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SRB's Surgery *for* DENTAL Students

Sriram Bhat M

Foreword
V Surendra Shetty

3rd Edition

JAYPEE

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CHAPTER 3

Wounds, Inflammation, Sinus, Fistula and Ulcer

Chapter Outline

- Wound
- Problems with Wound Healing
- Compartment Syndrome
- Crush Syndrome
- Keloid (Like a 'Claw')
- Hypertrophic Scar
- Sinus
- Fistula
- Median Mental Sinus
- Ulcer
- Trophic Ulcer
- Pressure Sore (Bedsore/ Decubitus Ulcer)
- Diabetic Ulcer (Diabetic Foot)
- Meleney's Ulcer
- Traumatic Ulcer
- Arterial/Ischemic Ulcer
- Carcinomatous Ulcer (Epithelioma, Squamous Cell Carcinoma)
- Marjolin's Ulcer
- Rodent Ulcer
- Melanotic Ulcer
- Tropical Ulcer
- Venous Ulcer (Gravitational Ulcer)
- Tuberculous Ulcer
- Lupus Vulgaris
- Ulcer due to Chilblains
- Ulcer due to Frostbite

WOUND

DEFINITION

Wound is disruption of any tissues (soft tissues, organs or bones) structurally and functionally. There is discontinuity or break in the integrity of tissues or organs. Ulcer is disruption or break in the continuity of any lining—may be skin or mucous membrane; ulcer is one of the type of wounds.

I. CLASSIFICATION (Rank and Wakefield)

a. **Tidy wounds**
They are wounds of surgical incisions and caused by sharp objects.
Usually primary suturing is done. Healing is by primary intention.

b. **Untidy wounds**
They are:
- Crushed
- Tear
- Avulsion
- Devitalized injury
- Vascular injury
- Multiple irregular wounds
- Burns, etc.

Fracture may be present.

Wound dehiscence, infection, delayed healing are common.

Liberal excision of devitalized tissue and allowing to heal by secondary intention is the management.

Worry does not empty tomorrow of its sorrow. It empties today of its strength

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II. CLASSIFICATION (Based on Type of Wound)

1. ***Clean incised wound***
   It is a wound caused by sharp objects like knife, glass or blades. Primary suturing is done and it heals by first intention leaving a thin, linear scar (Figs. 3.1 and 3.2).
2. ***Lacerated wound***
   Wound edge is devitalized, crushed and wide. It is treated by wound excision and delayed primary suturing. Scar formed is wide and prone for hypertrophic scar formation (Figs. 3.3A and B).
3. Bruising or contusion.
4. Hematoma (Figs. 3.4 and 3.5).
5. Closed blunt injury.
6. Puncture wounds and bites.
7. *Abrasion (Fig. 3.6)*
   It is superficial, and is due to shearing of skin in which surface is rubbed off. It heals by epithelialization.
8. Traction and avulsion and degloving injury (Fig. 3.7).
9. ***Crush injury***
   It is caused by war wounds, road traffic accidents, and tourniquet.
   It leads on to:
   - Compartment syndrome



Fig. 3.1: Incised and lacerated wounds in the leg.



Fig. 3.2: Incised wound over eyebrow.





Figs. 3.3A and B: Lacerated wound on the scalp.



Fig. 3.4: Hematoma leg after trauma.

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Fig. 3.5: Subungual hematoma.



Fig. 3.6: Abrasion over the face due to trauma.



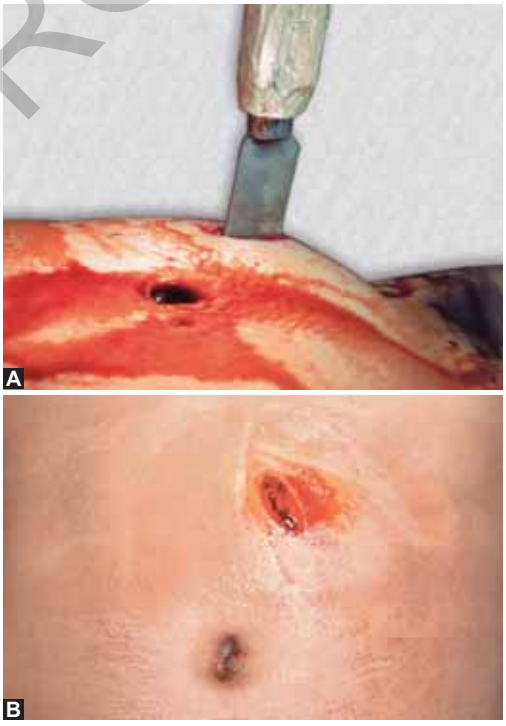
Fig. 3.7: Degloving injury in leg.

