International Journal of Psychosocial Rehabilitation, Vol. 24, Issue 01, 2020
ISSN: 1475-7192

Knowledge about slowing of wound healing among undergraduate dental students

Type of manuscript: Original Research
Running Title: KAP wound healing
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Abstract:
Introduction: *Healing of wounds is one of the most interesting phenomenon which characterizes the living organism. A healing of the wound results in the death of the organism. So wound healing is essential for the primary survival mechanism. Healing is a process, not an incident and it refers to the replacement of the damaged tissue by the living tissue to restore the function.*

Aim: *To evaluate the knowledge about slowing of wound healing among under graduate dental students.*

Materials and method: *200 under graduate dental students studying in a dental college in South India were selected by convenient sampling method. They were distributed with questionnaire about slowing of wound healing. Data is collected and statistically analyzed. Based on the questionnaire distributed to students, the result have been calculated by dividing the wound healing into three columns as types , phases and factors of wound healing. Data were collected using a self-administered questionnaire adapted from several studies addressing various aspects of knowledge about slowing of wound healing.*

Result: *On calculating overall percentage of students about knowledge of wound healing, 67% of students have knowledge about wound healing whereas remaining 33% of students lack knowledge about wound healing.*

Conclusion:*On concluding this research we come to know that students have more knowledge about wound healing . The students who lack knowledge about wound healing should get more attention to it and gain more knowledge about it and will be very useful for their clinical practices.*

Key Words: *wound healing, systemic factors, local factors, primary wound healing, secondary wound healing*

I. Introduction:

An unhealed wound will eventually result in the death of the organism. Therefore wound healing must be considered as one of the primary survival mechanism from birth onwards. Healing of a tissue is generally considered to be a phase of inflammatory reaction, since it cannot be separated from the preceding vascular and cellular phenomenon occurring in response to an injury. When the healing process is fast, where the wound edges are approximated it is known by Primary Healing and when there is a tissue loss with the wound edges cannot be opposed resulting in contraction of the wound with the formation of granulation tissue due to epitheliaisation across the wound surface it is known as Secondary Healing.Various growth factors and cytokines play an important role in wound healing, such asepidermal growth factor(EDGF) produced by the epithelium around the damaged area helps in the regeneration of epithelial tissue[1]. Macrophages liberate fibroblast growth factor (FBGF) which mediates fibroblast activity along with transforming growth factor α (TGFα).Endothelial growth factor triggers the formation of new blood vessels. Oral wounds are common due to some sustained accidental injuries, some inflicted by the dentist for specific purpose and some disease process. The unusual anatomy of the oral cavity with the protrusion of teeth, constant inflammation of gingival tissues, presence of countless micro organisms in a warm, moist medium of saliva all contribute to modify the healing reactions of various wounds.[2]

II. Wound Healing Function:

When an injury occurs, the initial phase is always an outpouring of lymphatic fluid and blood. It is during this process that adequate hemostasis is achieved. Both the extrinsic and intrinsic coagulation pathways are activated and play a role in

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Received: 10 Nov 2019 | Revised: 20 Dec 2019 | Accepted: 02 Jan 2020

