OSTEOMYELITIS

Depart.of Orthopaedics

Osteomyelitis – Osteon – Bone

Myelo – Marrow

itis – Inflammation

DEFINITION

- Inflammation of the bone caused by an infecting organism

Introduction

- The key to successful management is early diagnosis and appropriate surgical and antimicrobial treatment.
- A multi disciplinary approach is required, involving an orthopaedic surgeon, an infectious disease specialist, and a plastic surgeon in complex cases with significant soft tissue loss.

Classification

1) The duration - acute, subacute and chronic

2) Mechanism of infection – exogenous or hematogenous

3) The type of host response to the infection- pyogenic or non pyogenic

Acute hematogenous osteomyelitis

- Most common type of bone infection, usually seen in children
- Decrease in incidence, could be due to higher standard of living and improved hygiene.
- Bimodal distribution- younger than 2 years, and 8-12 years
- More common in males

Acute hematogenous osteomyelitis- causes

- Caused by a bacteraemia
- Bacteriological seeding of bone generally is associated with other factors such as localized trauma, chronic illness, malnutrition or an inadequate immune system.

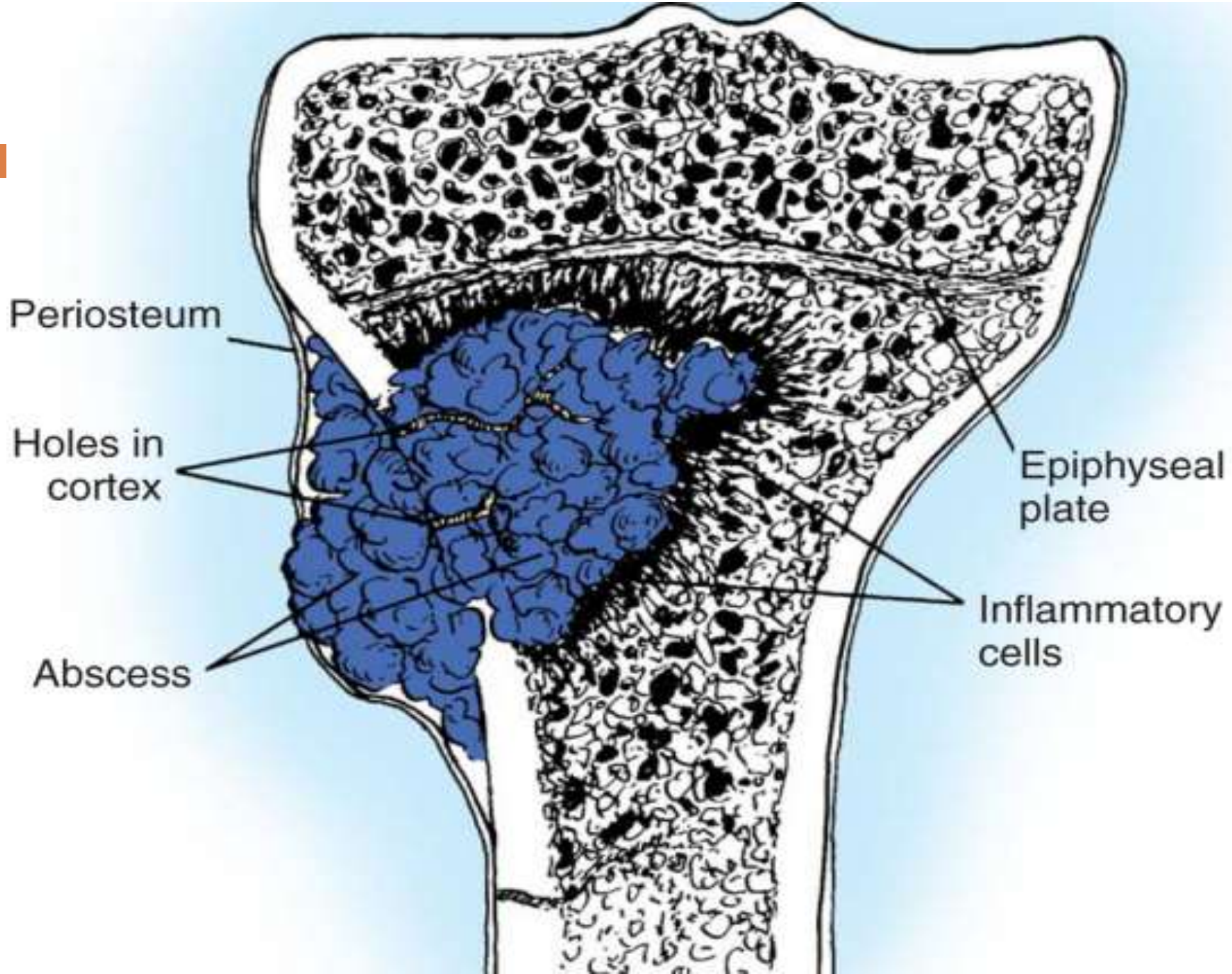
Acute hematogenous osteomyelitis
pathophysiology

- In children the infection generally involves the metaphyses of rapidly growing long bones
- Bacterial seeding leads to an inflammatory reaction which can cause local ischaemic necrosis of bone and subsequent abscess formation

Acute hematogenous osteomyelitis

pathophysiology

- As the abscess enlarges, intramedullary pressure increases causing cortical ischaemia, which may allow purulent material to escape through the cortex into the subperoisteal space.