  - Muscle ischemia
  - Gangrene, loss of tissue.

10. War wounds and gunshot injuries.
11. Injuries to ***bones*** and ***joints*** may be open or closed.
12. Injuries to ***nerves***, either clean cut or crush.
13. Injuries to ***arteries*** and ***veins*** (major vessels).
14. Injury to ***internal organs*** may be penetrating or non-penetrating (blunt) injuries (Figs. 3.8A and B).

III. CLASSIFICATION (Surgical Wounds)

1. **Clean wound**
   - Herniorrhaphy
   - Excisions
   - Surgeries of the brain, joints, heart transplant.
   - Infective rate *is less than 2%* (Fig. 3.9).



Figs. 3.8A and B: Stab wound in the abdomen using a sharp knife. It is a penetrating trauma.

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Fig. 3.9: Clean thyroidectomy wound.

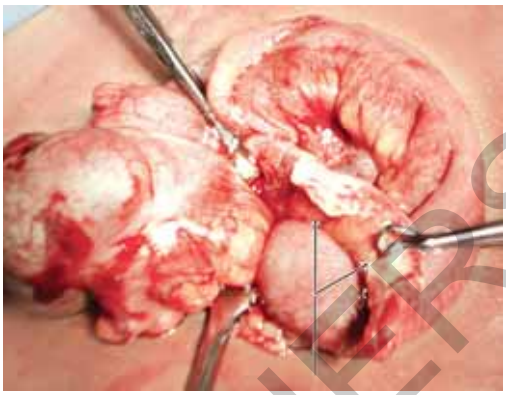


Fig. 3.11: Contaminated wound in suppurative appendicitis.



Fig. 3.10: Clean contaminated appendicectomy wound.

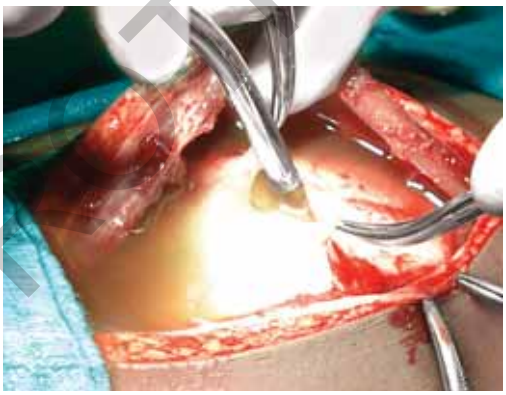


Fig. 3.12: Dirty wound of ileal perforation with peritonitis.

2. **Clean contaminated wound**
   - Appendicectomy (Fig. 3.10).
   - Bowel surgeries.
   - Gallbladder, biliary and pancreatic surgeries.
   - Infective rate is *up to 30%—high.*
3. **Contaminated wound (Fig. 3.11)**
   - Acute abdominal conditions.
   - Open fresh accidental wounds.
   - Infection rate up to 60%.
   - Infection rate >60%.
4. **Dirty infected wound (Fig. 3.12)**
   - Abscess drainage.
   - Pyocele.
   - Empyema gallbladder.
   - Fecal peritonitis.

Wound may be

- ***Closed wound*** like contusion, abrasion.
- ***Open wound*** like incised, lacerated, penetrating or crush injury.

