



Review Article

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AN AYURVEDIC APPRAISAL ON CONCEPT OF WOUND HEALING MECHANISM

Monika Guleria ^{*1}, Kuldeep R. Choudhary ², Sanjeev Sharma ³

¹Lecturer, Department of Shalya Tantra, Shri Satya Sai Murlidhar Ayurvedic college, Moga, Punjab, India

²Lecturer, Department of Kaumarbhritya, Shri Satya Sai Murlidhar Ayurvedic college, Moga, Punjab, India

³Professor, P.G. Department of Shalya Tantra, R.G.G.P.G. Ayu. College, Paprola, HP, India

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***Corresponding author**

E-mail: monikaheaquarian@gmail.com

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ABSTRACT

Wound healing is a complex phenomenon. Only after defining the specific biologic processes of a particular wound the clinician can formulate a rational treatment. Therefore, a good clinician should have a very clear definition of the mechanism of wound healing. Irrespective of the type of wound and extent of the tissue loss, the healing of every wound is a continuous process. Division of the healing process into phases refers to the fundamental morphological alterations in the course of the repair process without reflecting the actual complexity of the process. The usual division is into four healing phases viz. haemostasis, inflammation, proliferation, and maturation. It is a complex process requiring the collaborative efforts of a number of different tissues and cell mediators. In Ayurvedic literature particularly in Sushruta Samhita a vast description of etiology, pathogenesis, classification and treatment of the wounds is available. So far as the healing of a wound is concerned detailed clinical features of each phase of healing have been mentioned. Understanding of these features is very important for the successful and uneventful management of the wounds. Even in today's highly sophisticated era when we have a vast knowledge of the mechanism of healing at the cellular level the clinical presentation as described in Ayurvedic literature is of immense value. Critical analysis of the phased mannered features of wound healing described in ancient Indian texts reveals that they still stand true in the present time. This paper is an effort to elaborate and interpret these clinical features of wound healing according to the present day understanding.

Keywords: *Vrana*, Wound, Wound healing.

INTRODUCTION

Injury is the birth partner of man and he has always tried his best to overcome the problems arising from trauma since the dawn of civilization. Evidence of the success of man over the trauma is his journey from the primitive age to the modern highly advanced era. Understanding of injuries or wounds can be routed down from ancient Indian texts Vedas which are the rich source of knowledge. Description of Devasur Sangrama in Vedas is one of the best examples of sustaining trauma and consequently its management.¹ Sushruta the pioneer of Indian surgery has vividly described the various modes of injuries and their management accordingly. While defining health he says that health is the state of equilibrium of Dosha, Agni and functions of Dhatu and Mala and not merely an absence of disease.² At the time of Sushruta the Indian surgery was in its highest glory. Vrana (wounds) were the main subjects at that time also. Merely healing of the wound was not the ultimate target but the healed area should look like the pre-injured state so far as colour, surface, hairs etc. are concerned. Keeping this fact in mind Sushruta has described Shashti Upakrama (60 measures) for the management and proper healing of the Vrana (wounds and ulcers). Thus, he has been rightly called as 'Father of Indian Surgery' as surgery revolves around Vrana and its essence lies in uncomplicated Vrana Ropana (wound healing). Now-a-days also wound care is evolving constantly with advances in medical field but the search for ideal dressing material is still on as medical professionals are facing many challenges due to multi-resistant organisms, hence decreasing effect of antibiotics. So, ancient pathway of wound healing and healing methods are being revisited and explored by the clinicians.

Hence, it is very essential to understand the concept of healing before going for its management so that we should know in what stage the wound is and how can we interfere to enhance the healing of wound in a proper manner, thereby relieving the patient from pain (physically and mentally) he is suffering from. Mechanism and phases of wound healing in Ayurveda has been described in its own way. Thus, hereby an attempt has been made in present review article with an aim to compile the sequential wound / ulcer healing phases described in Ayurveda and its probable modern interpretation and to highlight the practical utility of healing phases described in our ancient texts in the management of Vrana.

