2) Wound Healing – Dr. Jalal

Wound Healing

Wound Healing is a mechanism by which the body attempts to restore the integrity of the injured part. During repair, a complex chain of events eventually leads to the formation of a scar. In certain circumstances, the cellular processes that contribute to repair become unregulated, leading to excessive scaring in the form of hypertrophic scars and keloids, at other times, abnormalities in repair occur, leading to deficiencies in wound healing such as are seen in chronic , non-healing wound.

Factors Influencing Healing of a wound

1. Site of the wound, Structures involved
2. Mechanism of wounding: Incision, Crush, Crush avulsion
3. Contamination (foreign bodies, bacteria)
4. Loss of tissue
5. Other local factors: vascular insufficiency (arterial or venous), previous radiation, pressure
6. Systemic factors:
   - Malnutrition or vitamin & mineral deficiencies
   - Disease (DM), Medications (e.g. Steroids), Smoking
   - Immune deficiencies (e.g. chemotherapy, AIDS)

Four Types of Wound Healing

1. *Primary healing:* Occurs when the wound is closed within hours of its creation. The wound edges are re-approximated directly using sutures or by some other mechanical means, collagen metabolism provides long-term strength to the wound, when normal, synthesis, deposition & cross – linking. Epithelization, provides coverage of the wound surface & acts as a barrier from bacterial invasion.
2. *Delayed Primary Healing:* Contaminated or poorly delineated wound is left open to prevent wound infection. After 3-4 days local phagocytic cell recruitment into the wound has occurred & angiogenesis has begun. Inflammatory cells are present that destroy contaminating bacteria. This decreases the risk of infection in contaminated wounds.
3. *Secondary Healing:* An open full-thickness wound is allowed to close by both wound contraction & epithelization. Appropriate for infected or contaminated wounds. Allows drainage of fluid. Allows debridement with dressing changes. Prolonged inflammatory phase leading to increased scaring & wound contracture. Contracture occur by myofibroblasts.
4. *Healing of Partial-thickness Wounds:* Partial-thickness wounds which involve the epithelium & the superficial portion of the dermis, heal mainly by epithelization. There is minimal collagen deposition & an absence of wound contraction.

Phases of Wound Healing

A. Inflammatory Phase

1. Begins at the time of injury, lasts 2-3days
2. Begins with vasoconstriction to achieve hemostasis (epinephrine & thromboxane)
3. Platelet plug forms & clotting cascade is activated, resulting in fibrin deposition
4. Platelets release platelet-derived growth factor (PDGF) & transforming growth factor –B (TGF-B) from their alpha granules, attracting inflammatory cells, particularly macrophages.
5. After hemostasis is achieved, vasodilatation occurs & vascular permeability increases (due to histamine , platelet-activating factor, bradykinin, prostaglandin I -2, prostaglandin E 2 & nitric oxide) aiding the infiltration of inflammatory cells into the wound.
6. Neutrophils peak at 24 hours & help with debridement
7. Monocytes enter the wound, becoming macrophages, & peak within 2-3 days
8. Macrophages produce PDGF&TGF-B, attracting fibroblasts &stimulating collagen

B. Proliferative Phase

Lasts from the 3rd day to the 3rd week

1. Fibroblasts: attracted & activated by PDGF& TGF-B, arrive day 3 reach peak numbers by day 7
2. Collagen synthesis mainly type III (blood vessels &immature scar), angiogenesis & epithelization occur.
3. Total collagen content increases for 3weeks until collagen production & breakdown become equal &the remodeling phase begins.

- Fibroblasts require vitamin- C to produce collagen.

C- Remodeling Phase

Increased collagen production & breakdown continue for 6 months to 1 year.

1. Type I collagen replaces type III until it reaches a 4 :1 ratio of type I to type III (that of normal skin & mature scar tissue)
2. Wound strength increases as collagen reorganizes along lines of tension & is cross-linked.
3. Vascularity decreases
4. Fibroblast & Myofibroblasts cause wound contraction during the remodeling phase.

Causes of abnormal wound healing

- Hyperglycemia
- Arterial disease –ischemia leads to inhibited collagen production & infection.
- Venous insufficiency, increase venous pressure lead to edema & decrease O_2 diffusion
- Abnormal pressure distribution
- Nutritional deficiencies
- Infection increases collagen breakdown & decrease epithelization

Scar & Scar Revision

Scar is a mark remaining after the healing of a wound or other morbid process.

Features of a good scars:

1. Fine line or series of lines to RSTL, contour junction & skin wrinkles.
2. No contour irregularities.
3. No pigmentation abnormalities.
4. No contractures or distortion of adjacent structures.

Types of Scars:

- Immature scar
- Mature scar

Objectives of Scar Revision:

1. To improve scar direction
2. To decrease scar width
3. To divide a long scar into smaller components.
4. To correct mal alignment or distortion of anatomical units.
5. To improve any surface irregularities.
6. To correct any pigmentation irregularities.

How to obtaining a fine line scar:

A. Controllable Factors
    1. A traumatic technique.
    2. Eversion of wound edges.
    3. Placement of the scar in the same direction of the skin lines.
B. Non controllable Factors:
    1. Age
    2. Site
    3. Type of the skin
    4. Length of the scar
    5. U–shaped scar
C. Complicating Factors:
    1. Skin disorders e.g.; Ehler–Danlos syndrome (genetically transmitted disease, hyper extensible & laxity occur prematurely -abnormal collagen maturation & tissue fragility - surgery ass. With prolonged healing, hemorrhage, and darkly pigmented or telangiectatic hypertrophic scars.- elective surgery is usually not advised)
    2. Infection
    3. Individual healing mechanism

Factors to be considered before performing - Scar Revision:

1. Time since injury.
2. Nature of the injuring agent.
3. Age
4. Location
5. Ethnic back ground e.g.; hyperpigmentation less in lighter skin
6. Skin tone & light effect; scars are visible by different color than the surrounding light reflection from the scar surface.
7. Healing of previous scar.
8. Nature of the scar: spread wide scar, hypertrophic scar true keloid.
9. Whether any skin lost.
10. Perception & expectations of the patient & family.

Treatment

- *Medical:* 1. Ionizing Radiation, 2. Steroid injection, 3. Pressure (i. pressure splint, ii. silicone gel sheet.)
- *Surgical:* 1- Excision, 2- Excision &undermining, 3- Z- Plasty, 4- W- Plasty, 5- Skin graft, 6- Skin flap, 7- Dermabrasion, 8- Tissue expansion, 9- Laser

Undesirable Results from Scar Revision:

1. *Hematoma:* if tissue dead space not closed by suturing or compression dressing
2. *Infection:* rare in scar revision of face, occur on trunk & extremities, retained FBs lead to infection
3. *Hyperpigmentation:* use of SPF 15%, Tretinoin & Hydroquinone
4. *Milia:* are small sebaceous inclusion cysts.
5. *Dehiscence:* due to excess tension or direct trauma after sutures removed.
- Use skin tapes after suture removal.