stopping the loss of blood. Aggregation of platelets follows the arterial vasoconstriction to the damaged endothelial lining. A releasing of adenosine 5´ diphosphate (ADP) results in the clumping of platelets and initiates the process of thrombosis [3]. The vasoconstriction is a short-lived process that is soon followed by vasodilatation, which allows the influx of white cells and more thrombocytes. The inflammatory phase begins with hemostasis and chemo taxis. Both the white cells and thrombocytes speed up the inflammatory process by releasing more mediators and cytokines. Besides platelet-derived growth factor, there are other factors which promote collagen degradation, the transformation of fibroblasts, growth of new vessels, and re-epithelialization. All of the processes occur at the same time but in a synchronized fashion [4]. Mediators like serotonin and histamine are released from platelets and increase cellular permeability. The platelet-derived growth factor attracts fibroblasts and, along with transforming growth factor, enhances division and multiplication of fibroblasts. The fibroblasts, in turn, synthesize collagen. Inflammatory cells, such as neutrophils, monocytes, and endothelial cells, adhere to a fibrin scaffold that is formed by platelet activation. The neutrophils enable phagocytosis of cellular debris and bacteria, allowing for decontamination of the wound. The proliferative or granulation phase does not occur at a discrete time but is occurring all the time in the background. By day 5 through 7 the fibroblasts have started to lay down new collagen and glycosaminoglycan [5]. These proteoglycans form the core of the wound and help stabilize the wound. Re-epithelialization starts to occur with the migration of cells from the wound periphery and adjacent edges. Initially, only a thin superficial layer of epithelial cells is laid down, but with time, a thicker and more durable layer of cells will bridge the wound. Neovascularization occurs through both angiogenesis, which is the formation of new blood vessels from existing vessels, and vasculogensis, which is the formation of new vessels from endothelial progenitor cells (EPCs).Once collagen fibers have been laid down on the fibrin framework, the wound starts to mature. The wound also begins to contract and is facilitated by continued deposition of fibroblasts and myofibroblasts. The maturational or remodeling phase starts around week 3 and can last up to 12 months. The excess collagen degrades, and wound contraction also begins to peak around week 3. Wound contraction occurs to a much greater extent in secondary healing than in primary healing. The maximal tensile strength of the incision wound occurs after about 11 to 14 weeks. The ultimate resulting scar will never have 100% of the original strength of the wound, and only about 80% of the tensile strength[6].

III. Clinical Significance of Wound Healing:

Clinical considerations in wound management include preventing and controlling infection and contamination, maintaining adequate moisture, treating edema, and preventing further injury. Wounds should be cleansed prior to closure. Wounds can be cleansed with either irrigation or scrubbed and irrigated with 0.9% saline solution. Alternately, wounds can be scrubbed with pluronic plyols and irrigated with normal saline. Tap water is frequently used by patients to irrigate wounds prior to seeking out medical attention. The advantage is that copious amounts of irrigants can be rapidly used; however, irrigation pressure may be difficult to control [7]. A study by Mosacati found that infection rates for wounds irrigated with tap water were comparable to those irrigated by a 0.9% saline solution.

IV. Wound Healing Process:

Wound healing is an orderly process initiated in a predictable manner whenever tissue damage occurs. In healthy individuals, healing progresses sequentially through three overlapping phases. There is (1) an inflammatory stage comprised of hemostasis or blood clotting and migration of inflammatory cells to the wound, (2) a proliferative phase involving migration and proliferation of keratinocytes, fibroblasts and endothelial cells, leading to re-epithelialization, neovascularization, and granulation tissue formation, and (3) a long remodeling phase involving extracellular matrix maturation aimed at restoring tissue structure and function [8]. Success in later phases is highly dependent on preceding phases. The inflammatory phase begins at the time of initial damage and typically lasts 5–7 days. The factors that initiate inflammation are released by resident cells after traumatic disruption of the intact tissue and the immediate response of platelets to ‘damage’ signals [9]. This initial inflammatory response serves to jump-start healing as inflammation stabilizes the wound by removing contaminating debris and controlling microbial invasion, and then inflammation creates an environment conducive to tissue repair. The inflammatory phase encompasses three critical elements that require the recruitment of cells from circulation:

(1) Passive aggregation of platelets for hemostasis,

(2) Neutrophil influx for infection control, and

(3) Macrophage accumulation to initiate repair [10].

The neutrophils and monocytes are attracted into injured tissues concurrently, but neutrophils predominate due to their abundance in the circulation. Both cell populations are recruited to the wound by a myriad of inflammatory chemokines and cytokines arising from the blood clot and injured cells at the margins of the wound. Among their many functions, these cells are phagocytic and remove bacteria from the wound [11]. In addition, newly recruited

monocytes, which differentiate into macrophages once they enter the tissue parenchyma, begin to initiate tissue repair.

Stages of wound healing; In healthy individuals, healing progresses sequentially through three overlapping phases:

(1) Inflammatory phase,

(2) Proliferative phase, and

(3) Remodeling phase.

