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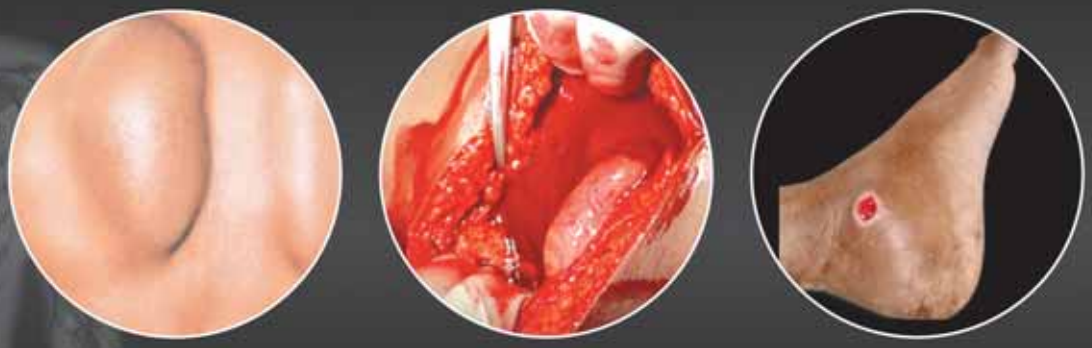

SRB's Manual of Surgery

As per the Competency-based Medical Education Curriculum (NMC)

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Forewords
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Complementary Online Learning Resource

- **Clinically Integrated MCQs**
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Competency Table

Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
Topic: Metabolic Response to Injury						
SU1.1	Describe basic concepts of homeostasis, enumerate the metabolic changes in injury and their mediators.	Y	Lecture, bedside clinic, small group discussion	Written/viva voce	1	1
SU1.2	Describe the factors that affect the metabolic response to injury.	Y	Lecture, bedside clinic, small group discussion	Written/viva voce	1	2
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Topic: Blood and Blood Components						
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Topic: Burns						
SU4.1	Elicit document and present history in a case of burns and perform physical examination. Describe pathophysiology of burns.	Y	Lecture, small group discussion	Written/viva voce	8	122
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SU4.3	Discuss the medicolegal aspects in burn injuries.	Y	Lecture, small group discussion	Written/viva voce	8	133
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Topic: Wound Healing and Wound Care						
SU5.1	Describe normal wound healing and factors affecting healing.	Y	Lecture, small group discussion	Written/viva voce	2	19
SU5.2	Elicit, document and present a history in a patient presenting with wounds.	Y	Lecture, small group discussion	Written/viva voce	2	15
SU5.3	Differentiate the various types of wounds, plan and observe management of wounds.	Y	Lecture, small group discussion	Written/viva voce	2, 22	15, 358

Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
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SU6.2	Enumerate prophylactic and therapeutic antibiotics. Plan appropriate management.	Y	Lecture, small group discussion	Written/viva voce	1, 4	13, 48
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SU7.1	Describe the planning and conduct of surgical audit.	Y	Lecture, small group discussion	Written/viva voce	55	1217
SU7.2	Describe the principles and steps of clinical research in general surgery.	Y	Lecture, small group discussion	Written/viva voce	1	10
Topic: Pre-, Intra- and Postoperative Management						
SU10.3	Observe common surgical procedures and assist in minor surgical procedures; observe emergency life-saving surgical procedures.	Y	DOAP sessions	Logbook	55	1217
SU10.4	Perform basic surgical skills such as first aid including suturing and minor surgical procedures in simulated environment.	Y	DOAP session	Skill assessment	55	1217
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SU11.1	Describe principles of preoperative assessment.	Y	Lecture, small group discussion	Written/viva voce	53	1205
SU11.2	Enumerate the principles of general, regional, and local anaesthesia.	Y	Lecture, small group discussion	Written/viva voce	53	1205
SU11.5	Describe principles of providing postoperative pain relief and management of chronic pain.	Y	Lecture, small group discussion	Written/viva voce	24	379
SU11.6	Describe principles of safe general surgery.	Y	Lecture, small group discussion	Written/viva voce	1	12
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SU13.2	Discuss the principles of immunosuppressive therapy. Enumerate indications, describe surgical principles, management of organ transplantation.	Y	Lecture, small group discussion	Written/viva voce	23	370
SU13.3	Discuss the legal and ethical issues concerning organ donation.	Y	Lecture, small group discussion	Written/viva voce	23	370
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Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
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SU16.1	Minimally invasive general surgery: Describe indications, advantages and disadvantages of minimally invasive general surgery.	Y	Lecture, demonstration, bedside clinic, discussion	Theory/practical/ orals/written/ viva voce	55	1217
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SU17.2	Demonstrate the steps in basic life support. Transport of injured patient in a simulated environment.	Y	DOAP session	Skill assessment	3	31
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SU17.5	Describe clinical features for neurological assessment and GCS in head injuries.	Y	Lecture, small group discussion	Written/viva voce	50	1150
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Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
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SU22.2	Describe the aetiopathogenesis of thyroidal swellings.	Y	Lecture, small group discussion	Written/viva voce	29	492
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SU22.4	Describe the clinical features, classification and principles of management of thyroid cancer.	Y	Lecture, small group discussion	Written/viva voce	29	492
SU22.5	Describe the applied anatomy of parathyroid.	Y	Lecture, small group discussion	Written/viva voce	30	540
SU22.6	Describe and discuss the clinical features of hypo- and hyperparathyroidism and the principles of their management.	Y	Lecture, small group discussion	Written/viva voce	30	540
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SU23.2	Describe the aetiology, clinical features, principles and management of disorders of adrenal gland.	Y	Lecture, small group discussion	Written/viva voce	30	540
SU23.3	Describe the clinical features, principles of investigation and management of adrenal tumors.	Y	Lecture, small group discussion, demonstration	Written/viva voce	30	540
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SU24.1	Describe the clinical features, principles of investigation, prognosis and management of pancreatitis.	Y	Lecture, small group discussion	Written/viva voce	37	735
SU24.2	Describe the clinical features, principles of investigation, prognosis and management of pancreatic endocrine tumours.	Y	Lecture, small group discussion, demonstration	Written/viva voce	37	735
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Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
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SU27.1	Describe the aetiopathogenesis, clinical features, investigations and principles of treatment of occlusive arterial disease.	Y	Lecture, small group discussion	Written/viva voce/skill assessment	13	193
SU27.2	Demonstrate the correct examination of the vascular system and enumerate and describe the investigation of vascular disease.	Y	DOAP session	Skill assessment	13	193
SU27.3	Describe clinical features, investigations and principles of management of vasospastic disorders.	Y	Lecture, small group discussion	Written/viva voce	13	193
SU27.4	Describe the types of gangrene and principles of amputation.	Y	Lecture, small group discussion	Written/viva voce/skill assessment	21	348
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SU27.6	Describe pathophysiology, clinical features, Investigations and principles of management of DVT and varicose veins.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	15	246
SU27.7	Describe pathophysiology, clinical features, investigations and principles of management of lymph oedema, lymphangitis and lymphomas.	Y	Lecture, small group discussion	Written/viva voce/skill assessment	16	275
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SU28.3	Describe causes, clinical features, complications and principles of management of peritonitis.	Y	Lecture, small group discussion, bedside clinic	Written/viva voce	32, 33	613, 630
SU28.4	Describe pathophysiology, clinical features, investigations and principles of management of intra-abdominal abscess, mesenteric cyst, and retroperitoneal tumors.	Y	Lecture, small group discussion, demonstration	Written/viva voce	32, 38	613, 773
SU28.5	Describe the applied anatomy and physiology of oesophagus.	Y	Lecture, small group discussion, demonstration	Written/viva voce	42	841
SU28.6	Describe the clinical features, investigations and principles of management of benign and malignant disorders of oesophagus.	Y	Lecture, small group discussion, demonstration	Written/viva voce	42	841
SU28.7	Describe the applied anatomy and physiology of stomach.	Y	Lecture, small group discussion	Written/viva voce	43	870
SU28.8	Describe and discuss the aetiology, the clinical features, investigations and principles of management of congenital hypertrophic pyloric stenosis, peptic ulcer disease, carcinoma stomach.	Y	Lecture, small group discussion	Written/viva voce/skill assessment	43	870

Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
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SU28.11	Describe the applied anatomy of spleen. Describe the clinical features, investigations and principles of management of splenic injuries. Describe the post-splenectomy sepsis—prophylaxis.	Y	Lecture, small group discussion, demonstration	Written/viva voce	36	721
SU28.12	Describe the applied anatomy of biliary system. Describe the clinical features, investigations and principles of management of diseases of biliary system.	Y	Lecture, small group discussion, demonstration	Written/viva voce	35	382
SU28.13	Describe the applied anatomy of small and large intestine.	Y	Lecture, small group discussion, demonstration	Written/viva voce	44, 45, 46	919, 944, 976
SU28.14	Describe the clinical features, investigations and principles of management of disorders of small and large intestine including neonatal obstruction and short gut syndrome.	Y	Lecture, small group discussion, demonstration	Written/viva voce	44, 45, 46	919, 944, 976
SU28.15	Describe the clinical features, investigations and principles of management of diseases of appendix including appendicitis and its complications.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	47	996
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SU29.2	Describe the clinical features, investigations and principles of management of congenital anomalies of genitourinary system.	Y	Lecture, small group discussion	Written/viva voce	49	1055
SU29.4	Describe the clinical features, investigations and principles of management of hydronephrosis.	Y	Lecture, small group discussion	Written/viva voce	49	1055
SU29.5	Describe the clinical features, investigations and principles of management of renal calculi.	Y	Lecture, small group discussion	Written/viva voce	49	1055
SU29.6	Describe the clinical features, investigations and principles of management of renal tumours.	Y	Lecture, small group discussion	Written/viva voce	49	1055
SU29.7	Describe the principles of management of acute and chronic retention of urine.	Y	Lecture, small group discussion	Written/viva voce	49	1111
SU29.8	Describe the clinical features, investigations and principles of management of bladder cancer.	Y	Lecture, small group discussion	Written/viva voce	49	1089
SU29.9	Describe the clinical features, investigations and principles of management of disorders of prostate.	Y	Lecture, small group discussion	Written/viva voce/skill assessment	49	1103
SU29.11	Describe clinical features, investigations and management of urethral strictures.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1111

Number	COMPETENCY The student should be able to	Core (Y/N)	Suggested teaching learning method	Suggested assessment method	Chapter No.	Page No.
Topic: Penis, Testis and Scrotum						
SU30.1	Describe the clinical features, investigations and principles of management of phimosis, paraphimosis and carcinoma penis.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1119
SU30.3	Describe the applied anatomy, clinical features, investigations and principles of management of epidydimo-orchitis.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1138
SU30.4	Describe the applied anatomy, clinical features, investigations and principles of management of varicocele.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1128
SU30.5	Describe the applied anatomy, clinical features, investigations and principles of management of hydrocele.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1128
SU30.6	Describe classification, clinical features, investigations and principles of management of tumours of testis.	Y	Lecture, small group discussion, demonstration	Written/viva voce/skill assessment	49	1138

CHAPTER

9 Ulcer

COMPETENCY

PM7.8 : Enumerate the causes of, describe and classify pressure sores, prevention, and treatment.
PM7.9 : Enumerate the indications of debridement, and split thickness skin grafting.
SU6.1 : Define and describe the aetiology and pathogenesis of surgical infections.

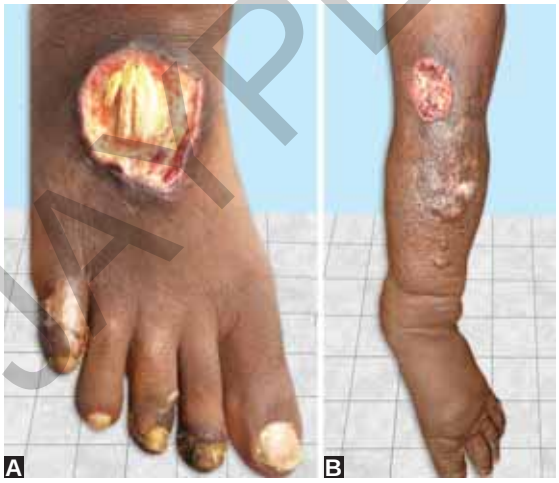
CHAPTER OUTLINE

- Ulcer
- Granulation Tissue
- Investigations for an Ulcer
- Management of an Ulcer
- Traumatic Ulcer
- Trophic Ulcer
- Ulcer due to Chilblains
- Ulcer due to Frostbite
- Martorell's Ulcer
- Arterial/Ischaemic Ulcer
- Bairnsdale Ulcer
- Carcinomatous Ulcer
- Rodent Ulcer
- Melanotic Ulcer
- Diabetic Ulcer and Diabetic Foot
- Meleney's Ulcer
- Lupus Vulgaris
- Tuberculous Ulcer
- Bazin's Disease
- Tropical Ulcer
- Venous Ulcer
- Syphilitic Ulcer
- Soft Chancre/Soft Sore/Ducrey's Ulcer/ Chancroid/Bubo
- Climatic Bubo/Tropical Bubo

ULCER

Definition

An ulcer is a break in the continuity of the covering epithelium, either skin or mucous membrane due to molecular death.



Figs. 9.1A and B: (A) Nonhealing ulcer foot; (B) Malignant (SCC) ulcer leg.

Parts of an Ulcer

a. *Margin*: It may be regular or irregular. It may be rounded or oval.
b. *Edge:* Edge is the one which connects floor of the ulcer to the margin. *Different edges are:*
Sloping edge. It is seen in a healing ulcer.
- Its inner part is *red* because of red, healthy granulation tissue.
- Its middle part is *blue* due to epithelial proliferation.
- Its outer part is *white* due to scar/fibrous tissue.

Undermined edge is seen in a tuberculous ulcer. Disease process advances in deeper plane (in subcutaneous tissue) whereas (skin) epidermis proliferates inwards.
Punched out edge is seen in a gummatous (syphilitic) ulcer and trophic ulcer. It is due to endarteritis.
Raised and beaded edge (pearly white) is seen in a rodent ulcer (BCC). Beads are due to proliferating active cells.
Everted edge (rolled out edge): It is seen in a carcinomatous ulcer due to spill of the proliferating malignant tissues over the normal skin.
c. *Floor:* It is the one which is seen. Floor may contain discharge, granulation tissue or slough.

It's only after you've stepped outside your comfort zone that you begin to change, grow, and transform.—**Roy T Bennett**

d. *Base:* Base is the one on which ulcer rests. It may be bone or soft tissue.

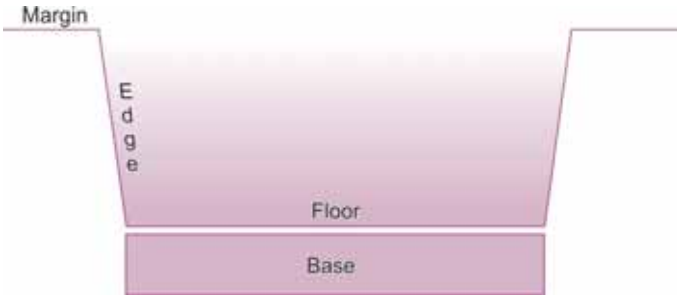


Fig. 9.2: Parts of an ulcer.

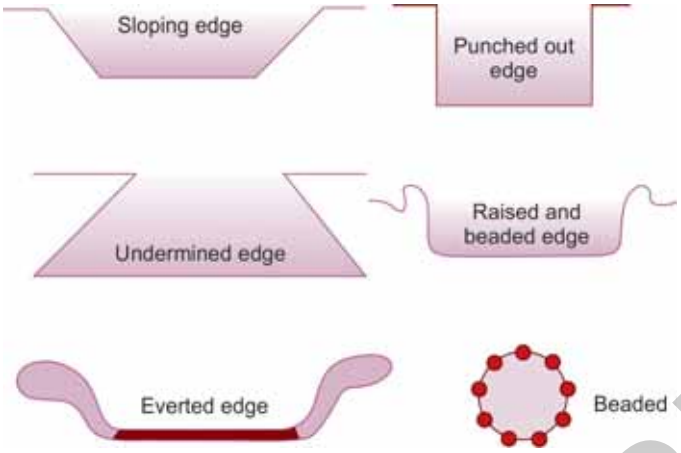


Fig. 9.3: Ulcer edges.

Induration of an Ulcer

Induration is a clinical palpatory sign which means there is a specific type of hardness in the diseased tissue. It is obvious in well-differentiated carcinomas. It is better felt in squamous cell carcinoma. It is also observed in long standing ulcer with underlying fibrosis. It is absent or less in poorly differentiated carcinomas and malignant melanoma. Less indurated carcinoma is more aggressive. Specific types of indurations are observed in venous diseases and chronic deep venous thrombosis. Brawny induration is a feature of an abscess. Induration is felt at edge, base and surrounding area of an ulcer. Induration at surrounding area signifies the extent of disease (tumour). Outermost part of the indurated area is taken as the point from where clearance of wide excision is planned.

Classifications

Classification I (Clinical)

1. ***Spreading ulcer***: Here edge is inflamed, irregular and oedematous. It is an *acute* painful ulcer; floor does not contain healthy granulation tissue (or granulation tissue is absent) but with profuse purulent discharge and slough; surrounding area is red and edematous. Regional (draining) lymph nodes are enlarged and tender due to inflammation. There will be associated fever, pain, impairment of functions with local tissue destruction and with little evidence of regeneration.