INFLAMMATION

It is *response of living tissue to injury*; injury may be of any type—trauma, burns, surgery, infection, chemicals or drugs and so on. Inflammation can be *acute and or chronic.*

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Features of Acute Inflammation

- *Rubor; tumor; calor; dolor and functio laesa.*
- *'Triple response'*: Brief blanching, followed by reddening, flare and wheal.
- *Microscopic features*: Dilatation of vessels; sludging of red cells; fluid leaks into the interstitium with increased permeability of vessels; tissue edema; neutrophil margination and emigration.
- *A transudate* has low protein content, usually caused by alterations in hydrostatic or oncotic pressure; implies a hydrostatic (pressure) problem. An *exudate* has high protein content, caused by increased vascular permeability; implies an inflammatory process.
- White blood cells *margination and emigration*; implies binding to endothelium then directional movement through vessel wall towards injured area.
- *Vasodilatation* increases delivery, increases temperature, removes toxins. *Exudate* delivers immunoglobulins, etc. dilutes toxins, delivers fibrinogen, and increases lymphatic drainage.
- *Increased lymphatic drainage* delivers bugs to phagocytes and antigens to immune system.
- *Cells* remove pathogenic organisms, necrotic debris, etc.
- Pain and loss of function enforces rest, reduces chance of further traumatic damage.
- *Acute phase response* with decreased appetite, altered sleep patterns and changes in plasma concentrations of *acute phase proteins* like—C-reactive protein (CRP), α_1 antitrypsin, haptoglobin, fibrinogen.

Phases of Inflammation

- *Immediate early response:* Histamine is mainly released from mast cells, basophils and platelets, in response to many stimuli. It lasts for half an hour.
- *Immediate sustained response:* It is due to direct damage to endothelium.
- *Delayed response*: It is lasts from 3 hours onwards due to various chemical mediators of inflammation.
- *Resolution of inflammation:* Vessel caliber and permeability becomes normal; neutrophils become normal; fibrin degradation sets in; healing occurs with scarring; tissue regenerate begins.

Chemical mediators of acute inflammation

- *Proteases:* Kinins (bradykinin and kallekrein); Complement system; Coagulation/fibrinolytic system
- *Prostaglandins/leukotrienes*: Numerous metabolites of arachidonic acid; Synthesis blocked by NSAIDS, e.g. aspirin.
- *Cytokines/chemokines*: Interleukins, PAF, TNF alpha, PDGF, TGF beta, MCP.
- Products from platelets: 5-hydroxy tryptamine, histamine, ADP; Platelet-derived growth factor, coagulation proteins.
- Products from neutrophils: Lysosomal constituents; Products released on neutrophil death.
- Products from endothelium: PGI_2 (prostacyclin); Nitric oxide; Endothelin; Plasminogen activators/inhibitors.
- Oxygen derived free radicals: Endothelial damage, inactivation of antiproteases, injury to other cells.

Definitions

- **Phagocytosis:** It involves—Margination; Emigration; Chemotaxis.
- **Neutrophil movement:** Diapedesis and emigration; Chemotaxis

After phagocytosis, lysosomes activate forming phagosomes which kill the agents like bacteria using either oxygen dependent or oxygen independent mechanisms using lysozymes, hydrolases, lactoferrins, cationic proteins.

Factors related to inflammation

- Diabetes mellitus.
- Deficiencies—nutritional; anemia, vitamin deficiencies.
- HIV, steroid therapy.

Contd...

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- Associated illness like bronchial asthma, tuberculosis, hypertension, cardiac, renal and liver diseases.
- Habits: Smoking, alcohol; tobacco.

Presentations of Inflammation

- *Fever* due to inflammatory and pyrogenic response.
- *Pain*—severity depends on the extent of injury and response.
- *Leukocytosis*.
- *Shock* with tachycardia, hypotension, tachypnea.
- *Effects* on other systems like kidney, liver, lungs, heart, brain.

Problems and Sequelae Caused by Inflammation

- *Local*: Swelling; pain; blockage of tubes like trachea, biliary system, intestine; abscess formation; loss of function.
- *Systemic:* Acute phase response; sepsis; multiorgan failure; spread of bacteria and toxins; SIRS, etc.