Vrana

Vrana is the most important part of Shalya Tantra and our texts have emphasised a lot on wound care occurring due to trauma or a result of vitiated Dosha. For providing proper and complete healing of wound, it is essential to understand what a wound is and through which phases a wound pass before getting healed.

Vrana has been described as 'vrana gatra vichurnane, vranyati iti vranah'⁴

(i) 'Gatra' means part of the body or tissue.

(ii) 'Vichurnane' means destruction, break or discontinuity.

So, Vrana is defined as the destruction, break or discontinuity of the body tissue.

Vrana is called so, since it covers (occupies) the skin or the area of body and also because the Vrana Vastu (scar) does not disappear even after healing and remains till the person survives.⁵

In modern texts, wound is said to occur when integrity of any tissue is compromised (i.e. skin, muscle or bone). It may be

caused by an act, such as a gunshot, chemicals, cut, heat, cold, pressure, fall, or surgical intervention; by an infectious disease; or by an underlying condition which leads to a number of responses at cellular and molecular level.⁶

Concept of Wound healing in Modern text

It is a complex biological response of the body towards an injury which involves a series of events right from the interaction of cells and molecules upto the morphological changes of the wounded area, in order to restore nearly normal structure and function. It consists of four phases: haemostasis, inflammation, proliferation and tissue remodelling or resolution.⁷

(1) Haemostasis^{8,9}

It occurs within minutes of the initial injury. Process involves vasoconstriction, platelet aggregation, fibrin deposition and clot formation at the end.

Vasoconstriction: It is initiated by release of vasoactive amines (epinephrine and norepinephrine) in response to dermal injury locally. Injured cells secrete prostaglandins (e.g. thromboxane), thereby contributing to vasoconstriction.

Platelet aggregation: Damaged cells release tissue factors which stimulate aggregation of platelets. On aggregation and adherence, platelets release the contents of alpha granules (immunomodulatory and proteinaceous factors), dense bodies (fuel-providing compounds i.e. calcium, serotonin, ADP, ATP) and lysosomes into their cytoplasm. These Alpha granules include albumin, fibrinogen, fibronectin, IgG, coagulation factors V and VIII, platelet-derived growth factor (PDGF), transforming growth factors a and b (TGF-a and TGF-b), fibroblast growth factor-2 (FGF-2), platelet-derived epidermal growth factors (EGFs), and endothelial cell growth factors.

Fibrin and the coagulation cascades: It consist of intrinsic and extrinsic pathways triggered separately leading to the production of thrombin which is responsible for conversion of fibrinogen to fibrin. Thrombin results in increased vascular permeability and facilitates extravascular migration of inflammatory cells. Fibrin forms the meshwork to stabilize the platelet plug and becomes an important component of the provisional matrix on which fibroblasts and other cells migrate during the healing. Vitronectin (derived from serum and aggregating platelets) coats fibrin and facilitates the binding of fibronectins (second important component of the provisional matrix) produced by fibroblasts and epithelial cells.

In short, in response to injury blood vessels constrict for short duration and later relax. The platelets aggregate and adhere to the exposed collagen and secrete factors that stimulate intrinsic pathway of clotting by the production of thrombin which converts fibrinogen to fibrin. The fibrin mesh converts aggregated platelets to a stabilized platelet plug. Platelets also secrete cytokines like platelet-derived growth factor (PDGF), which initiate further steps in the healing process. And the cells - inflammatory and reparative, are attracted towards the injured region.

(2) Inflammation^{8,9}

It presents as erythema, swelling, pain and warmth lasting up to 4 days after injury. It is characterized by increased vascular permeability and migration of leukocytes into the extravascular space. Main function of inflammation is to bring inflammatory cells towards the injured area to kill bacteria and eliminate the debris so that the healing can proceed.

Vasodilatation: It occurs 10 to 15 minutes after injury. Endothelial products, kinins and mast cell-derived factors such

as leukotrienes, prostaglandins and histamine are responsible for vasodilation which leads to capillary leakage. Complement factors C3a and C5a also contribute to capillary leakage and in addition, attract neutrophils and monocytes.