Fig. 9.4: Spreading ulcer copious purulent discharge with slough.

2. ***Healing ulcer***: Edge is sloping with healthy pink/red healthy granulation tissue with *scanty/minimal serous discharge* in the floor; slough is absent; regional lymph nodes may or may not be enlarged but when enlarged always non-tender. Surrounding area does not show any signs of inflammation or induration; base is not indurated. *Three zones* are observed in healing ulcer. *Innermost red zone* of healthy granulation tissue; *middle bluish zone* of growing epithelium; *outer whitish zone* of fibrosis and scar formation.

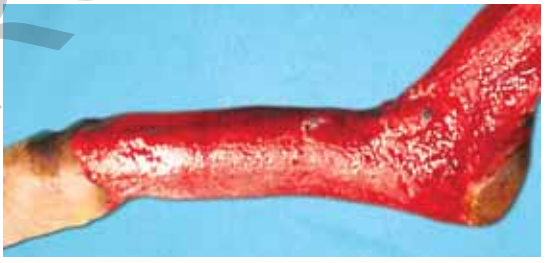


Fig. 9.5: Healing ulcer with healthy granulation tissue in the floor.

3. ***Nonhealing ulcer***: It may be a *chronic* ulcer depending on the cause of the ulcer; here edge will be depending on the cause—punched out (trophic), undermined (tuberculous), rolled out (carcinomatous ulcer), beaded (rodent ulcer); floor contains unhealthy granulation tissue and slough, and serosanguineous/purulent/bloody discharge; regional draining lymph nodes may be enlarged but non-tender.

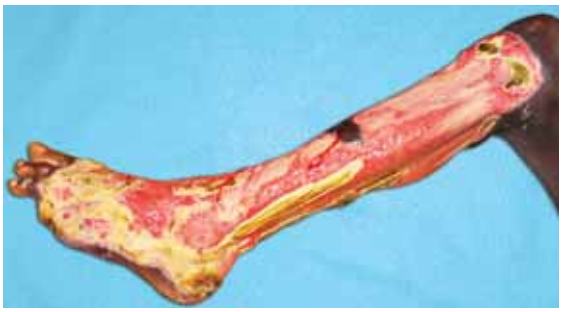


Fig. 9.6: Nonhealing ulcer with pale unhealthy granulation tissue with slough.

4. ***Callous (stationary) ulcer***: It is callousness towards healing; word "callous" means—insensitive and cruel; and also it means—hard skinned. It is also a *chronic nonhealing ulcer*; floor contains pale unhealthy, flabby, whitish yellow granulation tissue and thin scanty serous discharge or rarely copious serosanguinous discharge, with indurated nontender edge; base is indurated, nontender and often fixed. ***Ulcer does not show any tendency to heal.*** It lasts for many months to years. Tissue destruction is more with absence of or only minimal regeneration. Induration and pigmentation may be seen in the surrounding area. There is no/less discharge. Regional lymph nodes may be enlarged; are firm/hard and non-tender.

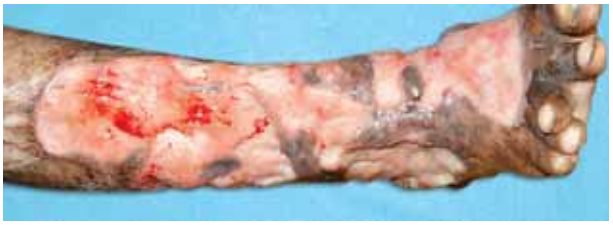


Fig. 9.7: Callous ulcer without any sign of healing and, without any granulation tissue.

Classification II (Based on Duration)

1. Acute ulcer—duration is less than 2 weeks.
2. Chronic ulcer—duration is more than 2 weeks (long).

Classification III (Pathological)

1. *Specific ulcers:* Tuberculous; syphillitic; actinomycosis and Meleney's ulcer.
2. *Malignant ulcers:* Carcinomatous; rodent (BCC); melanotic.

Figs. 9.8A and B: Maggots seen in a chronic ulcer.

3. *Non-specific ulcers:*
   - *Traumatic ulcer*: It may be mechanical, physical, chemical—common.
   - *Arterial ulcer:* Atherosclerosis, TAO
   - *Venous ulcer:* Gravitational ulcer, post-phlebitic ulcer.
   - *Trophic ulcer/pressure sore.*
   - *Infective ulcers*: Pyogenic ulcer.

Fig. 9.9: Tuberculous ulcer ankle area. Note the undermined edge. Discharge study, biopsy and later antituberculous drugs are the treatment. They are *usually painless.*

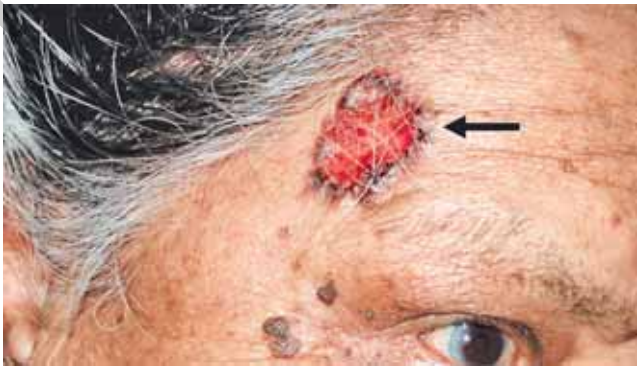


Fig. 9.10: Basal cell carcinoma of face (rodent ulcer). Ulcer edge is raised and beaded in appearance.

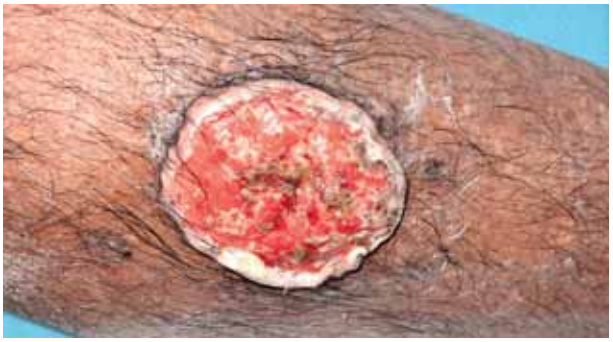
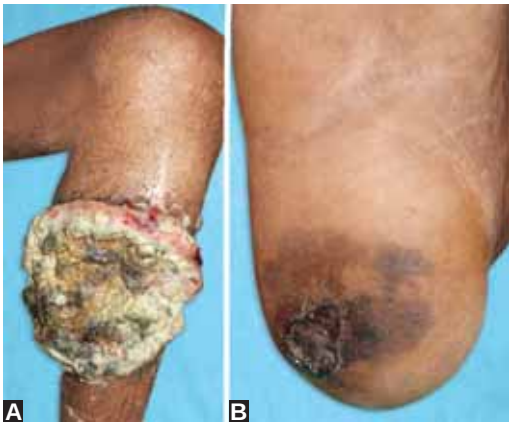


Fig. 9.11: Squamous cell carcinoma (SCC/epithelioma) leg with typical everted edge. SCC can be ulcerative, ulceroproliferative or proliferative ulcer on clinical look.



Figs. 9.12A and B: (A) Malignant proliferative ulcer; (B) Melanotic ulcer.

- *Tropical ulcers*: It occurs in tropical countries. It is callous type of ulcer, e.g., Vincent's ulcer.
- *Ulcers* due to chilblains and frostbite (*cryopathic ulcer*).
- *Martorell's hypertensive ulcer.*

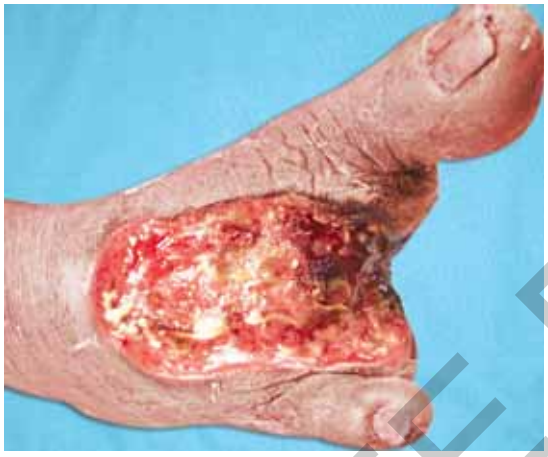


Fig. 9.13: Ischaemic ulcer foot. Middle three toes are already amputated because of gangrene.

Fig. 9.14: Venous ulcers in both feet. Site is around ankle (Gaiter's zone). There are healthy granulation tissues. It needs skin grafting and definitive procedure for varicose veins after evaluation.