Management

- *Evaluation*: Blood count; Peripheral smear; evaluation for specific conditions like liver or kidney function tests, blood sugar, imaging, CRP, other biochemical markers; Blood/urine/fluid cultures.
- *Treatment*: Anti-inflammatory drugs; *Rest*; antibiotics, drainage of an abscess; treating the specific conditions.

WOUND HEALING

Wound healing is complex method to achieve anatomical and functional integrity of disrupted tissue by various components like neutrophils, macrophages, lymphocytes, fibroblasts, collagen; in an organized staged pathways—hemostasis → inflammation → proliferation → matrix synthesis (collagen and proteoglycan ground substance) → maturation → remodeling → epithelialization → wound contraction (by myofibroblasts).

For proper wound healing, wounds must have a minimum oxygen tension of 30 mm Hg for normal cell division, and a minimum of 15 mm Hg for fibroblast proliferation. Phagocytosis of bacteria depends on a high partial oxygen pressure in the tissues. Adequate oxygenation is also required for cell proliferation, angiogenesis, collagen synthesis and reepithelialization.

The following clinical signs indicate poor wound healing: Persistent inflammation for longer than 7 days, malodorous wound and increased exudate and delayed epithelialization, maceration of the surrounding skin, wound dehiscence, and necrotic tissue.

Types of wound healing

- **Healing by primary (first) intention:** It occurs in a clean incised wound which is not contaminated. It leads into thin, linear, clean scar.
- **Healing by secondary (second) intention:** It occurs in a wide wound which is cannot be opposed. Here healing takes longer time; healing leads into wider, poor scar. Infection in the wound is a common factor.
- **Healing by tertiary (third) intention:** Here initially wound is left open to granulate and later is apposed by delayed primary closure using sutures once infection and edema are reduced.

Stages

- Stage of inflammation.
- Stage of granulation tissue formation and organization. Here as the result of fibroblastic activity, synthesis of collagen and ground substance occurs.
- Stage of epithelialization—it occurs in 48 hours.
- Stage of wound contraction and connective tissue formation.

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Factors affecting wound healing	
Local factors	***General factors***
1. Infection	Age
2. Presence of necrotic tissue and foreign body	Malnutrition
3. Poor blood supply	Vitamin deficiency (Vitamin C)
4. Venous or lymph stasis	Anemia
5. Tissue tension	Malignancy
6. Hematoma	Uremia
7. Large defect or poor apposition	Jaundice
8. Recurrent trauma	Diabetes
9. X-ray irradiated area	HIV and immunosuppressive diseases
10. Site of wound, e.g. wound over the joint and back has poor healing	Steroids and cytotoxic drugs

- Stage of scar formation and resorption.
- Stage of maturation.

Phases of Wound Healing

Inflammatory Phase (Lag/Substrate/Exudative)

It begins immediately after wound formation lasting for 4–6 days (Rubor, calor, tumor, dolor and loss of function—features of inflammation will develop). Macrophages secrete fibroblastic growth factor, which enhances angiogenesis. Polymorphonuclear leukocytes appear after 48 hours, which secrete inflammatory mediators and bacterial oxygen derived free radicals. Factors involved are—platelet derived growth factor (PDGF), epidermal growth factor (EGF), transforming growth factor (TGF), interleukins, tumor necrosis factor (TNF), prostaglandins, collagenase and elastase.

Proliferative Phase

It begins within 7 days and lasts for 6 weeks. Here, collagen and glycosamines are produced by fibroblasts. Hydroxyproline and hydroxylysine are synthesized by enzymes using iron, alpha ketoglutarate and vitamin C. About 50% of strength is achieved in 30 days.

Remodeling Phase (Maturation Phase)

It begins at 6 weeks and lasts for 6 months to 2 years. There is maturation of collagen by cross-linking and realignment of collagen fibers which is responsible for tensile strength of the scar.

Phases of wound healing

Vascular response → blood coagulation/thrombosis → inflammation → new tissue formation → epithelialization → wound contraction → remodeling.