Periosteum
Holes in cortex
Abscess
Epiphyseal plate
Inflammatory cells

Acute hematogenous osteomyelitis
pathophysiology

- A subperisoteal abscess then develops
- If left untreated this process eventually results in extensive sequetra formation and chronic osteomyelitis

Acute hematogenous osteomyelitis
pathophysiology

- In children younger than 2 years, blood vessels cross the physis, thus epiphysis may be involved

- Limb shortening or angular deformity may occur

Acute hematogenous osteomyelitis

pathophysiology

- Joint may be involved in some cases- hip joint most common, especially for intraarticular physes- proximal humerus,radial neck, distal fibula

- Metaphysis has relatively fewer phagocytic cells than the physis or diaphysis, hence more infection here

Acute hematogenous osteomyelitis
pathophysiology

- In children older than 2 years the physis effectively acts as a barrier to the spread of a metaphyseal abscess
- Metaphyseal cortex thicker, hence diaphysis more at risk
- After physes are closed acute hematogenous osteomyelitis is much less common

Acute hematogenous osteomyelitis
pathophysiology

- After the physes are closed, infection can extend directly from the metaphysis into the epiphysis and involve the joint

- Septic arthritis resulting from acute hematogenous osteomyelitis generally is seen only in infants and adults.

Acute hematogenous osteomyelitis
microbial pattern

- Staphylococcus aureus most common in older children and adults
- Gram negative bacteria- increasing trend- vertebral
- Pseudomonas most common in intravenous drug abusers
- Salmonella in sicke cell
- Fungal infections in chronically ill patients on long term intravenous therapy.

Acute hematogenous osteomyelitis
microbial pattern

- Infants- staph aureus most common but group B streptococcus and gram negative coliforms
- Prematures staph aureus andgram negative organisms
- Hemophilus influenzae primarily in children 6 months to 4 years old, incidence decreased dramatically by immunizations

Acute hematogenous osteomyelitis
diagnosis

- History and physical examination
  - Fever and malaise
  - Pain and local tenderness
  - Sweliing
  - Compartment syndrome in children

Acute hematogenous osteomyelitis
diagnosis

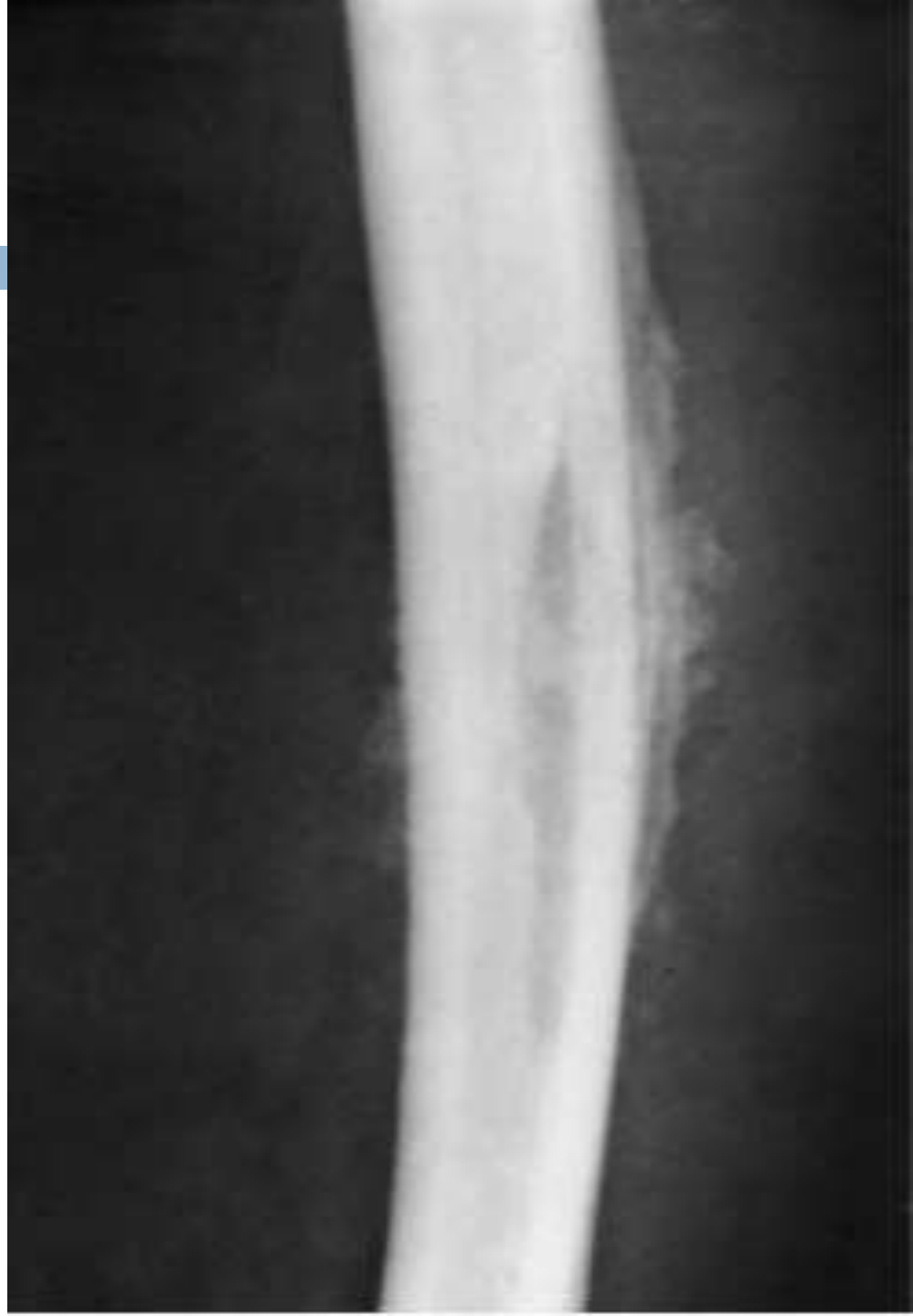
- Laboratory tests
  - White blood cell count
  - Erythrocyte sedimentation rate
  - C-reactive protein
    - checked very 2- 3 days post treatment initiation
  - Aspiration for suspected abscess