- *Bazin's ulcer.*
- *Diabetic ulcer.*
- *Ulcers* due to leukaemia, polycythemia, jaundice, collagen diseases, lymphoedema.
- *Cortisol ulcers* are due to long-time application of cortisol (steroid) creams to certain skin diseases. These ulcers are callous ulcers last for long time and require excision and skin grafting.

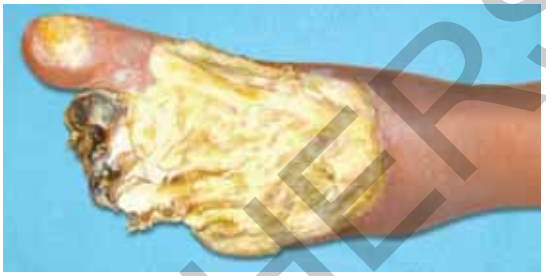


Fig. 9.15: Infective ulcer in the foot. Note the quantity of slough, exposed tendon and gangrenous toes. Patient requires below or above knee amputation.

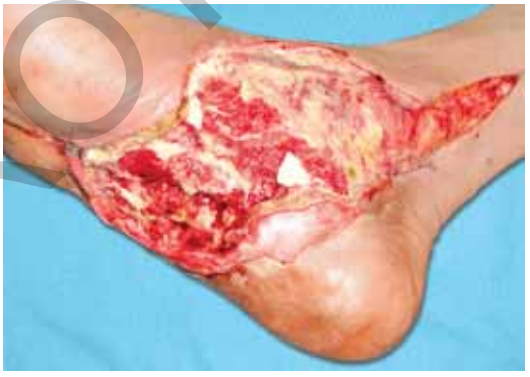


Fig. 9.16: Large ulcer in the foot and leg with exposed tendon.

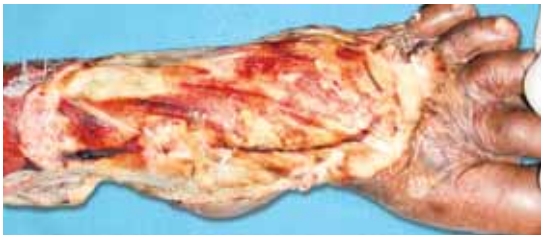


Fig. 9.17: Nonhealing ulcer foot in a diabetic patient with *Pseudomonas* infection. Note the greenish discharge in the wound. *Pseudomonas* infection is commonly hospital acquired.

Wagner's Grading/Classification of Ulcer

Grade 0 – Preulcerative lesion/healed ulcer
Grade 1 – Superficial ulcer
Grade 2 – Ulcer deeper to subcutaneous tissue exposing soft tissues or bone
Grade 3 – Abscess formation underneath/osteomyelitis
Grade 4 – Gangrene of part of the tissues/limb/foot
Grade 5 – Gangrene of entire one area/foot

Do not fear failure but rather fear not trying.—**Roy T Bennett**

BOX Stages of ulcer healing

1. *Stage of extension:* Ulcer floor is covered with slough, purulent discharge and inflamed edge and margin.
2. *Stage of transition:* Floor shows separated slough; healthy granulation tissue; serous discharge.
3. *Stage of repair:* Fibrosis, collagen deposition, scar formation occurs.

BOX Different discharges in an ulcer (as well as from a sinus)

a. *Serous*: In healing ulcer
b. *Purulent:* In infected ulcer
   - Staphylococci: Yellowish and creamy
   - Streptococci: Bloody and opalescent
   - *Pseudomonas:* Greenish colour due to pseudocyanin
c. *Bloody:* Malignant ulcer, healing ulcer from healthy granulation tissue
d. *Seropurulent*
e. *Serosanguinous*: Serous and blood
f. *Serous with sulphur granules*: Actinomycosis
g. *Yellowish:* Tuberculous ulcer

GRANULATION TISSUE

It is proliferation of new capillaries and fibroblasts intermingled with red blood cells and white blood cells with thin fibrin cover over it.

Types

- ***Healthy granulation tissue***: It occurs in a healing ulcer. It has got sloping edge. It bleeds on touch. It has got serous discharge. 5 Ps of granulation tissue—*Pink, Punctate haemorrhages, Pulseful, Painless, Pin head granulation.* Skin grafting takes up well with healthy granulation tissue. Streptococci growth in culture should be less than 10^5/gram of tissue before skin grafting.

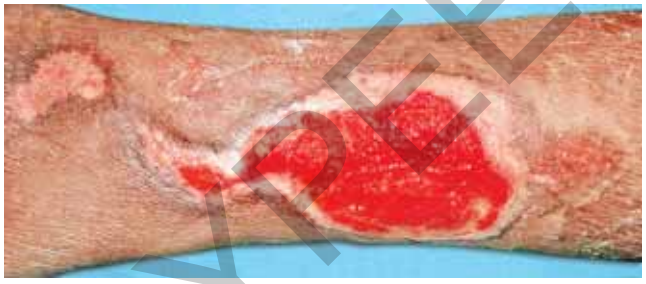


Fig. 9.18: Healing ulcer with healthy granulation tissue. Note the sloping edge.

- ***Unhealthy granulation tissue:*** It is pale with purulent discharge. Its floor is covered with slough. Its edge is inflamed and oedematous. It is a spreading ulcer.
- ***Unhealthy, pale, flat granulation tissue***: It is seen in chronic nonhealing ulcer (callous ulcer).
- ***Exuberant granulation tissue (Proud flesh)***: It occurs in a sinus or ulcer wherein granulation tissue protrudes out of the sinus opening or ulcer bed like a proliferating mass.
- ***Sprouting granulation tissue of sinus:*** It is often associated with a retained foreign body in the sinus cavity.

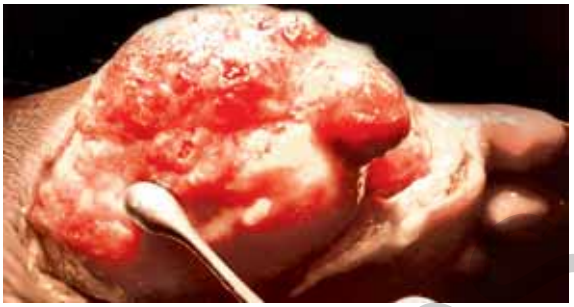


Fig. 9.19: Exuberant granulation tissue (Proud flesh) in an ulcer. It should be scooped out using Volkmann's scoop prior to skin grafting.

- ***Pyogenic granuloma***: It is a type of exuberant granulation tissue. Here granulation tissue from an infected wound or ulcer bed protrudes out, presenting as a well-localised, red swelling which bleeds on touching.
  - *Differential diagnosis*: Papilloma, skin adnexal tumours.
  - *Treatment:* Antibiotics, excision and sent for biopsy.

Fig. 9.20: Pyogenic granuloma.

INVESTIGATIONS FOR AN ULCER

- *Study of discharge*: Culture and sensitivity, AFB study, cytology.

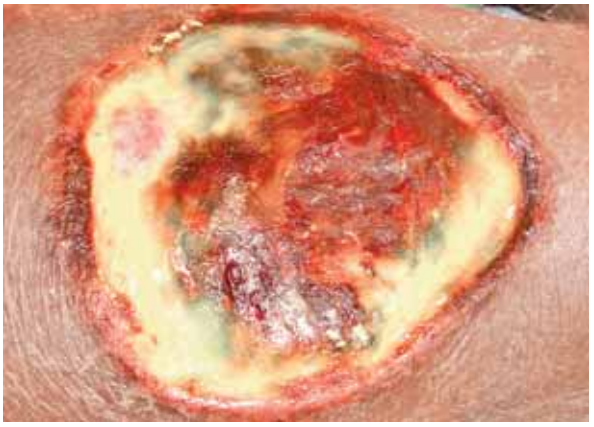


Fig. 9.21: Typical greenish coloured ulcer due to *Pseudomonas* infection.

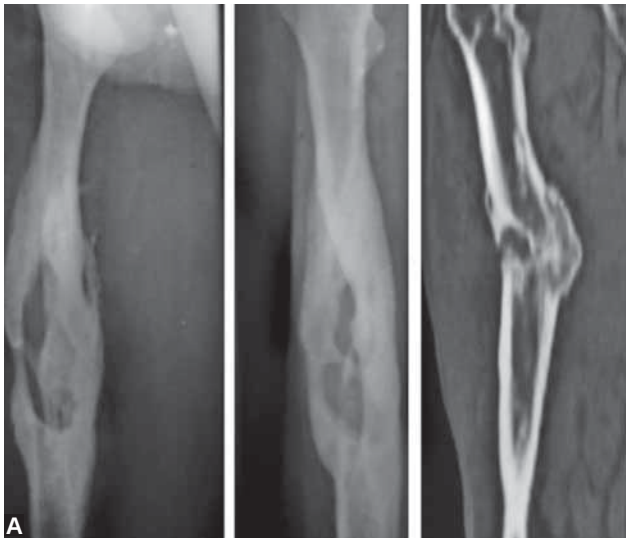


Fig. 9.22: X-ray showing osteomyelitis with sequestrum inside. Osteomyelitis prevents ulcer healing. Bone thickening on clinical examination is typical.