Management of Wounds

- Wound is inspected and classified as per the type of wound.
- If it is in the vital area, then:
  - The airway should be maintained.
  - The bleeding if present should be controlled.
  - Intravenous fluids are started.
  - Oxygen, if required may be given.
  - Deeper communicating injuries and fractures, etc. should be looked for.
- If it is an incised wound, then primary suturing is done after thorough cleaning.
- If it is a lacerated wound then the wound is excised and primary suturing is done.
- If it is a crushed or devitalized wound, there will be edema and tension in the wound. *Wound debridement* is done by excising all the devitalized tissues and the edema is allowed to subside in 5–6 days. Then, delayed primary suturing is done.
- If it is a deep devitalized wound, after wound debridement, it is allowed to granulate completely. Later, if the wound is small, secondary suturing is done.

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Fig. 3.13: Degloving injury, thigh and leg. It is extensive and needs regular dressing, debridement and later skin grafting.

If the wound is large, a split skin graft (Thiersch graft) is used to cover the defect (Fig. 3.13).

- In a wound with tension, fasciotomy is done so as to prevent the development of compartment syndrome.
- Vascular or nerve injuries are dealt with accordingly. Vessels are sutured with 6-zero polypropylene nonabsorbable suture material. If cut ends of the nerves are having clean-cut, it can be sutured primarily with polypropylene 6-zero or 7-zero suture material. If there is difficulty in identifying cut ends of nerves or if the cut ends of nerves are crushed then a marker stitch using silk is placed at the site and later secondary repair of the nerves are done.
- Internal injuries have to be dealt accordingly (intracranial by craniotomy, intrathoracic by intercostal tube drainage, intra-abdominal by laparotomy). Fractured bones also should be identified and properly dealt with.
- Antibiotics, fluid and electrolyte balance, blood transfusion, tetanus toxoid or antitetanus globulin injection (ATG).

Wound Debridement (Wound toilet, or wound excision) is liberal excision of all devitalized tissues at regular intervals (of 48–72 hours) until healthy, bleeding, vascular tidy wound is created.

Primary suturing means suturing the wound immediately. It is done in clean incised wounds.
Primary suturing after wound excision is done within 6 hours when wound edges are ragged, devitalized or if wound contains foreign bodies. Proper irrigation and excision of devitalized tissues is done and sutured.

Contd...

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Delayed primary suturing means suturing the wound in 48 hours. It is done in lacerated wounds, wound with edema, hematoma or contamination. This time is allowed for the edema to subside. Wound excision may be added whenever required.

Secondary suturing means suturing the wound in 10–14 days. It is done in infected wounds. Secondary suturing is done once the infection is controlled and healthy granulation tissue appears.

Specific characteristics of the wound healing in oral cavity

Wound healing in the oral cavity is a complex process with high bacterial and viral load with complex anatomy and outcome like speech, swallowing, cosmesis, reconstruction, implants during surgeries.

In the oral cavity, healing process varies in palate, bone, dental epithelium, periodontal region, facial burns, large surgical defects, reconstructed flaps and implants.

There may be excessive bleeding or absence of blood clot formation as seen in alveolitis sicca; the granuloma formation; sinus polyps; fistulas; wound dehiscence; ulcers; perforations; wound necrosis; flap necrosis; pus formation; chronic infections with or without granulation tissue formation; keloid formation; fibrosis, and trismus.

Special factors affecting the oral cavity wound healing

- *Anatomical:* Extent of involvement of soft tissues and bone; skin and mucosal loss; posterior extension; bilateral or unilateral; dentures, implants used; flap reconstruction.
- *Comorbid status:* Diabetes mellitus, steroid therapy, vitamin and protein deficiency, hypoxia, anemia, alcohol intake, smoking and tobacco use, immunosuppression, HIV infection.
- Previous surgery or radiotherapy or chemotherapy.
- Recurrence of the disease; in case of malignancy, stage of the malignant tumor.
- Infection, salivation, aspiration.
- *Trismus due to fibrosis* of the wound is a major problem in oral cavity wounds.

Management of oral cavity wounds

- *Evaluation of the wound for*—its anatomical location, extent, defect, structures involved, cosmetic postulation, functional outcome.

Contd...