Acute hematogenous osteomyelitis
diagnosis

- Plain radiographs
- Technetium-99m bone scan +/- MRI

Radiographs

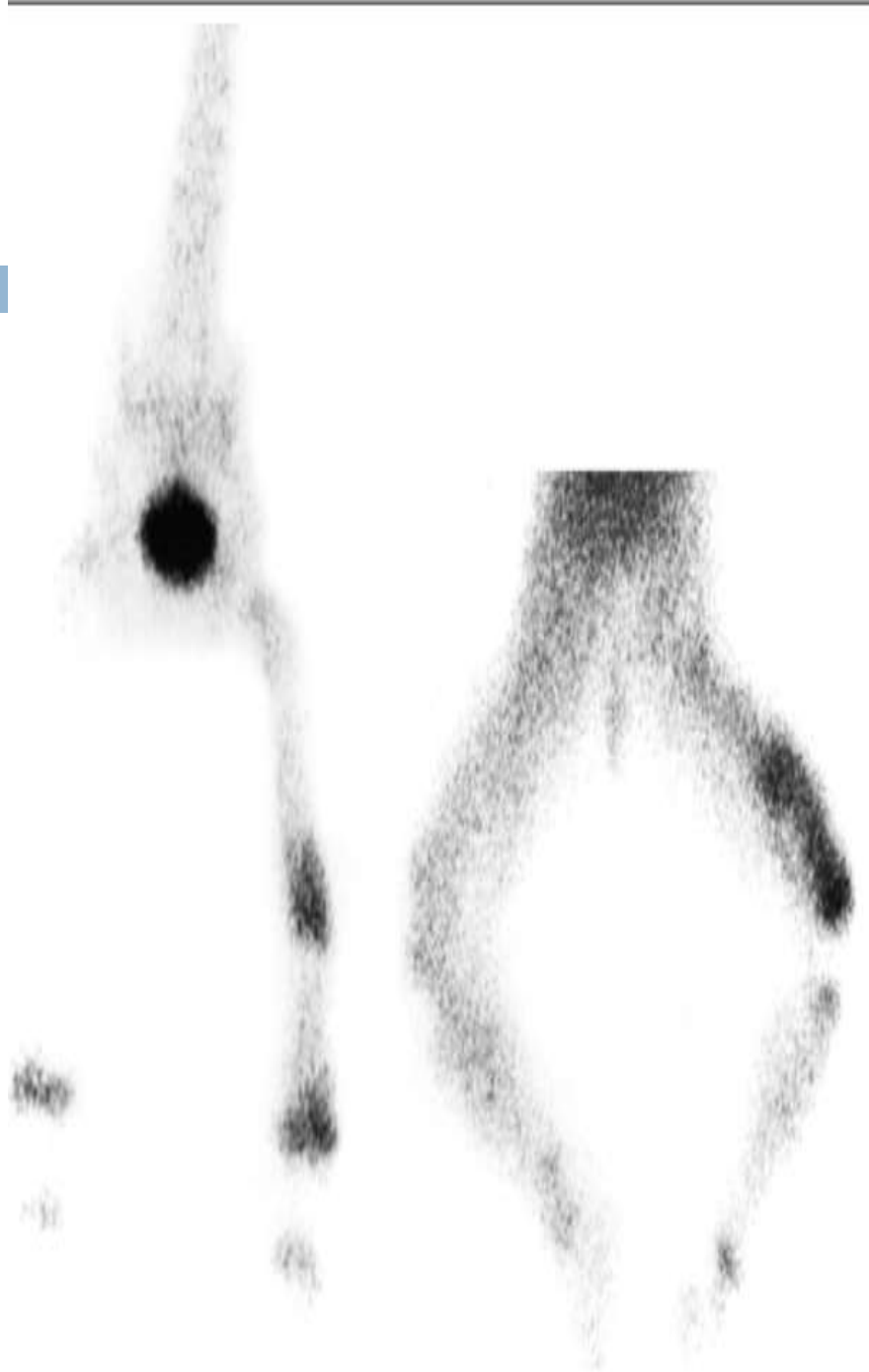
- Soft tissue swelling
- Periosteal reaction
- Bony destruction
  (10-12 days)



Bone scan

Can confirm diagnosis

24-48 hrs after onset



Treatment

- Surgery and antibiotic treatment are complementary, in some cases antibiotics alone may cure the disease.

