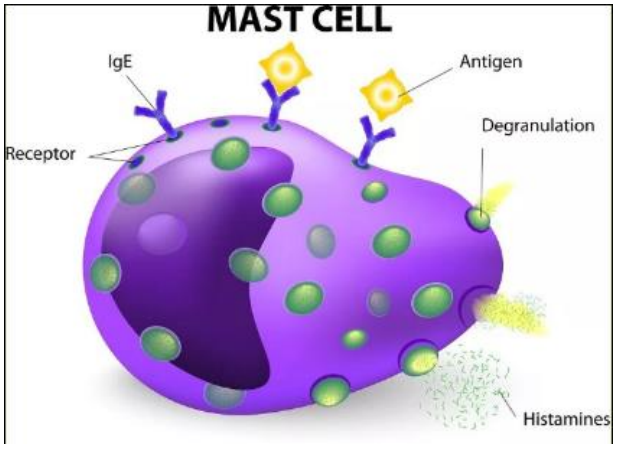

Figure3. Mast Cell

Factors affecting wound healing

LOCAL FACTORS

Healing after gingival and periodontal surgery can be delayed and altered by numerous local factors. Some of these factors include -

1) Infection

2) Poor blood supply

3) Foreign bodies

4) Movement

5) Ionizing radiation

SYSTEMIC FACTORS

1. Age

2. Nutrition

3. Glucocorticoids

4. Un-controlled diabetics

5. Hematological abnormalities

Age- Wound healing is rapid in young and somewhat slow in aged and debilitated people due to poor blood supply to the injured area in the latter.

Nutrition- Deficiency of constituents like protein, vitamin C (scurvy) and zinc delays the wound healing.

Systemic infection- delays wound healing.

Administration of Glucocorticoids has anti-inflammatory effect

Uncontrolled diabetics are more prone to develop infections and hence delay in healing.

Wound healing following various periodontal Therapies

Monitoring of wound healing (WH) following periodontal surgery is of importance. But it ismore important to know the term wound or injury which means disturbance of the anatomicafunction and structure in any part of body,[9] whereas healing is the body reaction to trauma inorder to repair normal function and structure. On the basis of the nature of wound, soft tissueloss and presence of infection, the wound healing is classified into three general categories:primary, secondary and third intention healing.[10]

Primary intention of WH has following features: clean and uninfected; surgically incised;without abundant loss of tissue and cells ; and margins of wound are closed by sutures.

Secondary WH has following features: large tissue defect which is open, sometimes infected;abundant loss of tissues and cells; and the wound is left open not closed by sutures.[11]

Moreover, tertiary intention occurs when there is considerable loss of tissue, healing occurs by contraction of wound edges and Granulation Tissue (GT) formation.

Outcome of Periodontal Wound Healing

Regeneration, repair and new attachment are the aspects of healing that have a special bearing on the outcome of periodontal treatment [12]

 Regeneration: It is biological process by which the function and architecture of lost tissues restored completely by formation of new cementum, periodontal ligament and alveolar bone.

 Repair: It is healing of tissues in which the lost tissues are not completely restored.

 New Attachment: Reunion of CT with the surface of root that is exposed pathologically.

 Reattachment: Reunion of CT and surface of root that is separated by injury or incision.

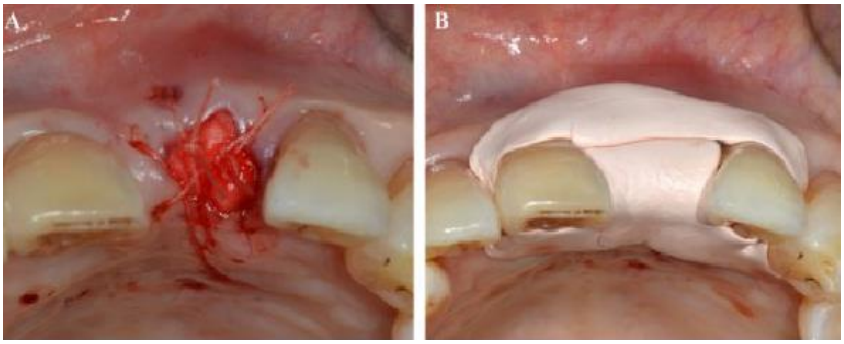

Figure 4. Periodontal dressing application. (A) After tooth extraction, collagen material wasapplied to the extraction socket. (B) Coe-Pak was applied to the surgical site to protect the wound.