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- Reconstruction using different flaps like pectoralis major, myocutaneous flap, foreheads flap, local flaps (nasolabial), free flaps using microanastomosis (osteomyocutaneous flap).
- *Prevention* of infection, aspiration pneumonia, avoiding wound dehiscence; proper rest to oral cavity by nasogastric tube feeding, interdental wiring; positioning of the area to avoid tension over the flap or kinking.
- *Rehabilitation*, diet, speech therapy, prevention of trismus.

PROBLEMS WITH WOUND HEALING

- *Wound infection* is common in devitalized deep difficult wounds. Diabetes, immunosuppression, cytotoxic drugs, anemia, malnutrition, malignancy increases the chances of wound infection.
- *Wound dehiscence* is common in all the above-mentioned adverse factors. Wound suddenly gives away with pain causing copious serosanguineous discharge. Laparotomy when done especially as an emergency procedure as in trauma, acute abdomen and also in malignancy, abdominal closed wound may burst in 5–7 days. Usually, all layers of abdomen give away causing discharge, occasionally bowel will extrude out. It needs emergency closure of the abdominal wound using specialized sutures or retention sutures.
- *Hypertrophic scar or keloid* formation due to altered collagen synthesis in the wound-healing process. Collagen synthesis is increased *three times* in hypertrophic scar and *20 times* in keloid.
- Deeper wound will cause *specified problems* like paresthesia, ischemia, paralysis, etc.

COMPARTMENT SYNDROME

- It is common in calf and forearm.
- Closed injury causes hematoma leading to increased pressure.
- It is often associated with fracture, which compresses the major vessel further aggravating the ischemia causing ***pallor, pulseless, pain, paresthesia, diffuse swelling and cold limb***.
- If allowed to progress, it may eventually lead to ***gangrene or chronic ischemic contracture*** with deformed, disabled limb.
- ***Muscle necrosis*** releases myoglobulin, which is excreted in the urine damaging kidneys leading into renal failure.

Treatment

- These patients require longitudinal lengthy, deep incisions, i.e. ***fasciotomies*** to relieve the pressure and prevent compression (Fig. 3.14).
- Antibiotics.
- Bladder catheterization.
- Mannitol or diuretics to create diuresis so as to flush the kidney.
- Fresh blood transfusion.
- Hyperbaric oxygen.

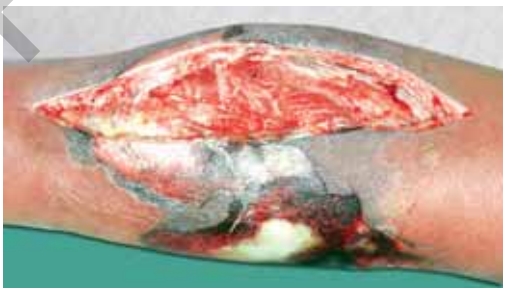


Fig. 3.14: Fasciotomy done for compartment syndrome.

CRUSH SYNDROME

It is due to crushing of muscles causing extravasation of blood and release of myohemoglobin into the circulation, leading to *acute tubular necrosis and acute renal failure.*

Causes

- Earthquakes, road traffic accidents.
- Mining and industrial accidents.

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- Air crash.
- Tourniquet.

Initially tension increases in the ***muscle compartment*** commonly in the limb, which itself impedes the circulation and increases the ***ischemic damage***. In 3 days, urine gets discolored and scanty. Patient becomes restless, apathy and delirious with onset of ***uremia***. Crush syndrome is often life-threatening.

Note: Crush injury of small area my not cause crush syndrome (Fig. 3.15).

Effects of crush syndrome

- **Renal failure.**
- **Toxemia; septicemia.**
- **Disability with extensive tissue loss.**
- **Gas gangrene.**



Fig. 3.15: Crush injury of finger.

Treatment

- Tension in the muscle compartment is relieved by placing *multiple, parallel, deep incisions* in the limb so as to prevent further damage.
- Rheomacrodex, or mannitol is given to improve the urine output by improving the renal function.
- Alkalization of the urine is done using sodium citrate or sodium bicarbonate.
- Hemodialysis is done sometimes as a life-saving procedure.
- Other measures:
  - Bladder catheterization.
  - Oxygen therapy.
  - Antibiotics.
  - Blood transfusion.