- Choice of antibiotics is based on the highest bacteriocidal activity, the least toxicity and the lowest cost

Acute hematogenous osteomyelitis

Treatment

- Nade's 5 principles of treatment
  1. An appropriate antibiotic is effective before pus formation
  2. Antibiotics do not sterilize avascular tissues or abscesses and such areas require surgical removal

Acute hematogenous osteomyelitis

Treatment- nades principles

3. If such removal is effective, antibiotics should prevent their reformation and primary wound closure should be safe

4. Surgery should not damage already ischaemic bone and soft tissue

5. Antibiotics should be continued after surgery

Acute hematogenous osteomyelitis

Treatment

- The two main indications for surgery in acute hematogenous osteomyelitis are:
  1. The presence of an abscess requiring drainage
  2. Failure of the patient to improve despite appropriate intravenous antibiotic treatment

Acute hematogenous osteomyelitis

Treatment- surgery

- The objective of surgery is to drain any abscess cavity and remove all non viable or necrotic tissue
- Subperiosteal abscess in an infant-several small holes drilled through the cortex into the medullary canal
- If intramedullary pus is found, a small window of bone is removed
- Skin is closed loosely over drains and the limb splinted

Acute hematogenous osteomyelitis

Treatment

- Generally a 6 week course of intravenous antibiotics is given
- Orthopedic and infectious disease followup is continued for at least 1 year

SUBACUTE HEMATOGENOUS OSTEOMYELITIS

- More insidious onset and lacks severity of symptoms
- Indolent course hence diagnosis delayed for more than two weeks.

SUBACUTE HEMATOGENOUS OSTEOMYELITIS

CLINICAL FEATURES

- The indolent course of subacute osteomyelitis is due to:
  - increased host resistance
  - decreased bacterial virulence
  - administration of antibiotics before the onset of symptoms
- Systemic signs and symptoms are minimal
- Temperature is only mildly elevated
- Mild to moderate pain

SUBACUTE HEMATOGENOUS OSTEOMYELITIS

INVESTIGATIONS

- White blood cell counts are generally normal
- ESR is elevated in only 50% of patients
- Blood cultures are usually negative
- Plain radiographs and bone scans generally are positive

SUBACUTE HEMATOGENOUS OSTEOMYELITIS

INVESTIGATIONS

- S. Aureus and Staphylococcus epidermidis are the predominant organisms identified in subacute osteomyelitis

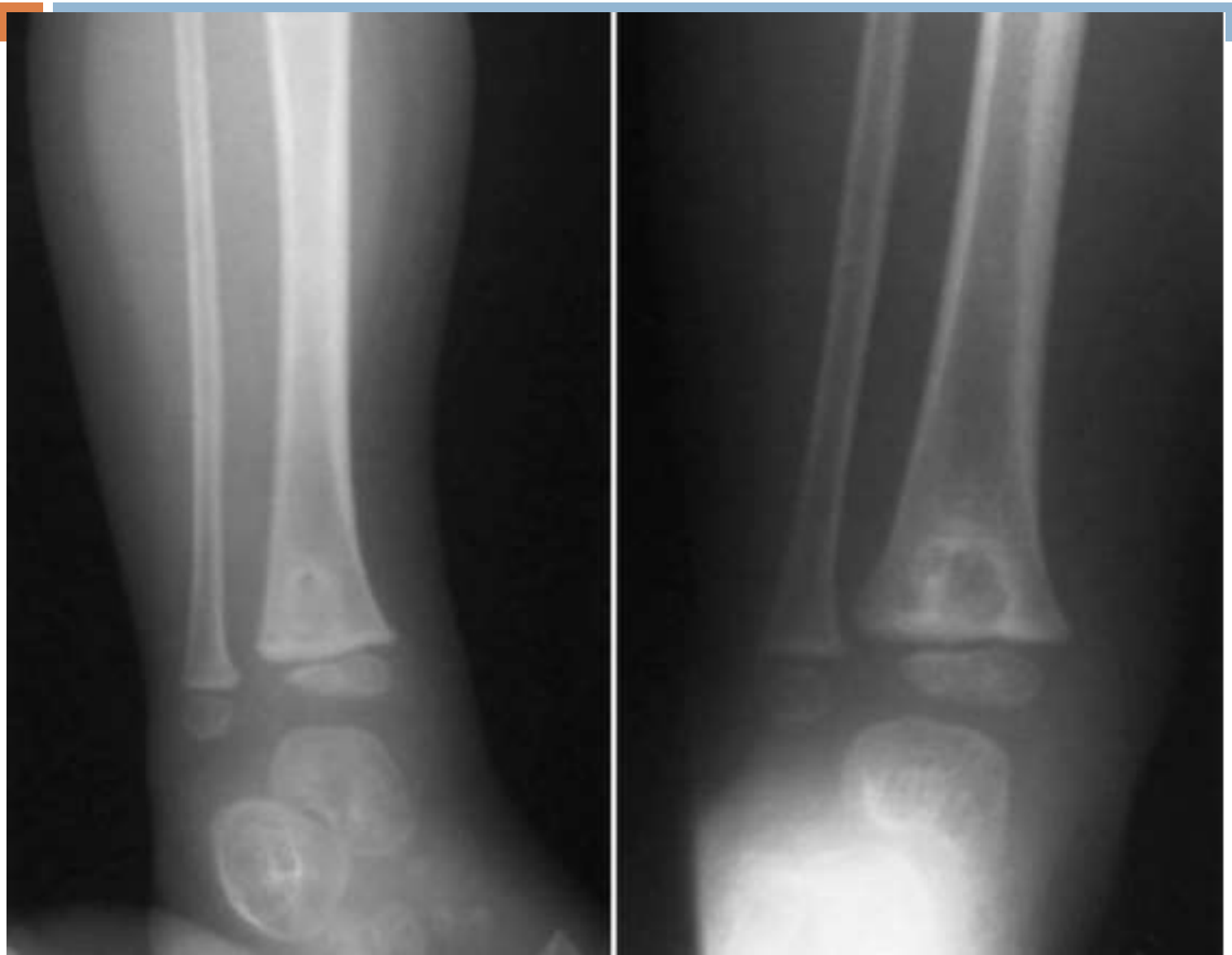
SUBACUTE HEMATOGENOUS OSTEOMYELITIS

BRODIE ABSCESS

- Localized form of subacute osteomyelitis occuring most commonly in the long bones of the lower extremeties