Margination: The adhesion of leukocytes to endothelial cells lining the capillaries of injured area is called margination. Collagen, elastin breakdown products, complement factors, and immunomodulatory factors including TGF-b, tumor necrosis factor-a (TNF-a), interleukin-1 (IL-1), PDGF, leukotriene B₄, and platelet factor IV stimulate this leukocyte margination.

Diapedesis: Here transmigration of leukocytes through endothelium is facilitated by platelet factor IV and platelet activating factor which increase the expression of CD11/CD18 (an integrin on neutrophils).

Inflammatory cells: Chemotactic factors such as fibronectin, elastin derived from damaged matrix, complement components, TGF-b etc. stimulate transformation of migrating monocytes into macrophages. IL-2 and INF- γ derived from T lymphocytes activate macrophages which alongwith neutrophils initiate wound debridement by phagocytosis of bacteria and foreign material which are engulfed and digested by oxygen radicals and hydrolytic enzymes within the cells. Other inflammatory cell types like eosinophils and basophils are nonspecific effectors of immune responses and reach their highest concentrations in wound at 24 to 48 hours after injury. As the healing process proceeds, inflammatory cells remain in the wound for approximately 7 days and then sloughed out.

Cytokines: The wound-healing process is mainly regulated by ordered production of cytokines responsible for cellular migration and proliferation and synthetic activities. Important sources of cytokines are platelets and macrophages. Tissue hypoxia is a signal for cytokine release and has been shown to stimulate the release of TNF-a, TGF-b, vascular endothelial growth factor (VEGF), and IL-8 (IL-8) from fibroblasts, endothelial cells, and macrophages. Collagen synthesis and scar formation are influenced by cytokines such as PDGF, TGF-b, and FGF-2.

In short, second phase of wound healing involves cleaning up of debris by neutrophils or PMN's (polymorphonucleocytes, the first inflammatory cells recruited). The neutrophils and macrophages provide the first and second line of defense respectively. Degradation of fibrin attract further cells involved in the process. Macrophages secrete a number of chemotactic and growth factors such as fibroblast growth factor (FGF), epidermal growth factor (EGF), transforming growth factor beta (TGF- β) and interleukin-1 (IL-1).

(3) Repair/Phase of proliferation^{8,9}

Cellular migration and proliferation (Fibroplasia): The cellular components of the wound undergo considerable changes after a week of injury. Cytokines contribute to fibroplasia, epithelialization, and angiogenesis, so they continue to be a part of healing. Fibroblasts migrate from adjacent tissues (stimulated by PDGF, TGF-b, EGF and fibronectin) and the undifferentiated cells in the vicinity of wound also transform into fibroblasts under the influence of cytokines. The fibrin-fibronectin matrix which was initially dominated by inflammatory cells is now densely populated by fibroblasts and endothelial cells.

Angiogenesis: It starts after 2 days of injury and is regulated by a variety of cytokines (FGF-2 and VEGF are the two most important). The contributing factors are high lactate levels, acidic pH and decreased oxygen tension. The endothelial buds derived from intact capillaries at the periphery of wound during this phase grow by cellular migration and proliferation. As the chain of endothelial cells elongates, they develop a curvature and a lumen. Then the endothelial bud comes in contact with

another bud and get interconnected giving rise to a new capillary.

Epithelialization: After injury, reconstruction of injured epithelium is an important step for re-establishment of the barrier functions of the skin. Basal cells at the wound edge elongate and migrate across the denuded surface of the wound during first 24 hours of injury. Epithelial appendages, if safe, also contribute migratory epithelial cells. Migration of epithelial cell requires actin filaments within the cytoplasm. Re-establishment of a basement membrane (in case it is destroyed) under the migrating cells involves the secretion of tenascin, vitronectin, and type I and V collagens. The cells become more basaloid and their further proliferation forms a multilaminated new epidermis covered by keratin.