KELOID (LIKE A 'CLAW')

- Keloid is common in blacks. Common in females. Common in *Negroes*.
- Genetically predisposed. Often familial.
- There is defect in maturation and stabilization of collagen fibrils.
- Keloid continues to grow even after 6 months, may be for many years.
- It extends into adjacent normal skin.
- It is brownish black in color, painful, tender and sometimes hyperesthetic.

Sites

Common *over sternum*. Other sites are upper arm, chest wall, and lower neck in front (Figs. 3.16A and B, and 3.17A).

Differential diagnosis: Hypertrophic scar.

Treatment

Controversial.

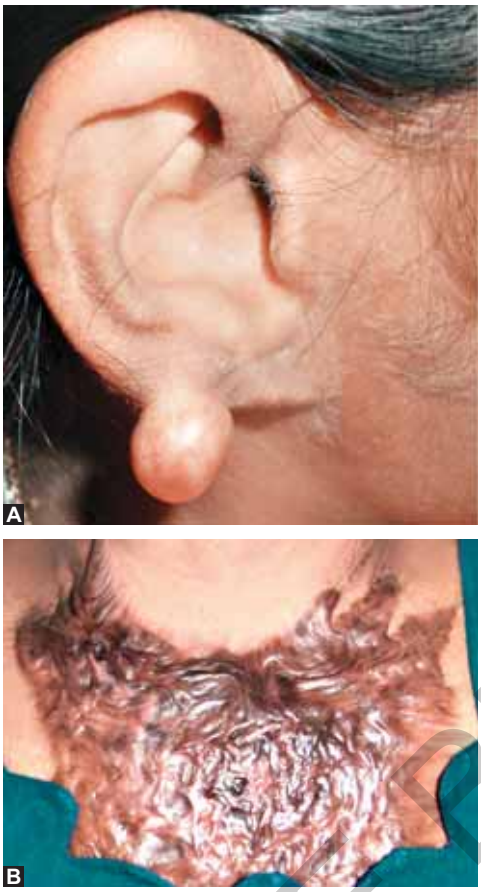
Modes of treatment

- Steroid injection: ***Triamcinolone*** is given ***intrakeloidally***, at regular intervals, may be once in 7–10 days, of 6–8 injections.
- *Intralesional excision* retaining the scar margin which may prevent recurrence. *It is ideal and better than just excision.*
- Steroid injection → Excision → Steroid injection—*not well accepted.*
- Methotrexate and vitamin A therapy into the keloid.

Recurrence rate is very high

Note: Excision and primary suturing has got high recurrence rate; hence it is not usually practiced.

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Figs. 3.16A and B: Keloid in ear. Note the keloid at sternum, which is the commonest site.



Figs. 3.17A and B: Keloid over sternum and hypertrophic scar over thigh.

HYPERTROPHIC SCAR

- It occurs anywhere in the body (Fig. 3.17B).
- Not genetically predisposed. Not familial.
- Growth usually limits up to 6 months.
- It is limited to scar tissue only. It will not extend to the normal skin.
- It is pale brown in color, not painful, non-tender.
- Often, self-limiting also. It responds very well for **steroid** injection.
- Recurrence is uncommon.
- It is controlled by pressure ***garments*** or often revision excision of scar and closure, if required with skin graft.

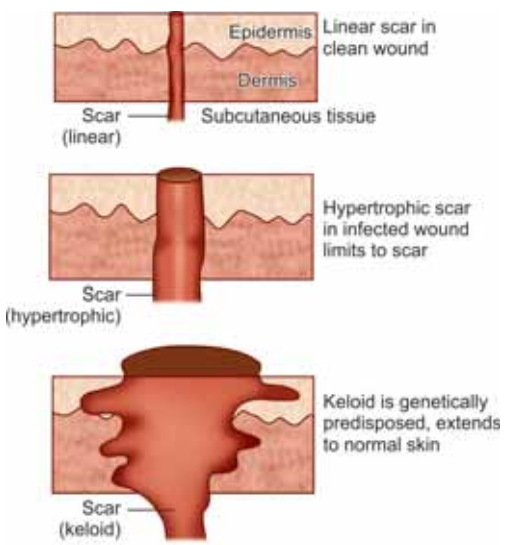


Fig. 3.18: Diagrammatic representation of linear scar, hypertrophic scar and keloid.