- Intermittent pain of long duration is most times the presenting compliant, along with tenderness over the affected area

Brodie abscess



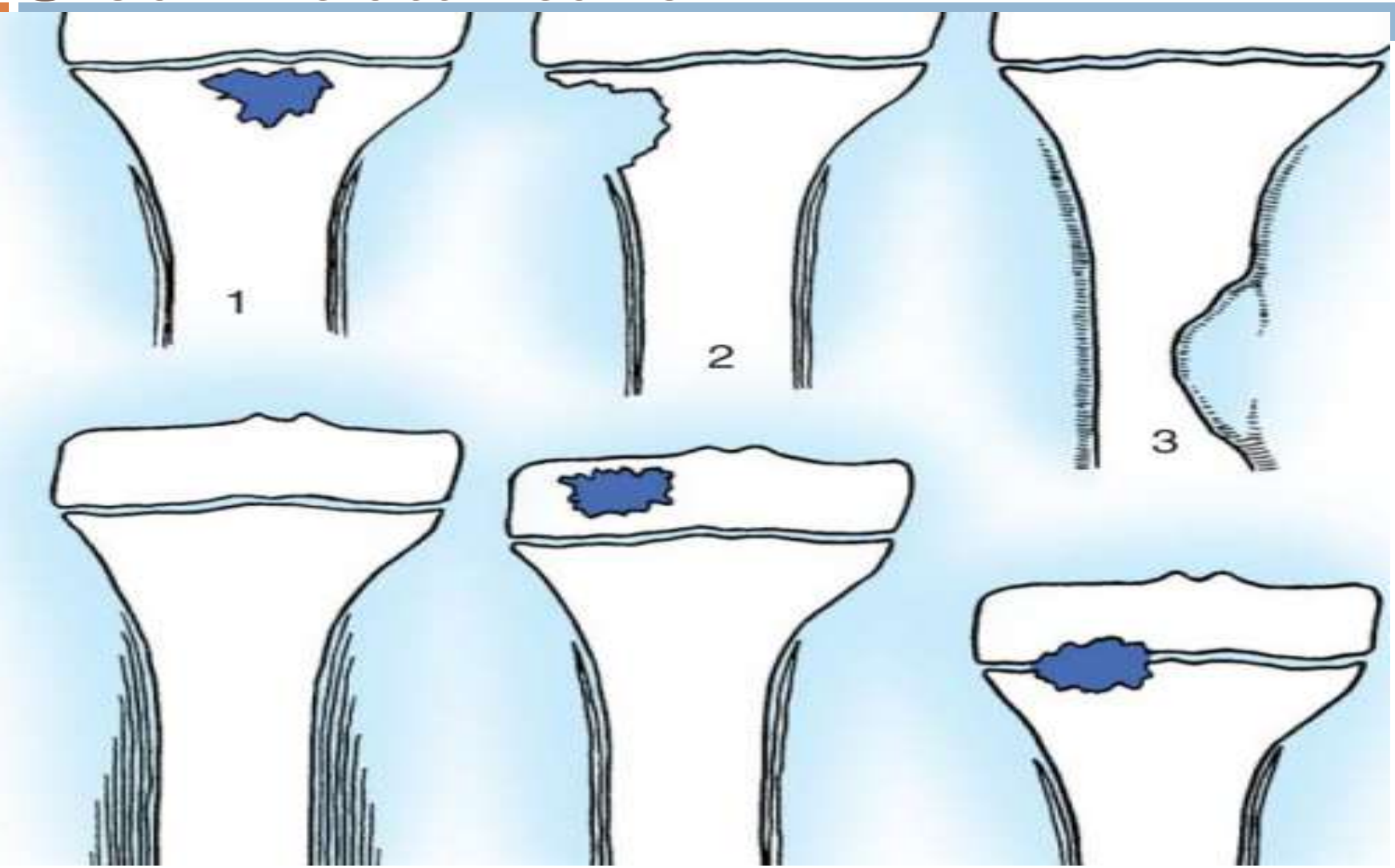
SUBACUTE HEMATOGENOUS OSTEOMYELITIS

BRODIE ABSCESS

- On plain radiographs appears as a lytic lesion with a rim of sclerotic bone
- S aureus is cultured in 50% of patients and in 20% the culture is negative
- The condition requires open biopsy with curetage to make the diagnosis
- The wound should be closed loosely over a drain

SUBACUTE HEMATOGENOUS OSTEOMYELITIS

Gledhill classification



SUBACUTE HEMATOGENOUS OSTEOMYELITIS

TREATMENT

- Biopsy and curettage followed by treatment with appropriate antibiotics for all lesions that seem to be aggressive

- For lesions that seem to be a simple abscess in the epiphysis or metaphysis biopsy is not recommended- IV antibiotics for 48 hrs followed by a 6 week course of oral antibiotics

CHRONIC OSTEOMYELITIS

ETIOLOGY

- Inadequate / Irregular / Delayed treatment in acute stage.
- Impaired host resistance → Acute OM → Chronic OM.
- High virulence of organism.