Protein deposition: The quality and quantity of matrix deposited during this phase affects the strength of a scar (mainly constituted by collagen). Fibroblasts are responsible for the synthesis of collagen and other proteins regenerated during the repair process. Collagen synthesis is stimulated by TGF- β , PDGF and EGF and continues at a maximal rate for 2 to 4 weeks, slows down after that. With increase in protein synthesis, the nature of the wound matrix changes as collagen and other proteins (such as proteoglycans, important part of mature matrix) gradually replace fibrin. In intact dermis, type I collagen predominates and makes up 80% to 90% of the collagen and remaining 10% to 20% is type III collagen.

Wound contraction: It begins 4 to 5 days after injury and continues for approximately 2 weeks. The average rate of contraction is approximately 0.6 to 0.7 mm per day. Predominance of myofibroblasts at the wound periphery characterizes wound contraction. Myofibroblasts are modified fibroblasts that appear 4 to 6 days after injury and are commonly seen in the wound during coming 2 to 3 weeks. The wound contraction does not require collagen synthesis, it is cell mediated (TGF- β and other cytokines are involved).

In short, the granulation stage (involves 3 sub phases – fibroplasia, matrix deposition and angiogenesis) starts approximately four days after injury and lasts up to 3 weeks. It is characterized by the presence granulation tissue, replacement of dermal and sometimes subdermal tissues (epithelialization) and wound contraction. Specialized fibroblasts are responsible for wound contraction which occurs by dehydration, contraction of collagen and myofibroblasts. In angiogenesis, the outer layers of capillaries are regenerated by pericytes and the lining by endothelial cell. Epithelialization occurs by detachment, migration, proliferation and differentiation of cells (keratinocytes) and in its last stage, the keratinocytes differentiate to form stratum corneum.

(4) Remodelling / Phase of maturation and scar tissue formation^{8,9}

It is the last phase of wound healing, where remodelling of the dermal tissues produces greater tensile strength. The wound never regains full strength as of normal dermis but achieve 80% of it at 3 months. The principle cells involved in this process are the fibroblasts. This stage starts approximately 21 days after injury and may continue up to 2 years. There is decrease in collagen synthesis (mediated by γ -interferon, TNF- α , and collagen matrix) and it equals the rate of its degradation.

With remodeling the nature of the matrix changes as immature scar contains disorganized collagen fibers, which are replaced by thicker fibers gradually and arranged parallel to the skin stresses. Also, the number of cross-links within and between the molecules increases gradually. The collagen matrix becomes less cellular by apoptosis of cells involved in wound healing.

In short, this stage involves 3 changes - conversion of soft febrile collagen fibrils to insoluble elastic fibers, embryonic active fibroblasts mature into adult resulting fibrocytes and devascularisation. All these changes provide tensile strength to the wound.

Concept of Wound healing phases in Ayurveda and its correlation with Modern science

(1) Dushta Vrana^{10,11} / Infected / Untidy wound

In Ayurvedic texts Dushta Vrana has been described as the one having clinical features as given below- Dirghakaalanubandhi (chronic in nature); Atisamvrita or Ativivrita (extremely narrow or wide/broad mouthed); Atikathina or Atimridu (too hard or too soft); Utsanna or Avsanna (elevated or depressed); Atisheeta or Atiushna (too hot or too cold); Bhairava (fierce/unpleasant/ugly looking); Krishna, Rakta, Peeta and Shukla Varna (colour – black, red, yellow, white); Puti (cadaverous/foul smell); Shvayathu (swelling); Atiruk (very painful); Daah (burning sensation); Kandu (itching); Raaga (redness); Paaka (suppuration); Pidika updruta (pustules crop up); Putimamsasirasayanayuprabhritibhi (full of sloughing muscles, vein, ligament); Putipuyaparistrutih (exuding putrifying pus moving in abnormal directions); Dushtashonitastavi (exuding vitiated blood) and Upadrava (complications).