- *Wedge biopsy*: Biopsy is taken from the edge because edge contains multiplying cells. Usually two biopsies are taken. Biopsy taken from the centre may be inadequate because of central necrosis.
- X-ray of the part to look for periostitis/osteomyelitis.
- FNAC of the lymph node.
- Chest X-ray, Mantoux test in suspected case of tuberculous ulcer.
- Haemoglobin, ESR, total WBC count, serum protein estimation (albumin).

Note:

Ulcer will not granulate if haemoglobin is <8 g% and serum albumin is <3 g%.

BOX Assessment of an ulcer

- Cause of an ulcer should be found—diabetes/venous/arterial/infective
- Clinical type should be assessed
- Assessment of wound is important—anatomical site; size and depth of the wound; edge of the wound; mobility; fixity; induration; surrounding area; local blood supply. Wound perimeter may be useful in assessing this
- Wound imaging is done by tracing it on a transparent acetate sheet at regular intervals
- Presence of systemic features; regional nodal status; function of the limb/part; joint movements; distal pulses; sensations should be assessed
- Severity of infection should be assessed—culture of discharge
- Specific investigations like wedge biopsy; X-ray of part; blood sugar; arterial/venous Doppler; angiogram

MANAGEMENT OF AN ULCER

- Cause should be found and treated.
- Correction of the anaemia, deficiencies like of protein and vitamins.
- Proper investigation as needed.
- Transfusion of the blood if required.
- Control the pain and infection.
- *Rest, immobilisation, elevation*, avoidance of repeated trauma.
- Care of the ulcer by debridement, ulcer cleaning and dressing. Desloughing is done either mechanically or chemically. Mechanically it is done using scissor by excising the slough. Hydrogen peroxide which releases nascent oxygen is used as chemical agent. Acriflavine is antiseptic and irritant and so desloughs the area and promotes granulation tissue formation. ***Eusol*** (Edinburgh University Solution) which contains sodium hypochlorite releases nascent chlorine which forms a water-soluble complex with slough to dissolve it. Use of povidone iodine in ulcer cleaning is controversial (open wound is not suitable; it is mainly for cleaning the surgical field prior to incision). Maggots if present in the wound will cause crawling sensation and are removed using turpentine solution.
- Removal of the exuberant granulation tissue is also required when present.
- *Ulcer cleaning and dressing* is done daily or twice daily or once in 2–3 days depending on the type of ulcer and type of dressing used. Normal saline is ideal for ulcer cleaning. Various dressings are available. Films (opsite/semipermeable polyurethane), hydrocolloids (duoderm), hydrogels (polyethylene oxide with water), hydroactives (nonpectin-based polyurethane matrix), foams (polyurethane hydrophilic or hydrophobic non-occlusive), impregnates (non-adherent fine mesh impregnated with antibacterials), calcium alginates, etc.
- Topical antibiotics for infected ulcers are not essential but like framycetin, silver sulphadiazine, mupirocin may be used.
- *Vacuum-assisted closure (VAC) therapy*: It is by creation of negative pressure (25–200 mmHg), continuous or intermittent over the wound surface; it causes reduced fluid in the interstitial space, reduces oedema, increases the cell proliferation and protein matrix synthesis, promotes formation of healthy granulation tissue. Sterile foam is placed over the ulcer bed covering widely; tube drain with multiple holes is kept within it and end of the tube comes out significantly away; foam is sealed airtight using a sterile adhesive film. Tube is connected to suction system. Suction is maintained initially continuously later intermittently. Redressing is done only after 4–7 days.

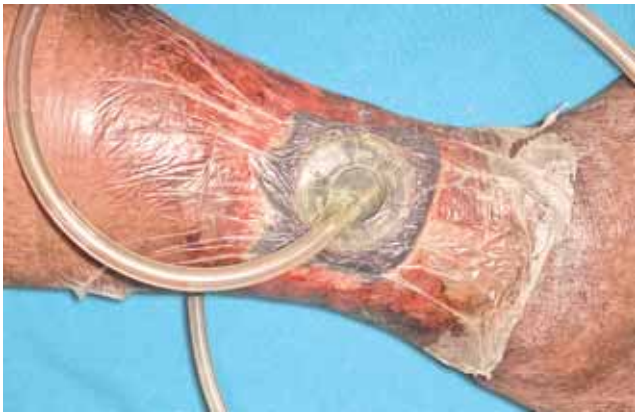


Fig. 9.23: Vacuum-assisted closure of an ulcer.

- Therapy using infrared/short wave/ultraviolet rays to decrease the ulcer size is often used but their benefits are not proved. Amnion to promote re-epithelialisation, chorion to promote granulation tissue formation is also often used.
- Antibiotics are not required once healthy granulation tissues are formed.
- *Maggot debridement therapy*: It is used as biotherapy (but not commonly) by placing cultured live disinfected maggots. Maggots are larvae of the green bottle fly, also known as the green blowfly (*Lucilia sericata*). They act by dissolving and engulfing dead necrotic tissues; they may reduce the bacterial content in the wound. They can inhibit many bacteria including MRSA (methicillin-resistant bacteria), anaerobic and aerobic bacteria. They secrete proteolytic enzymes to have mechanical effects; secretion of ammonia alters the pH in the ulcer bed which inhibits bacterial growth. They increase the granulation tissue formation also.
- Once ulcer granulates, defect is closed with secondary suturing, skin graft or flaps.

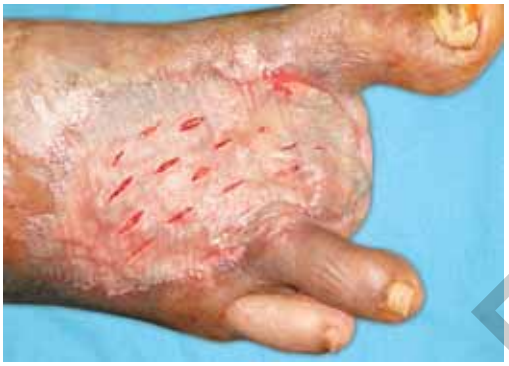
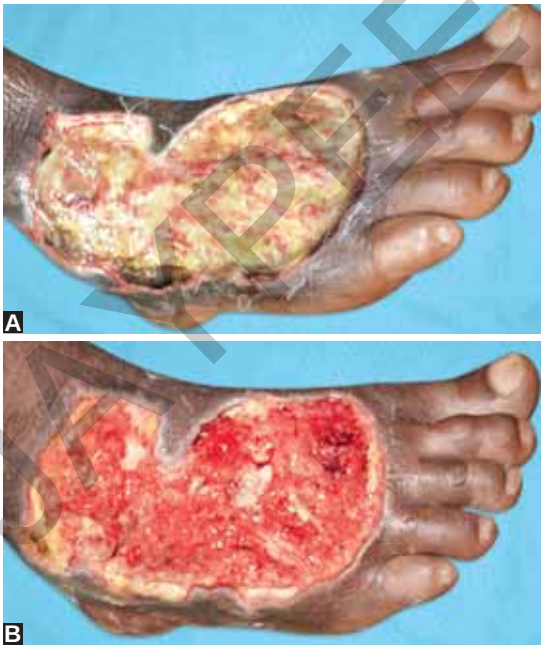
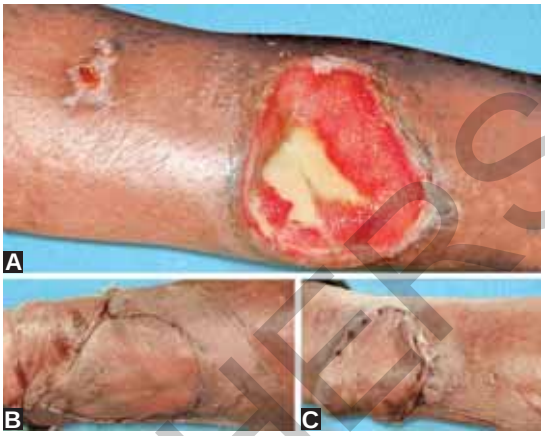


Fig. 9.24: Usually skin grafting is used to cover the defect (healthy ulcer bed).



Figs. 9.25A and B: (A) Ulcer with slough which is nonhealing; (B) It becomes healing ulcer with healthy granulation tissue which will be covered with split skin graft.