Stress can affect progression through these stages via multiple immune and neuroendocrine pathways [12]. The current review focuses on the interactive role of glucocorticoids and cytokines (e.g. IL-8, IL-1, IL-1, IL-6, TNF- α, and IL-10). However, additional cytokines, chemokines, and growth factors are important to healing. These include CXC chemokine ligand 1 (CXCL1), CC chemokine ligand 2 (CCL2), granulocyte-macrophage colony stimulating factor (GM-CSF), monocyte chemotactic protein-1 (MCP-1), macrophage inflammatory protien-1 alpha (MIP-1), vascular endothelial growth factor (VEGF), transforming growth factor- (TGF-), keratinocyte growth factor (KGF), platelet-derived growth factor (PDGF), and basic fibroblast growth factor (bFGF). Macrophages produce enzymes such as hyaluronidase, elastase, and collagenase, which degrade hyaluronic acid, elastin and collagen in connective tissue. In doing so, the macrophage weakens the extracellular matrix to allow for migration and in-growth of fibroblasts, keratinocytes and endothelial cells that build the new tissue during the subsequent or proliferative phase of healing [13]. Not only do the macrophages prepare the extracellular matrix for tissue growth, they also synthesize and release multiple growth and regulatory factors, critical to the coordination of new tissue formation. Once macrophages begin to produce these growth factors, the repair part of healing actually begins. In fact, the formation of new capillaries from preexisting blood vessels (angiogenesis) and the deposition of new extracellular matrix by tissue fibroblasts are characteristic of the proliferative phase of repair. Once new tissue has been built to fill the void left by the initial injury, the final stage of healing begins. It involves contraction and tissue remodeling and is important for the approximate restoration of the original tissue's structure and function [14]. Whereas the first two stages of repair can be completed in as little as 12–14 days, this last phase of healing may continue for weeks, months or even years after injury. Although healing is a consistent and regulated process, stress can affect its progression via multiple immune and neuroendocrine pathways. For one, pro-inflammatory cytokines are key to successful healing. Neutrophils and macrophages are major sources of pro-inflammatory cytokines, including interleukin-1 (IL-1), IL-1, IL-6, IL-8 and tumor necrosis factor- (TNF-α). These cytokines help to prevent infection, prepare injured tissue for repair, and enhance recruitment and activation of additional phagocytic cells. In addition, cytokines regulate the ability of fibroblasts and epithelial cells to remodel damaged tissue [15]. Therefore, pro-inflammatory cytokines play a critical role in the healing cascade. Demonstrating this, IL-6-deficient mice exhibit up to a 3-fold greater healing time as compared to healthy wild type mice. The effects of stress on glucocorticoid function are another key-related mechanism in the stress-healing association. Glucocorticoids, which are responsive to stress, affect inflammatory processes. For example, stress-induced elevations in glucocorticoids can transiently suppress both IL-1 and TNF production in human .Similarly, mice treated with glucocorticoids showed impairment in the induction of IL-1 and TNF, as well as deficient wound repair. Although other mechanisms are implicated in the link between stress and healing and the substantial evidence for the interactive roles of pro-inflammatory cytokines and glucocorticoid hormone [16].

V. Intervention of Enhancing Wound Healing:

A variety of interventions have been used to target stress or the effects of stress with the goal of improving healing. For one, exercise influences immune and endocrine function as well as psychological responses to stress. Recent data demonstrate that regular physical activity can speed healing. Short-term exercise (1 hour/day for 3 days) did not affect rate of wound healing in a sample of 10 women. However, older adults who completed a 4-week exercise intervention (1 hour/day for 3 days/week) healed standard punch biopsy wounds 25% more quickly than did their less active counterparts. Notably, these effects were found despite low self-reported stress across the sample; effects may be even greater among individuals reporting more distress. Social contact may also buffer the effects of stress on healing. Among hamsters subjected to restraint stress, those who were pair-housed had significantly lower serum cortisol concentrations than did those who were socially isolated.