CLASSIFICATION

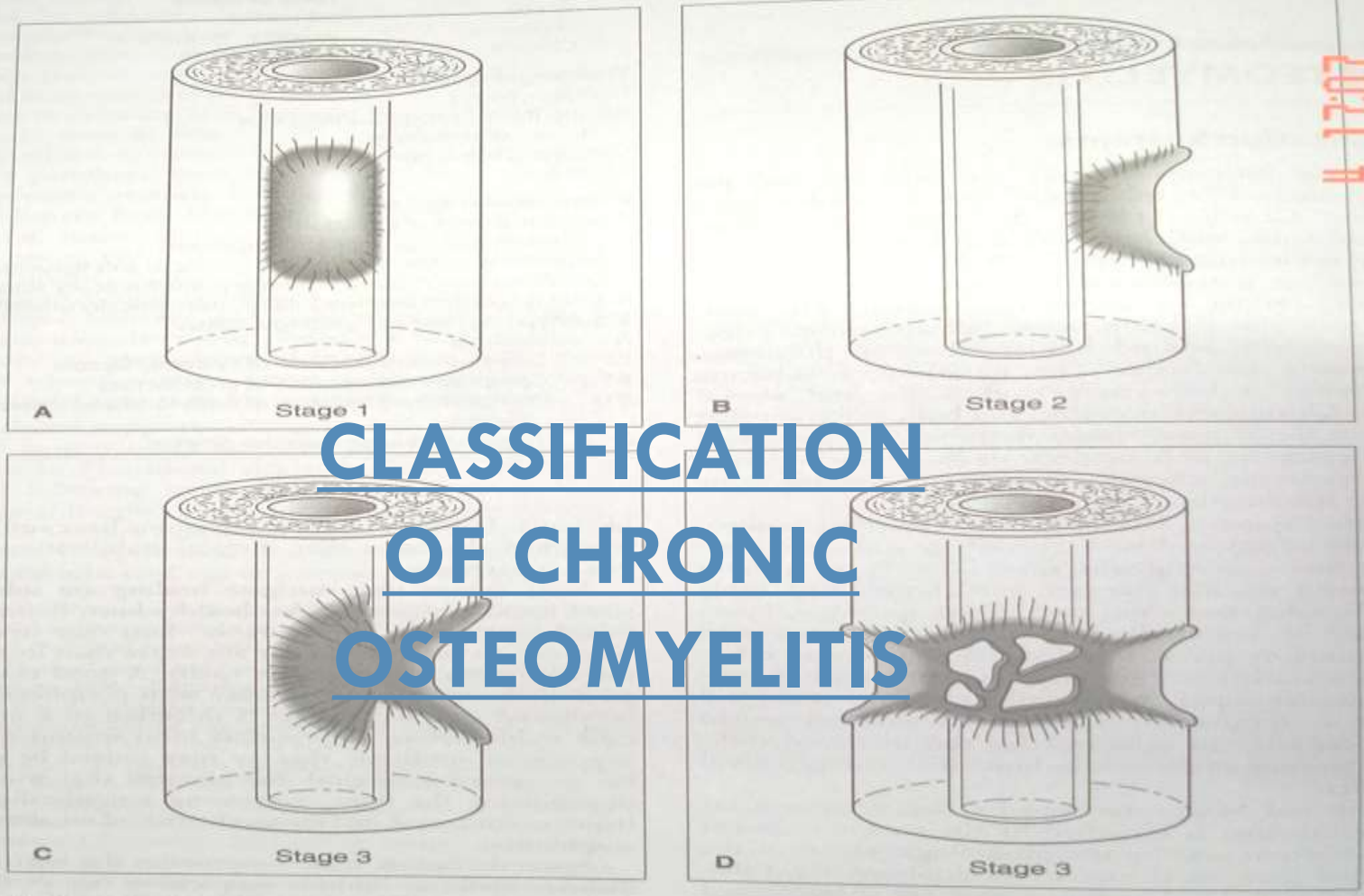
Based on –

- Route of infection
  - Exogenous
  - Endogenous
- Type of infection
  - Granulomatous
  - Pyogenic
- Mode of onset
  - Primary
  - Secondary

CIERNY AND MADER CLASSIFICATION

- Based on anatomic and Physiological criteria.
- Determines the clinical stages of infection.
- Helpful in treatment planning.

BOX 8-1. THE CIERNEY AND MADER STAGING SYSTEM.



CLASSIFICATION OF CHRONIC OSTEOMYELITIS

The Cierny and Mader staging system for osteomyelitis is classified by the anatomic extent of the infection and by the physiologic status of the host rather than by chronicity or etiology. The four stages are characterized by stage 2—superficial cortex only, stage 3—localized medullary and cortical, and stage 4—diffuse medullary and cortical.

PHYSIOLOGICAL CRITERIA -

Class A Hosts- Normal hosts.

Class B Hosts- Systemic compromise (Bs)

Local compromise (Bl)

Class C Hosts- Treatment potentially worse than disease.