- Autologous bone marrow monocytes injection into the ulcer area is new concept by Professor Sribatsa Mohapatra but yet to confirm.



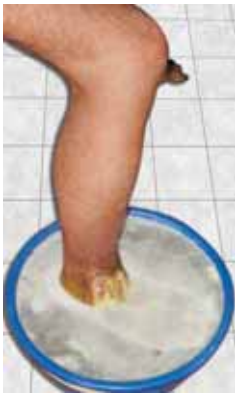
Figs. 9.26A to C: Ulcer leg with exposed bone. Patient underwent local rotation flap to cover. Area from where flap is rotated is covered with split skin graft. When the bone is exposed, skin grafting is not possible.

BOX Debridement of an ulcer

- It is removal of devitalised tissue
- Small ulcers are debrided in the ward
- Large ulcers are debrided in operation theatre under general anaesthesia
- All dead, devitalised, necrotic tissues are removed
- Slough should be separated adequately before debridement
- Often devitalised tissue separates on its own by autolysis
- Enzymes like collagenase are used for debridement
- Hydrotherapy and dressings are mechanical nonselective method of debridement
- Debridement can be surgical, mechanical, autolytic or enzymatic

BOX Dressing of an ulcer is done

- To keep ulcer moist
- To keep surrounding skin dry
- To reduce pain
- To soothen the tissue
- To protect the wound
- As an absorbent for the discharge



Figs. 9.27A and B: Solutions commonly used for ulcer and surrounding area cleaning and dressing—povidone iodine (brownish); EUSOL (colourless); hydrogen peroxide and normal saline. Note the EUSOL bath. Dilute EUSOL solution in a basin is used wherein ulcer foot is dipped and kept in place for 20–30 minutes. EUSOL removes the slough and cleans the ulcer bed. Hydrogen peroxide releases nascent oxygen and helps in removing necrotic material. *Povidone iodine is not used for open wound*; it is only a surface antiseptic.

BOX Ulcer dressings

- *Cotton dressing*—cheap but traumatic
- *Paraffin dressing*
- *Polyurethane dressings* used in clean wounds
- *Alginates (seaweed) dressing* used when there are heavy exudates
- *Type 1 collagen dressings* cause haemostasis, proliferation of fibroblasts and improve the blood supply
- *Foam dressings* are highly absorbent, decrease the wound maceration, and reduce the frequency of dressing—hydrophilic polyurethane foam
- *Hydrocolloid dressings* help in separation of slough and autolysis of dead tissues
- *Transparent film dressings* are waterproof, permit oxygen and water vapour across and prevent contamination
- *Hydrogel dressings* used for clean wounds

BOX Causes of formation of chronic/nonhealing ulcer

Local causes:

- Recurrent infection
- Trauma, presence of foreign body or sequestrum
- Absence of rest and immobilization
- Poor blood supply, hypoxia
- Oedema of the part
- Loss of sensation
- Periostitis or osteomyelitis of the underlying bone
- Fibrosis of the surrounding soft tissues
- Lymphatic diseases

General/specific causes:

- Anaemia, hypoproteinaemia
- Vitamin deficiencies
- Tuberculosis, leprosy
- Diabetes mellitus, hypertension
- Chronic liver or kidney diseases
- Steroid therapy locally or systemically
- Cytotoxic chemotherapy or radiotherapy
- Malignancy

TRAUMATIC ULCER

- Such ulcer occurs after trauma. It may be *mechanical*—dental ulcer along the margin of the tongue due to tooth injury; *physical* like by electrical burn; *chemical* like by alkali injury.
- Such ulcer is acute, superficial, painful and tender. Secondary infection or poor blood supply of the area make it chronic and deep.
- *Footballer's ulcer* is a traumatic ulcer occurring over the shin of males due to direct knocks on the shin. It is staphylococcal infection with a chronic and deep ulcer.
- Traumatic ulcers can occur anywhere in the body due to trauma.

Fig. 9.28: Traumatic ulcers in the upper limb.

Fig. 9.29: Degloving ulcer with devitalised exposed tendons on the dorsum of the hand.

- Trauma causes infection, necrosis, fasciitis, crush injury, endarteritis of the skin leading into formation of large/deep nonhealing ulcer.
- Treatment depends on size and extent of ulcer. Regular dressing, later skin grafting is done.

TROPHIC ULCER (PRESSURE SORE/ DECUBITUS ULCER)

- Pressure sore is tissue necrosis and ulceration due to prolonged pressure. Blood flow to the skin stops once external pressure becomes more than 30 mmHg (more than capillary occlusive pressure) and this causes tissue hypoxia, necrosis and ulceration. It is more prominent between bony prominence and an external surface.
- ***It is due to***—impaired nutrition; defective blood supply; neurological deficit, pressure, anaemia, injury, moisture. It is common in patients with orthopaedic and head injuries, comatose and stroke patients, old age and tetanus patients.

- ***Sites:*** Over the ischial tuberosity; sacrum; heel; heads of metatarsals; buttocks; shoulder; occiput.
- Due to the presence of neurological deficit, trophic ulcer is also called as *neurogenic ulcer/neuropathic ulcer.* Initially it begins as callosity due to repeated trauma and pressure, under which suppuration occurs and gives way through a central hole which extends down into the deeper plane up to the underlying bone as *perforating ulcer (penetrating ulcer*).
- *Bedsores are trophic ulcers.*

BOX Factors causing pressure sore

- Normal stimulus to relieve the pressure is absent in anaesthetised patient
- Nutritional deficiencies worsens the necrosis
- Inadequate padding over the bony prominences in malnourished patients
- Urinary incontinence in paraplegia patient causes skin soiling-maceration-infection-necrosis

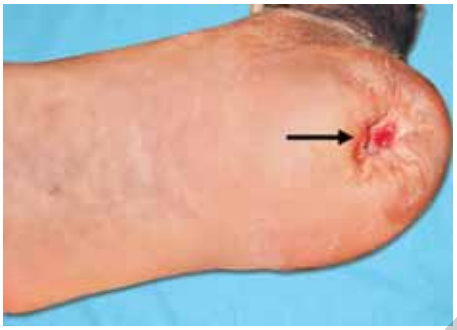
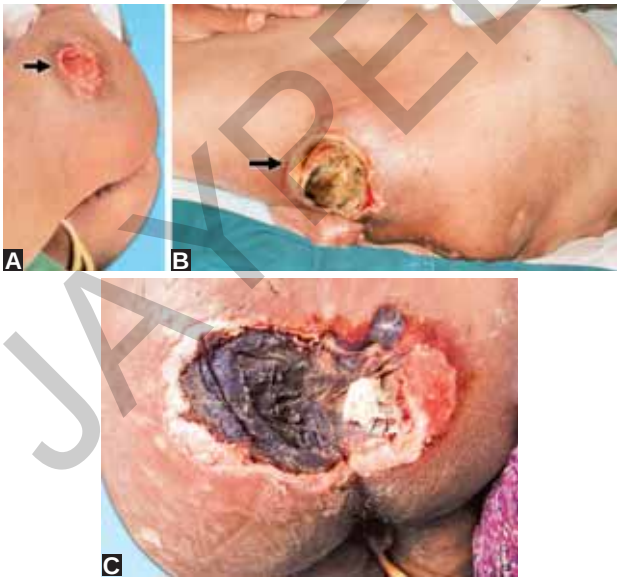


Fig. 9.30: Trophic ulcer—heel. It is deep punched out ulcer. It often requires rotation flap/transposition flap.

- ***Features:*** It occurs in 5% of all hospitalised patients. It is painless deep, nonmobile, ulcer which is punched out with base formed by bone.



Figs. 9.31A to C: *Bedsore (decubitus ulcer)* over the greater trochanter, ischium and sacrum. Bedsore is a trophic ulcer. It is usually with punched out edge.

BOX Neurological causes

- Diabetic neuropathy
- Peripheral neuritis
- Tabes dorsalis
- Spina bifida
- Leprosy
- Spinal injury
- Paraplegia
- Peripheral nerve injury
- Syringomyelia

- ***Investigations:*** Study of discharge; blood sugar; wedge biopsy from the edge; X-ray of the part and spine.

BOX Staging of pressure sore

- Nonblanching erythema—early superficial ulcer
- Partial thickness skin loss—late superficial ulcer
- Full thickness skin loss extending into subcutaneous tissue but not through fascia—early deep ulcer
- Full thickness skin loss with fascia and underlying structures like muscle/tendon/bone, etc.—late deep ulcer

- ***Treatment***
  - Cause should be treated, correction of diabetes and anaemia.
  - Nutritional supplementation.
  - Rest, antibiotics, slough excision, regular dressings.
  - *Vacuum-assisted closure (VAC).*
  - Once ulcer granulates well, flap cover or skin grafting is done.
  - Excision of the ulcer and skin grafting.
  - Flaps—local rotation or other flaps (transposition flaps).
  - Cultured muscle interposition.
  - *Proper care:* Change in position once in 2 hours; lifting the limb upwards for 10 seconds once in 10 minutes; nutrition; use of water *bed/air bed/ripple bed/air-fluid flotation bed* and pressure dispersion cushions to the affected area; urinary and faecal care; hygiene; psychological counselling. Regular skin observation; keeping skin clean and dry (using regular use of talcum powder); oil massaging of the skin and soft tissues using clean, absorbent porous clothing; control and prevention of sepsis helps in the management.