Above mentioned cascade of the features of Dushta Vrana is not the feature of any single type of Dushta Vrana rather few of them are seen in a particular Vrana according to Doshic involvement and Samprapti (etiopathogenesis). Dalhana while commenting on this has clarified the involvement of each Dosha according to the clinical features.¹² This means that in a particular type of Vrana (wounds or ulcers) particular group of clinical features will be found depending upon the etiopathogenesis. However, in the case of Sadyovrana (traumatic wounds) the features of each wound depend upon the causative weapon or object or mode of injury and it remains Shuddha (devoid of any contamination) for 7 days. But after 7 days of occurrence these wounds (Sadyovrana) may become Dushta Vrana (contaminated or unhealthy wounds) due to the involvement of Doshas (body humours) and will have features of Dushta Vrana according to the involvement of Dosha and then require same treatment as is given in Dushta Vrana.¹³

Dushta Vrana can be compared with anyone of the following as per its features – contaminated, dirty, infected, untidy, unhealthy, delayed or nonhealing wound. Various specific ulcers like tuberculous, malignant, diabetic, venous, decubitus, trophic or tropical ulcers also fall under the category of Dushta Vrana.

Unhealthy wound/Infected wound: It has foul smell, slough or necrotic tissue (black, yellow, green in color), wound margins sloping, punched out, raised, rolled, and undermined, large amount of discharge, increased pain, increase or decrease in size, surrounding skin is red, hot and swollen, other constitutional symptoms.¹⁴

Untidy wound: It is the wound which is irregular, contains devitalised tissue, tendons, arteries, nerves may be exposed and injured. These wounds are prone to infection leading to delayed healing, may lead to gas gangrene and even death. Their management involves excision of all the dead and devitalised tissue to convert it into a clean wound and then treat it.¹⁵

From the above description it is clear that Dushta Vrana is an untidy, unhealthy, non healing wound having infection showing features of inflammation and suppuration. The pus formed makes it soft and hot and tracks down through various paths (to make it elevated) by destroying the underlying tissues leading to broadening of mouth of the wound and drains out with foul odour (making the wound depressed). Such kind of wounds, if

not treated properly enter into state of chronicity and fail to heal, may become hard due to fibrosis and give an ugly look due to presence of pus, dead tissue (slough). Its colour may be black, red, yellow, pale due to the presence of necrosed tissue, inflammation, unhealthy over granulation and slough. If still left untreated, complications may result.

Acharya Charaka has mentioned clearly that the wound with foul odour, profuse discharge and intense pain are Ashudha Vrana and in these Shodhana should be done. By doing this the wound will become clean, which can now be treated easily and will heal properly, as mentioned for the untidy wounds above.¹⁶

Infection and wound healing: Inflammation (a part of the wound-healing) is important as it removes the contaminating micro-organisms and foreign bodies. In presence of bacteria and endotoxins there is prolonged elevation of pro-inflammatory cytokines (such as IL-1 and TNF- α) leading to prolongation of inflammatory phase and may make the wound chronic. Prolonged inflammation causes increase in level of matrix metalloproteases (MMPs) that can degrade ECM and also there is a decrease in level of protease inhibitors. Growth factors in chronic wound degrade rapidly due to this imbalance.^{17,18,19} In infected wound bacteria occur in form of biofilms (complex of aggregated bacteria embedded in matrix).¹⁸ Mature biofilms are resistant to conventional antibiotic treatment as they shield the bacteria from phagocytosis by PMNs leading to failure of antibiotics in chronic wounds.²⁰

(2) Shudha Vrana ^{21,22,23,24} /Wounds with healthy early granulation tissue

Lakshana (Clinical features) of Shudha Vrana are as follows - Tribhirdosharanakrantah (unaffected by Tridosha); Avedano (absence of pain); Nirasravo (absence of discharge or moisture); Naatirakto(not very red colour); Naatipandu (not very pale colour); Naatishyavo (not very black colour); Nachautsano(not elevated); Nachautsangi (not protruded); Shyavaoshta (Wound edges are bluish black in colour); Pidkismah (small eruptions/granulation tissue which is even); Kinchidunnatmadhyo (slightly raised floor); Jihavatalabho (Resembles the tongue); Mridu(soft); Snigdha(moist);Shalakshana (smooth); Suvyavasthita (even, good looking) and Anupdrava (not accompanied with complications).