ANATOMICAL CRITERIA -

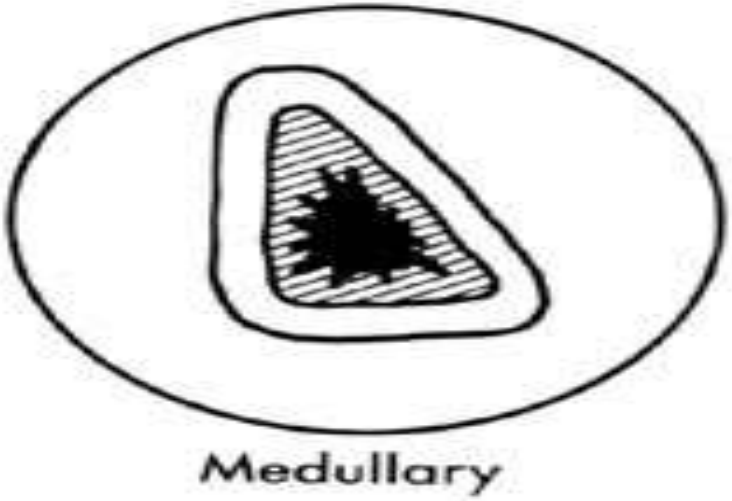
Type I- Endosteal/Medullary Osteomyelitis

Type II- Superficial Osteomyelitis (coverage defect)

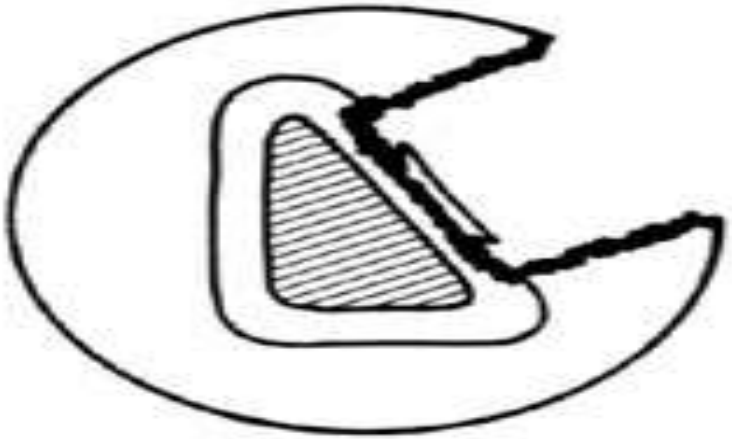
Type III- Localized, full thickness, unicortical but stable Osteomyelitis

Type IV- Diffuse mechanically unstable Osteomyelitis

ANATOMICAL CRITERIA-



Medullary



Superficial

Localized

Diffuse

BACTERIOLOGY

- Staphylococcus- Most common.
  ↓ incidence over past 10-15 yrs.
- Klebsiella- Vertebral body infections
  ↑ incidence in recent yrs.
- Pseudomonas- Prominent organism in I.V. drug users.
- Salmonella- Most common in Sickle cell disease.
  Tends to be diaphyseal rather than Metaphyseal.
  Multiple bones may be involved.

BACTERIOLOGY

- Haemophilus Influenzae- Common in patients 6 months-4 yrs.of age.
- Fungal- In chronically ill patients, receiving long term IV therapy/Parenteral nutrition.
- Mixed flora osteomyelitis- Seen in exogenous type of osteomyelitis.

PATHOGENESIS

Hematogenous infection → 1º involves marrow spaces + haversian canals + subperiosteal space

↓

Bone involved 2º & at metaphysis

cancellous bone + end arteries & capillary loops + near active physis – active phagocytosis = An excellent culture media

↓

Destroyed by proteolytic enzymes

Necrosed by obliteration of blood supply

Decalcified by inactivity and hyperemia

↑ ↓

The debris + exudate ↑ in unyielding bone, under pressure compresses blood vs. → further necrosis

PATHOGENESIS ctd.

Exudates follow path of least resistance

Haversian + Volkmans	Medullary canal	Physis
Canals → Subperiosteal space	Marrow space	Joint cavity RARE Common in hip

Accumulation of abscess, necrotic debris, exudates

Periosteal stripping Initiates periosteal Reaction	Destroys marrow elements & blood supply to bone	Synovium & cartilage Septic arthritis

Entire cortex surrounded by pus, joint filled by pus

Viability of bone & joint endangered

BONE AND JOINT MAY LIVE OR DIE

PATHOGENESIS ctd.