ULCER DUE TO CHILBLAINS

- It is due to exposure to intense cold causing blisters and ulcerations in the feet.
- These ulcers are ***superficial.***
- It is due to excessive cutaneous arteriolar constriction.
- The condition is also called as ***perniosis.***

ULCER DUE TO FROSTBITE

- It is due to exposure of a part to wet cold below the freezing point (cold wind).
- There is arteriolar spasm, denaturation of proteins and cell destruction.
- It leads to gangrene of the part.
- Ulcers here are always ***deep.***

MARTORELL'S ULCER (1945)

- It is seen in *hypertensive* patients often with atherosclerosis.
- It is seen in *calf*. Often it is *bilateral* and *painful*.

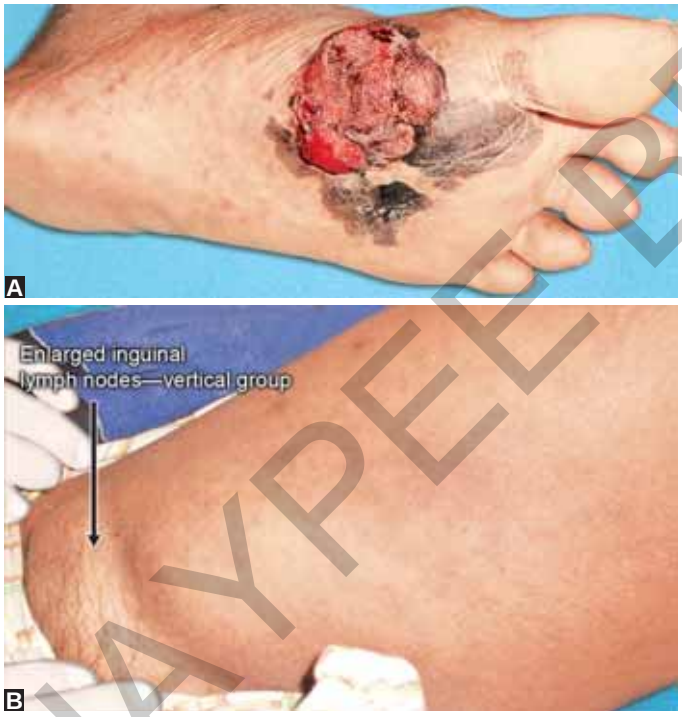
Conflict is the primary engine of creativity and innovation.—**Ranold Heifetz**

RODENT ULCER

- It is ulcerative form of basal cell carcinoma which is common in face.
- Ulcer shows central area of dry scab with peripheral raised active and *beaded (pearly white)* edge. Often floor is pigmented. It erodes into deeper plane like soft tissues, cartilages and bones hence the name—*rodent ulcer*.
- As lymphatics are blocked early in the disease by large tumour cells, it does not spread to regional lymph nodes. Blood spread is absent. It is only *locally malignant.*
- It is common in face; rarely can it occur over tibia, external genitalia, mucocutaneous junction. It does not occur in mucosa.
- ***Management:*** Wedge biopsy, CT scan of the part to see the depth, wide excision.

MELANOTIC ULCER

- It is ulcerative form of melanoma. It can occur in skin as *de novo* or in a pre-existing mole. Ulcer is pigmented often with a halo around.
- Ulcer is rapidly growing, often with satellite nodules and '*in-transit*' lesions. It is very aggressive skin tumour arising from melanocytes.



Figs. 9.35A and B: Melanotic ulcer in the foot with enlarged inguinal lymph nodes.

- It spreads rapidly to regional lymph nodes which are pigmented. Blood spread to liver, lungs, brain, and bones is common. It can occur in mucosa, genitalia, and eye. It is a systemic malignant disease.
- ***Management:*** Excision biopsy (usually *incision biopsy is not done*), FNAC lymph node, US abdomen. *Treatment* is wide local excision, regional node block dissection, chemotherapy.

DIABETIC ULCER AND DIABETIC FOOT

Diabetic Ulcer

About 15–20% of diabetes patients develop diabetic ulcers especially in the foot. It can occur in leg, back, thighs and hands other than feet. *Neuropathy, vascular problems with arterial occlusive disease and angiopathy and infection* are the main pathological events. *Demyelination in peripheral nerves* due to higher sorbitol level is the cause for neuropathy.

- ***Causes***
  - Increased glucose in the tissue precipitates infection.
  - Diabetic microangiopathy which affects microcirculation.
  - Increased glycosylated haemoglobin decreases the oxygen dissociation.
  - Increased glycosylated tissue protein decreases the oxygen utilization.
  - Diabetic neuropathy involving all sensory, motor and autonomous components.
  - Associated atherosclerosis.
- *Sites:* Foot-plantar aspect—is the most common site; leg, upper limb, back, scrotum, perineum.
  - Diabetic ulcer may be associated with ischaemia.
  - Ulcer is usually spreading and deep.
- *Investigations:* Blood sugar both random and fasting. Urine ketone bodies; discharge for culture and sensitivity; X-ray of the part to see osteomyelitis. Arterial Doppler of the limb; glycosylated haemoglobin estimation, *CT angiogram*.

Note:

Diabetic neuropathy is due to conversion of sugar to sorbitol which causes nerve demyelination; neuropathy is distal, diffuse with a stocking distribution; it takes around 10 years to develop diabetic neuropathy. Glucose forms sorbitol by aldose reductase enzyme and galactose forms galactitol. If sorbitol level raises it gets trapped in peripheral nerves, retina and lens causing neuropathy, cataract and retinopathy. There may be formation of abnormal arteriovenous communications under the skin leading into nerve ischaemia and neuropathy. ***Sensory neuropathy*** causes loss of *vibration sense, sense of position, touch*, pain and temperature. Sensation may be absent in the entire sole due to medial and plantar nerve involvement. Foot feels dead, senseless with walking like in the sand and prone for minor repeated traumas which are not noticed by the patient**.** ***Motor neuropathy*** causes paralysis of intrinsic muscles of foot leading into claw toes and hammer toe. Longitudinal arch of the foot is lost and it becomes flat causing loss of foot curvature; loss of joint position and loss of foot muscle strength leading into altered gait and positions of foot which in turn lead into deep seated trophic ulcers over the heads of the metatarsals. ***Autonomic neuropathy*** causes absence of sweating and loss of skin elasticity making more prone for breakdown and infection. Autosympathectomy is common in diabetics involving foot area.

Diabetic angiopathy may be macro- or microangiopthy (thickening of the basement membrane of vessels and capillaries).

Sepsis in diabetes: Cellulitis, deep seated abscess, ulcer formation, gangrene foot, osteomyelitis of metatarsals, septicaemia, multiorgan dysfunction syndrome can occur faster in diabetes. Phagocytic activity in diabetes is reduced significantly; granulocyte mobilization is reduced in diabetic ketoacidosis. Neuropathy, angiopathy, high tissue glucose level, associated smoking, hypertension, hyperlipidaemia, reduced immunity increases the chances of sepsis in diabetics. Polymicrobial and fungal infections are common.

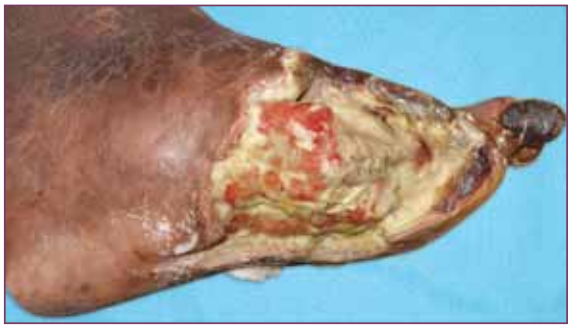


Fig. 9.36: Foot is the most common area for diabetic infective problems. It can cause abscess, ulcer, osteomyelitis, gangrene, septicaemia. Initially patient undergoes toe amputation but later eventually land with below knee or above knee amputation.