Clinical Requirements for effective healing after Periodontal Surgery

 The application of initial therapy prior to surgical intervention

 The selection of surgical approach specific for the cure of the particular inflammatory lesion

 The type of tissue environment that exists after surgery

 The degree of fibrosis of gingiva prior to and after surgery

 Method by which the surgical wound is protected in the postoperative period

 The maintenance of the dentition and the periodontium by the patient and the dentist daily

and in periodic visits.

Healing after various periodontal procedure

Healing following Scaling[13]

 Day 0: Bleed and exudation of GCF (Gingival Crevicular Fluid) will remove irritants

 Epithelial attachment is severed, acute inflammatory reaction in C.T

 Day 1: After an initial lag of 12 - 24 hrs, epithelium migration begins

 Day 2: Inflammation reduces, epithelialization is enhanced.

 Day 5 : New epithelial attachment begins

 1 - 2 weeks: Residual rete pegs involute and clinically gingiva appears healthy.

Healing following Root Planning

 In 2 hours: Numerous PMN leucocytes seen between crevicular surface and residual epithelial cells

 There is dilation of blood vessels, oedema & necrosis of the pocket wall.

 After 24 hrs: infiltration of inflammatory cells and keratinocytes migration seen.

 In 2 days: epithelialisation of entire pocket is seen.

 In 4 - 5 days at bottom of sulcus a new epithelial attachment appears.

 In 1 - 2 weeks depending on the depth of gingival crevice and severity of inflammation, complete epithelial healing is seen.

 Within 3 weeks CT repair by immature collagen fibres occur.

Healing Following Curettage

 Immediately blood clot is seen in gingival sulcus which does not have epithelial lining.

 Large number of PMNs occupy site of wound after which there is rapid GT proliferation.

 Epithelium: Healing of sulcular epithelium takes 2 - 7 days and JE (Junctional Epithelium) 5days.

 Connective tissue: Within 3 weeks immature collagen fibres appears.

Healing following Surgical Gingivectomy

 2nd day

 Formation of clot

 4th day

 Replacement of clot by GT (Granulation Tissue)

 A part of epithelial surface extends without rete pegs

 There is dense inflammatory infiltration

 6th day Stratified squamous epithelium covers the wound

 In CT, formation of collagen starts

 16th day

 Epithelium with rete pegs and dense collagenous CT occurs

 21st day

 Well-developed epithelial rete pegs and stratum corneum is thickened

 In CT, increased formation of collagen and clinically normal gingiva appears.

Healing following Gingival Depigmentation

 Immediately: clot formation takes place and acute inflammation of the underlying tissues.

 Replacement of clot by GT

 Capillaries of the PDL (Periodontal Ligament) migrate into GT

 Within 2 weeks, capillaries connect with gingival vessels.

 As healing takes place, initially vascularity increases then gradually decreases.

 Surface epithelialization is complete by 5 - 14 days.

 In 3 - 4 weeks: Complete epithelial repair takes place.

Healing Periodontal Flap Surgery

 Within 24 hours after suturing, contact is established by blood clot into the flap and tooth or bone surface. There is tissue injury as a result of bacteria and an exudate or transudate.

 One to three days following flap surgery— there is thin gap between flap and tooth or bone. During the close adaptation between flap and alveolar process epithelial cells move over the borders of flap and there is only a minimal inflammatory response.

 1 week following surgery—an epithelial attachment to the root has been established with the help of basal lamina and hemidesmosomes.

 2 weeks following surgery—collagen fibres appear parallel to tooth surface. Clinically it

appears normal but the union of flap and tooth is still weak.

 1 month following flap surgery—a well-defined epithelial attachment with fully-epithelialized gingival crevice is present. Supracrestal fibres begin to adapt a functional arrangement.

Conclusion

Wound care has evolved over time. However, societal changes, such as smoking and diabetes mellitus, can impact wound healing, leading to new and challenging wounds in patients, hindering body-wide healing.Periodontal tissues form a dentogingival junction, crucial for bone and ligament preservation, and safeguarding its integrity is essential for optimal periodontal health.Periodontal disease can be treated using various non-surgical and surgical modalities, including scaling, root planing, subgingival curettage, and LASER. The type of wound healing depends on cellular and molecular activities.

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