A mistake in judgment is never fatal, but too much anxiety about judgment is

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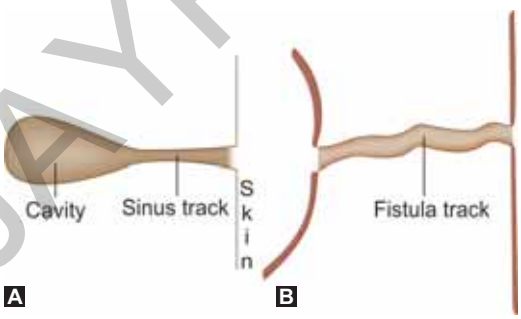
Differences between keloid and hypertrophic scar (Fig. 3.18)		
	Keloid	***Hypertrophic scar***
a. Genetic predisposition	Yes	No
b. Site of occurrence	Chest wall, upper arm, lower neck, ear	Anywhere in the body, Common in flexors
c. Growth	Continues to grow without time limit. Extends to normal skin	Growth limits for 6 months. Limited to scar tissue only
d. Treatment	Poor response	Good response to steroids
e. Recurrence	Very high	Is uncommon
f. Collagen synthesis	20 times more than normal skin	6 times more than normal skin
g. Relation of size of injury and lesion	No relation. Small healed scar can form large keloid	Related to size of injury and duration of healing
h. Age	Adolescents, middle age	Children
i. Sex	Common in females	Equal in both
j. Race	More in blacks	No racial relation

Complication

- Repeated breakdown of the scar often occurs causing infection and pain.
- After repeated breakdown it may turn into ***Marjolin's ulcer***.

SINUS (FIG. 3.19A)

- "Sinus" means "hollow" or "a bay" (Latin).
- It is a blind track lined by granulation tissue leading from an epithelial surface into the surrounding tissues.
- Sinus sprouts outside and it does not have a floor clinically.



Figs. 3.19A and B: (A) Sinus; (B) Fistula.

- Discharge can be seen on the mouth of the sinus.

Causes of Sinus

- ***Congenital:*** Preauricular sinus.
- ***Acquired:*** *Chronic osteomyelitis* of bone causing bone spicules and discharge to come out of the sinus opening. *Median mental sinus* in the mentum in lower jaw is due to tooth infection. *Pilonidal sinus* is due to entering of hairs in the interbuttock cleft over the sacrum. *Madura foot* in the foot and leg is due to Nocardia Madurai fungal infection causing multiple discharging sinuses. *Tuberculous sinus* is eventual outcome of cold abscess in neck, groin, etc. discharging cheesy, *caseating*, yellowish material.

FISTULA (FIG. 3.19B)

- It is an abnormal communication between the lumen of one viscus to another or the body surface or between the vessels.
- Fistula means "flute" or "a pipe or tube".

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Sriram Bhat M MS (General Surgery) is Professor and Head, Department of Surgery, Kasturba Medical College Mangalore, Mangaluru, Karnataka, India, and Honorary Surgeon, Government Wenlock District Hospital, Mangaluru, Dakshina Kannada, Karnataka. He, under the aegis of Manipal Academy of Higher Education (MAHE), has lived up to the principles and expectations of profession. He is also a popular practicing surgeon in Mangaluru.

A dedicated teacher, adored by his students for he has been an unending source of motivation and inspiration at viewing and understanding surgery as the subject it is. A simple person by nature, it is his love and dedication towards his profession that has made him what he is at present. With a teaching experience spanning over a period of 25 years, it will be no exaggeration if said that the art of teaching naturally comes to him. Other books written by him are: *SRB's Manual of Surgery, SRB's Clinical Methods in Surgery, SRB's Bedside Clinics in Surgery, SRB's Surgical Operations: Text and Atlas,* and *SRB's Surgery for Nurses* under Jaypee Brothers Medical Publishers.

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