Recent origin wounds are the acute wounds. Acute wounds which are clean and non-infected are considered to be the Shuddha Vrana. They lack any kind of discharge and pain as there is no infection and inflammation present. The look of the wound resembles tongue and has small evenly distributed eruptions which are red in colour. This can be considered as the stage of wound healing where granulation is taking place (sub phase- angiogenesis).

Furthermore, Shuddha Vrana also refers to that Dushta Vrana (infected or unhealthy wound / ulcer) which after proper treatment has attained the status of a clean ulcer and ready to heal. Such clean ulcer will also exhibit the above mentioned clinical features.

Granulation tissue formation: Granulation tissue is called so because it is granular in appearance. It is profuse fibrous connective tissue which replaces the clot in the process of healing of wound. It gives bumpy or granular appearance, light red or pink in color having new capillary buds within it and is soft to touch. It can be explained by 5 P's – Pink, Punctate haemorrhages, Pulseful, Painless, Pin head granulation.²⁵ It involves 3 sub-phases - fibroplasias, matrix deposition and angiogenesis.

Angiogenesis /neovascularisation: It occurs when endothelial cells migrate to the wound and fibroblasts proliferate. The area

looks red in color because of presence of capillaries in tissue (as angiogenesis is occurring).¹⁸ The endothelial basement membrane breaks leading to detachment of endothelial cells from pre existing capillaries and post capillary venules and migrate towards the wound leading to the formation of new vessels. New sprouting buds organize themselves and fuse giving rise to new capillary networks.⁸

Acharya Sushruta has specifically mentioned that in Shudha Vrana these eruptions are Nachautsano, Nachautsangi (even, not raised) that means there should not be overgranulation (when granulation over grows beyond the surface of wound).²¹ It is also called as hypergranulation, proud flesh, hypertrophic granulation or exuberant granulation. Wound healing by second intention usually show over granulation identified clinically as friable red, often shiny and soft appearance raised above the skin.²⁷ The tissue involved in over granulation can be healthy or not²⁸ and may bleed easily. If it is not treated, then healing is delayed because its moist surface acts as an ideal medium for bacterial growth and formation of biofilms.²⁹ Over granulating wound does not heal as the epithelial tissue is not able to migrate across the wound and contraction of wound is also halted. The wound may remain in the first stage of healing due to infection and may lead to chronic inflammation converting the wound into chronic or delayed healing.³⁰

Hence, Shudha Vrana is an acute clean wound or treated unhealthy ulcer which is in the stage of early granulation tissue formation and proceeding towards further healing.

(3) Ruhyamana Vrana ^{31,32} / Healing wound/Collagen deposition & Epithelialisation

It has got following features - Kledavarjita (no discharge); Kapotavarnaprathima (color – resembling pigeon) and Sthirashchapitikavanto (has scales or flakes of skin adhered firmly).

Healing wound: It is pink or ruby red in color, has small to moderate clear or serous discharge, size is decreasing and the surrounding skin is warm, pink and healthy.

Ruhyamana Vrana is the one where the wound is healing and has firmly adhered skin which shows that it is at a stage where collagen deposition is taking place providing strength to the wound and epithelialisation is also occurring.

The fibrin fibronectin clot does not provide resistance against further injury, it only holds the wound closed.³³ Collagen deposition is very important in wound healing as it gives strength to the wound. The collagen matrix is laid by fibroblasts and on this the cells of inflammation, angiogenesis and connective tissue construction, attach, grow and differentiate.³⁴ Gradually granulation ceases and fibroblasts also decrease in number. At the end of granulation phase, apoptosis of fibroblasts starts and now granulation tissue is populated mainly by collagen.³⁵

In open wound, re-epithelialisation occurs as epithelial cells migrate and form a barrier between the wound and external environment. Main cells of epithelialization phase include basal keratinocytes from wound edges and dermal appendages. These cells progress in a sheet like pattern (epithelial tongue) across the wound and proliferate.³⁶