BOX Problems with diabetic ulcer

- Neuropathy, in foot—clawing of toes, hammer toe (due to intrinsic muscle paralysis)
- Multiple deeper abscesses; osteomyelitis of deeper bones are common
- Reduced leukocyte function; resistant infection; spreading cellulitis
- Arterial insufficiency
- Septicaemia; diabetic ketoacidosis
- Associated cardiac diseases like ischaemic heart disease

❖ *Treatment*

- Control of diabetes using insulin.
- Antibiotics.
- Nutritional supplements.
- Regular cleaning, debridement, dressing.
- Once granulates, the ulcer is covered with skin graft or flap.
- *Revascularisation* procedure is done by endarterectomy or thrombectomy or balloon angioplasty or arterial bypass graft. But if distal vessels are involved then success rate is less.
- Toe/foot/leg amputation.
- Microcellular rubber (MCR) shoes to prevent injuries; care of foot.

Fig. 9.37: Microcellular rubber (MCR) chappal is used in patients with diabetic neuropathy involving foot.

Diabetic Foot

Diabetic foot is a more complex disease as it involves foot complex which is anatomically, mechanically and physiologically a dynamic system as an essential part of the walk. Disease involves foot bones, muscles, arches, ligaments, tissue planes and skin. Chances of amputations at different levels are higher even though with proper care and therapy foot can be salvaged with a reasonable functioning capacity.

Problems are: Osteomyelitis of metatarsal and tarsal bones, gangrene, ulceration, callosities, corns, fungal infection of toes, nails, recurrent bacterial infection of soft tissues with abscess formation, hammer toes, ingrowing toe nails, deformities, Charcot's joint. ***Neuropathy, ischaemia and infection are most dangerous triad of diabetic foot***.

Risk factors for foot problems in diabetics: Age >50 years; smoking; hypertension; diabetes >10 years; uncontrolled diabetes; high lipid levels; foot deformity; peripheral neuropathy; peripheral occlusive arterial disease.

Diabetic neuropathy in foot: It is sensory, motor, autonomic neuropathies. Loss of sensation allows unknowingly repeated trauma. Loss of vibration, touch, pain and temperature sensations occur. Trophic ulcers, repeated sepsis sets in. Tibial, medial and lateral plantar nerves are involved. Tibial nerve involvement causes intrinsic muscle paralysis leading onto claw and hammer toes. Plantar nerve involvement leads into loss of sensation in the sole causing recurrent multiple ulcers. Foot is felt like dead with felling like walking in sand. Charcot's neuropathic joint is hypermobile unstable joints of the foot often with high vascularity. Sweating, muscle strength, functions of the arches of the foot, joint function and position and skin elasticity—are lost.

Peripheral arterial occlusive disease in foot in diabetics: It is 20% common. It may be atherosclerosis of major vessels like dorsalis pedis or posterior tibial arteries (***macroangiopathy***) leading into severe ischaemia of foot OR it may be thrombotic blockage of small vessels and arterioles causing thickening of the basement membrane and blockage of the vessels (***microangopathy***). Collateral vessels are also involved in diabetics very early aggravating the ischaemic events.

Infection events in diabetic foot: Initial recurrent cellulitis → recurrent involvement and spread along with fascial planes → multiple recurrent deep abscess formation → necrotising fasciitis with or without myonecrosis → involvement of tarsal and metatarsal bones as osteomyelitis → gangrene formation → initially distally in toes, foot, then spreading proximally into leg → sepsis/septicaemia.

Reasons for delayed healing of diabetic foot ulcers: Polymicrobial infection, neuropathy, ischaemia, poor immunity, nutritional deficiency, biofilm formation, anaemia, hypoproteinaemia, antibiotics resistance, systemic problems like cardiac diseases, smoking, hypertension, alcohol intake with liver cell dysfunction and chronic kidney disease (CKD is common as diabetic nephropathy).

- Secondary syphilitic stage shows plenty of circulating *Treponema spirochaetes* in blood whereas in tertiary stage spirochaetes are less or absent.
- *Early syphilis* lasts for 2 years and the patient is infective during this period. *Primary syphilis* occurs within 4 weeks; secondary in 12 weeks; *latent* lasts from 2 years to life time.
- ***Investigations:*** VDRL, Kahn test; *Treponema pallidum* haemagglutination test (TPHA); *Treponema pallidum* immobilisation test (TPIT).
- ***Treatment:*** Penicillin (drug of choice); doxycycline (100 mg 3 times a day for 15 days); erythromycin; tetracycline; cephalosporins. *Jarisch-Herxheimer reaction* is commonly seen after penicillin therapy.

SOFT CHANCRE/SOFT SORE/DUCREY'S ULCER/CHANCROID/BUBO

- These multiple irregular genital ulcers appear 3 days after infection with *Haemophillus ducreyi* as a venereal disease.
- They are acute painful, tender, nonindurated ulcers. Floor shows yellowish slough with purulent discharge. Edge is oedematous and inflamed. Acute regional lymphadenitis with suppuration presenting as tender, soft or firm swelling is common. Such soft fluctuant inguinal swelling is termed as ***bubo***.
- *Treatment* is by drugs like co-trimoxazole, erythromycin, ciprofloxacillin, ceftriaxone; aspiration of ***bubo***.

CLIMATIC BUBO/TROPICAL BUBO

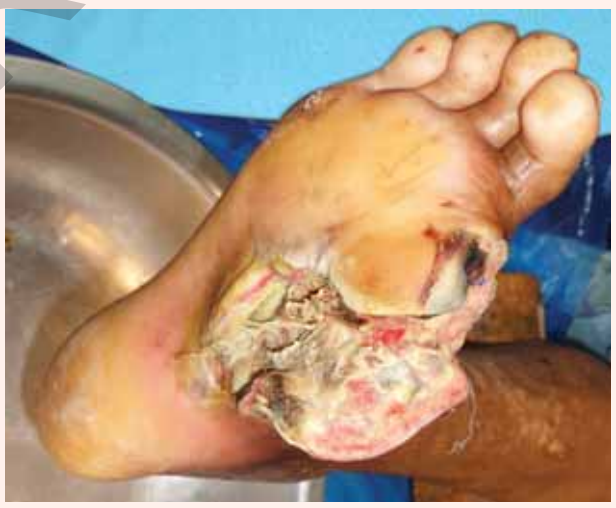
- It is due to *lymphogranuloma inguinale*, a *venereal* spreading organism (LGV, *Chlamydia* type L1, 2, 3).
- In LGV, lesion of *primary genital stage* is small, painless and commonly unnoticed.
- Lesion *of secondary stage* develops in 2 weeks. In males inguinal lymph nodes; in females intrapelvic and pararectal nodes are involved. Suppuration of inguinal nodes eventually occurs leading into discharging sinuses. *Frei intradermal test* becomes positive in 6 weeks and remains positive for life time.
- *In tertiary stage,* eye, joint, meninges may get involved after many years. Repeated chronic inflammation, lymphatic blockage, scarring can cause rectal stricture and vulval elephantiasis (*esthiomene*) in females.
- *Treatment* is tetracycline for 3 weeks.

OTHER ULCERS

- Ulcers can occur, in various parts like over shin, legs, feet, face, chest wall, in various diseases like anaemia, polycythaemia, sickle cell disease, hereditary spherocytosis, leukaemia, vasculitis, autoimmune diseases like rheumatoid arthritis, Paget's disease of bone (deep, nonmobile, fixed to bone; common in tibia), ulcerative colitis, etc.
- *Treponema pertenue* causing *Yaws* (Frambesia) can have multiple painless ulcers in leg and feet due to walking with bare foot (organism enters through abrasion) which heals spontaneously leaving a *tissue paper like scar*.
- Poor hygiene and dressings can cause multiple, small, red often scabbed *Staphylococcus aureus ulcers* on the skin over the leg and feet which is often recurrent and disturbing.
- *Buruli ulcer:* It is chronic ulcer caused by *Mycobacterium ulcerans* (acid-fast bacillus). Initially it forms a subcutaneous nodule later forming non-caseous necrosis showing slow healing with severe scarring.
- *Veld sore*: It is cutaneous diphtheria through the skin causing vesicles with a straw coloured fluid; once vesicles rupture chronic tender shallow ulcers will form.
- *Delhi boil (oriental sore)*: Here *Leishmania tropica* infection causes indurated papule on the face and exposed body parts causing indolent chronic ulcer which heals producing an ugly pigmented scar.

Case Scenario

A 55-year-old patient presents with ulcer with pain the right foot. It started 20 days back with pain, redness, swelling and fever; later skin got soughed off with discharging pus with persistent pain and fever. What is your probable diagnosis? What are the possible causes? Explain management.



Answer:
It is spreading ulcer. Cause is nonspecific; could be diabetic. Causes are—trauma, cellulitis, diabetes, smoking, bacterial infection. Pus should be sent for culture. Proper wound debridement and vacuum dressing should be done. X-ray foot, arterial Doppler, cardiac and systemic evaluation is a must. Once granulates wound cover is done by split skin grafting or appropriate flap.

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