This reduction in cortisol had an impressive influence on wound repair: there were no significant differences in healing between pair-housed animals subjected to restraint stress and those maintained under non-stressful conditions. In part, the protective effects of social housing appeared to be mediated by oxytocin, a hormone released during social contact that facilitates social bonding. The administration of an oxytocin antagonist to socially housed animals delayed healing [17]. Moreover, treatment of socially isolated animals with oxytocin attenuated stress-

induced cortisol increases and speeded healing. These data are consistent with a large body of literature demonstrating health benefits of social support in humans and suggest a promising area of future research: the effects of social support on healing among patient populations [18]. In addition to its effects on serum cortisol, stress also induces substantial tissue hypoxia in the wound. As oxygen is necessary for healing, oxygen therapy may also prevent negative effects of stress on wound healing. Although wounding itself is associated with tissue hypoxia (normal tissue pO 2 ; 60 mm Hg; wound pO 2 ; 10–60 mm Hg) and hypoxia is thought to be important for healing (e.g. angiogenesis), stress drives tissue oxygen levels to a lower level where healing is impaired (stressed wound pO 2 ; 110 mm Hg)[19]. Therefore, increased oxygen demand and decreased oxygen supply at the site of the wound during times of stress may be a mechanism underlying the stress-healing association. In support of this hypothesis, restraint-stressed mice exposed to hyperbaric oxygen therapy twice per day during the first 5 days of healing healed at a rate nearly equal to unstressed mice [20]. Oxygen appeared to improve wound healing via its attenuating effects on inducible nitric oxide synthase gene expression.

VI. Materials and Method:

200 under graduate dental students who are studying in a dental college in south India were selected randomly by convenience sampling technique. They were distributed with a self-structured questionnaire to assess their knowledge about slowing of wound healing. Data was collected and statistically analyzed. Based on the questionnaire distributed to students, the result have been calculated by dividing the knowledge of wound healing into three columns as types, phases and factors of wound healing[21]. Data were collected using a self-administered questionnaire adapted from several studies addressing various aspects of knowledge about slowing of wound healing [22].

VII. Result:

Based on the questionnaire distributed to students, the result have been calculated by dividing the knowledge of wound healing into three columns as types, phases and factors of wound healing.

SYSTEMIC FACTORS		**LOCAL FACTORS**	
YES	**NO**	**YES**	**NO**
45%	**55%**	**37.5%**	**62.5%**

TABLE 1: Knowledge on factors affecting wound healing

TYPES		**PHASES**		**FACTORS**	
YES	**NO**	**YES**	**NO**	**YES**	**NO**
80%	**20%**	**77.5%**	**22.5%**	**43.5%**	**56.5%**

TABLE 2: Knowledge on, types, phases and factors affecting wound healing

VIII. Discussion:

By calculating the result, the percentage of students who knew about the types, phases, factors (systemic and local) of wound healing is compared to the students who lack knowledge in knowing about the wound healing. 80 % of students know about the types of wound healing whereas 20% of students lack knowledge about types of wound healing [23]. 77.5%of students know about the phases of wound healing whereas 22.5% of students lack knowledge of phases of wound healing. 43.5 % of students know about factors of wound healing whereas 56.5% of students lack knowledge about factors of wound healing. On dividing factors of wound healing into systemic and local factors, 45% and 37.5% of students know about the systemic and local factors of wound healing whereas 55% and 62.5% of students lack knowledge about systemic and local factors of wound healing.

On calculating overall percentage of students about knowledge of wound healing, 67% of students have knowledge about wound healing whereas remaining 33% of students lack knowledge about wound healing. Many studies were discussed and compared during this research. Some of the studies are compared with wound healing process. By

analyzing those data's collected related to this research are collected and compared in this research about wound healing process. Some of the studies compared with this research are wound healing by psychological process, wound healing during diabetes mellitus and wound healing response in age related macular degeneration [24,25].

IX. Conclusion:

Wound healing, as a normal biological process in the human body, is achieved through four precisely and highly programmed phases: hemostasis, inflammation, proliferation, and remodeling. For a wound to heal successfully, all four phases must occur in the proper sequence and time frame. A better understanding of the influence of these factors on repair may lead to therapeutics that improve wound healing and resolve impaired wounds. On concluding this research we come to know that students have more knowledge about wound healing . The students who lack knowledge about wound healing should get more attention to it and gain more knowledge about it and will be very useful for their clinical practices.

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Received: 10 Nov 2019 | Revised: 20 Dec 2019 | Accepted: 02 Jan 2020