(4) Samyak Rudha Vrana ³⁷ / Properly Healed wound

Wound which has healed, has got following features -Agranthim (no eruptions/swelling); Arujam (no pain); Ashunam (no swelling); Samtalam (even surface) and Twaka savarnam (colour- same as of skin)

Maturation and remodeling: The maturation phase starts when collagen production and degradation becomes equal.³³ During

maturation, replacement of type I collagen by type III takes place. The disorganized collagen fibres get re-organization and align themselves along the stress lines.³⁸ The tensile strength of the wound increases and gains 80% strength of the normal tissue at the end.³⁹ Major goal of this phase is to reduce excessive ECM, align it and provide tensile strength to the wound. It involves 3 changes - soft friable collagen fibrils get converted to insoluble elastic fibres, embryonic active fibroblasts mature into adult resting fibrocytes and devascularisation. Because of this the wound edges get firmly attached and the colour of wound changes from red/pink to normal as of the skin as given in Lakshana of Samyak Rud Vrana.

(5) Vrana Vastu / Scar formation

Acharya Sushruta has mentioned about VranaVastu (scar) which persists lifelong even after completion of the wound healing.⁵ By this it can be considered as an indication of stage of scar formation during wound healing. The word is derived from Greek word schara, meaning place of fire. It is the residual visible mark of the wound i.e. an area of skin replaced by fibrous tissue after injury or disease, which remains for lifetime. From above content two things become very clear. First one is that in Ayurveda the clinical presentation of each phase of wound healing has been well described. By understanding these features a clinician can have an accurate idea about the status of Vrana and can plan the treatment accordingly. Modern science, however, has attained the success in getting the knowledge of the wound healing up to cellular and molecular level yet many things are still not clear. Ayurveda has its own philosophy to describe the healing process at molecular level on the basis of different theories like Dhatu Nirmana theory, Panchamahabhoota theory and Doshic theory.

Second important issue which evolves is that Sushruta was very clear about the fact that scar formation is inevitable in any kind of wound healing which he has narrated while mentioning the definition of the wound.⁵

Here he has mentioned that Vrana Vastu (scar) will remain throughout the life. In other words, it can be stated that newly formed tissue in the place of damaged tissue is different than that of the original damaged tissue and leaves scar. Although Sushruta has advocated so many measures to correct the defect for cosmetic purposes as Shashti Upakrama yet scar is inevitable. Here it is pertinent to mention that whatsoever the features of different healing have been advocated by Sushruta are the description of secondary healing.

Mammalian healing is primitive in comparison with that of the "lower" forms of life. It is paradoxical that in creatures considered low on the phylogenic scale tissue heals by a process of regeneration that is much more sophisticated than the primitive repair process in human beings – "wound healing" – which involves mechanism of haemorrhage, inflammation, matrix deposition, and scarring.⁴⁰

CONCLUSION

Hence, wound healing is a complex biological process. Only after defining the specific biological process of a particular wound the clinician can formulate a rational approach to therapy. This healing process for understanding purposes has been divided into certain phases which are sequential rather a continuous phenomenon without any lag period. However, this process can be influenced and diverted by numerous known and unknown factors. The ongoing process of wound healing is reflected clinically by a typical clinical presentation of the wound. Clinician should have the ability to recognize the phase of healing by the naked eye appearance of the wound and then

he should plan the best treatment for that wound. Wound and its healing have been described throughout the recorded Indian history i.e. from the Vedic periods. But Sushruta the 'Father of Indian surgery' has clearly defined the clinical presentation of the wounds and ulcers. He was well aware about the process and events which take place during the healing of a wound and has described them well. These clinical features of ongoing wound healing process (Shuddha Vrana, Ruhyamana Vrana and Rudha Vrana) are the best guidelines for the attending clinician to plan further treatment strategies to accomplish best healing with the restoration of anatomical and physiological integrity of wounded area. If a wound is properly assessed and addressed on the basis of the principles laid down in Ayurveda much suffering and crippling can be prevented successfully.

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