BONE AND JOINT MAY LIVE OR DIE

↓

Resorption by osteoclasts, granulation tissue + Reconstruction by osteoblasts

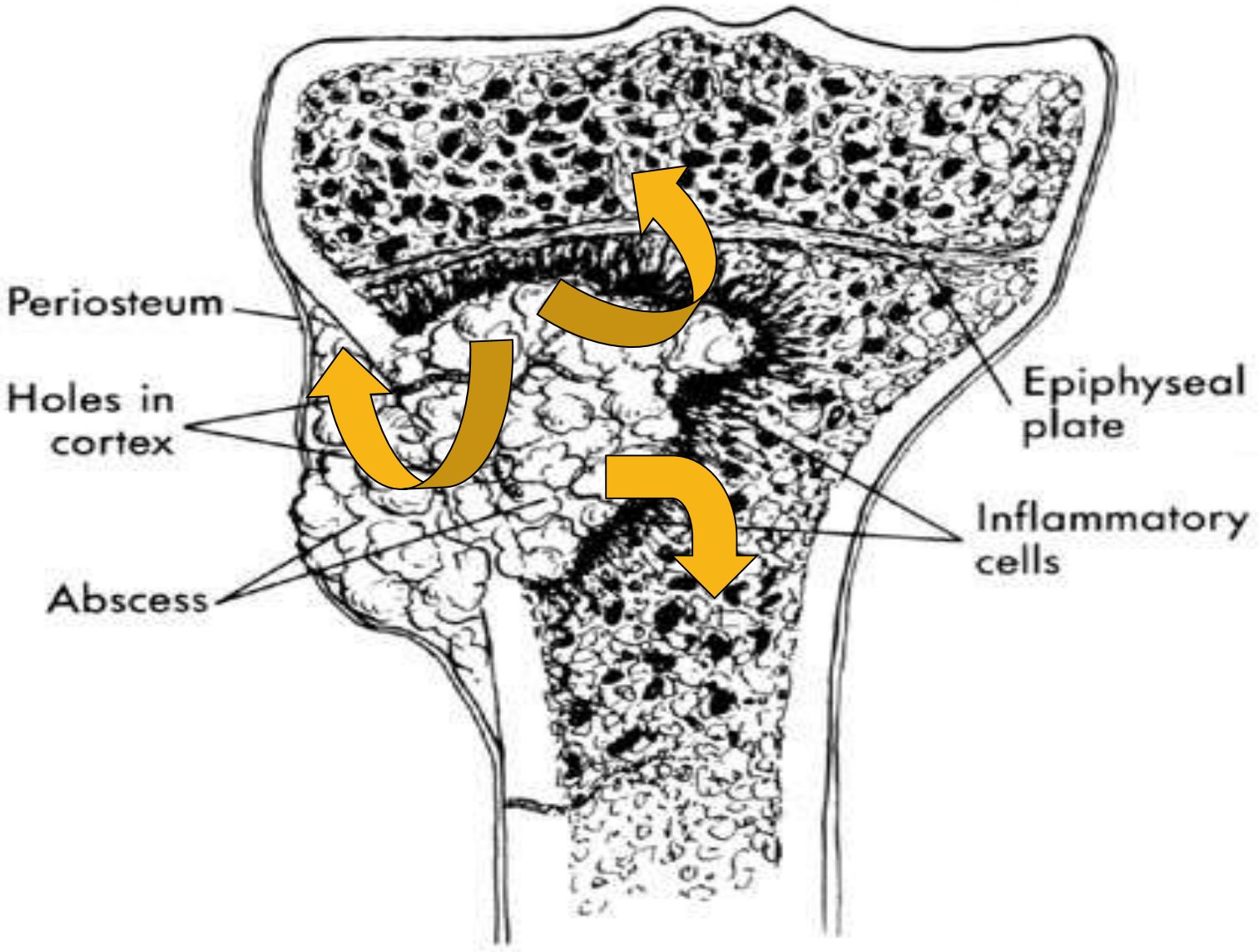
↓

Separated dead avascular bone inside live bone harbours bacteria inspite of antibiotics → SEQUESTRUM

↓	↓
Slowly absorbed / extruded	Walled off by dense fibro-osseous tissue
↓	↓
Till then exudate forms	**Brodie's abscess**
↓	↓
Persistent discharging sinus	Intermittent recrudescence of activity
↓	↓

CHRONIC OSTEOMYLETIS

Pathophysiology of hematogenous seeding



CLINICAL FEATURES

The findings of a chronic discharging sinus with unhealthy sprouting granulation tissue at its mouth in the presence of thickened, irregular bone is the hallmark of chronic osteomyelitis.

CLINICAL FEATURES

Symptomatology:

- Patients give a history of long duration of symptoms.
- Persistent pus discharge.
- History of passing of bone fragments (sequestrum)
- History of intermittent fever.

CLINICAL FEATURES

Clinical findings:

- Sinuses: Fixed to bone.
  With/without sprouting granulation tissue at its mouth
  Discharging pus/ bony spicules.
- Sequestrum: May be seen through the sinus.
- Skin: Surrounding, is dusky, adherent, scarred, pigmented & poorly vascularised.
- Muscles: Atrophic& wasted.

CLINICAL FEATURES

Clinical findings:

- Bone: Underlying bone is thickened& irregular.
- Joints: Restricted range of motion, due to scarring and adhesion of muscles.
- Lymph nodes: Regional lymph nodes are enlarged.
- Signs of inflammation present in acute exacerbation.

INVESTIGATIONS

- Haematology: Hb, ESR, CRP.
- X-Rays: To confirm the diagnosis

  To assess sequestrum (size, extent& separation).

  To assess involucrum (size, extent)
- Sinogram: To confirm the sinus.

  To mark the sinus-makes excision easy during surgery.
- Micro biology:Pus C/S.-for antibiotic protocol.
- Others: Bone scan, CT scan & MRI.

TREATMENT

- PREVENTION IS BETTER THAN CURE.

TREATMENT OPTIONS

- Antibiotics.
- PMMA Antibiotic beads.
- Closed suction & irrigation.
- Curettage & Sinus tract excision.
- Sequestrectomy.
- Saucerization.
- Soft tissue resection and transfer.
- Open bone grafting- Papineau technique.

TREATMENT OPTIONS

- Illizarov technique for bone transport.
- Electrical stimulation.
- Excision of the infected.
- Amputation

TREATMENT-

A. Improvement of general condition-

Hydration & electrolyte replacement.

Nutrition and protein replacement.

TREATMENT-

- B. Immobilization-

Very important, protects the part.

Prevents and decreases the spread of infection.

Prevents a pathological fracture.

A window can be made for dressings.

TREATMENT-

- C. Antibiotics& Anti-inflammatory drugs-

In the acute stage- Broad spectrum antibiotics → followed by oral antibiotics depending upon culture sensitivity report.

Weekly ESR - assessment of response.

No role in presence of sequestrum.

TREATMENT-

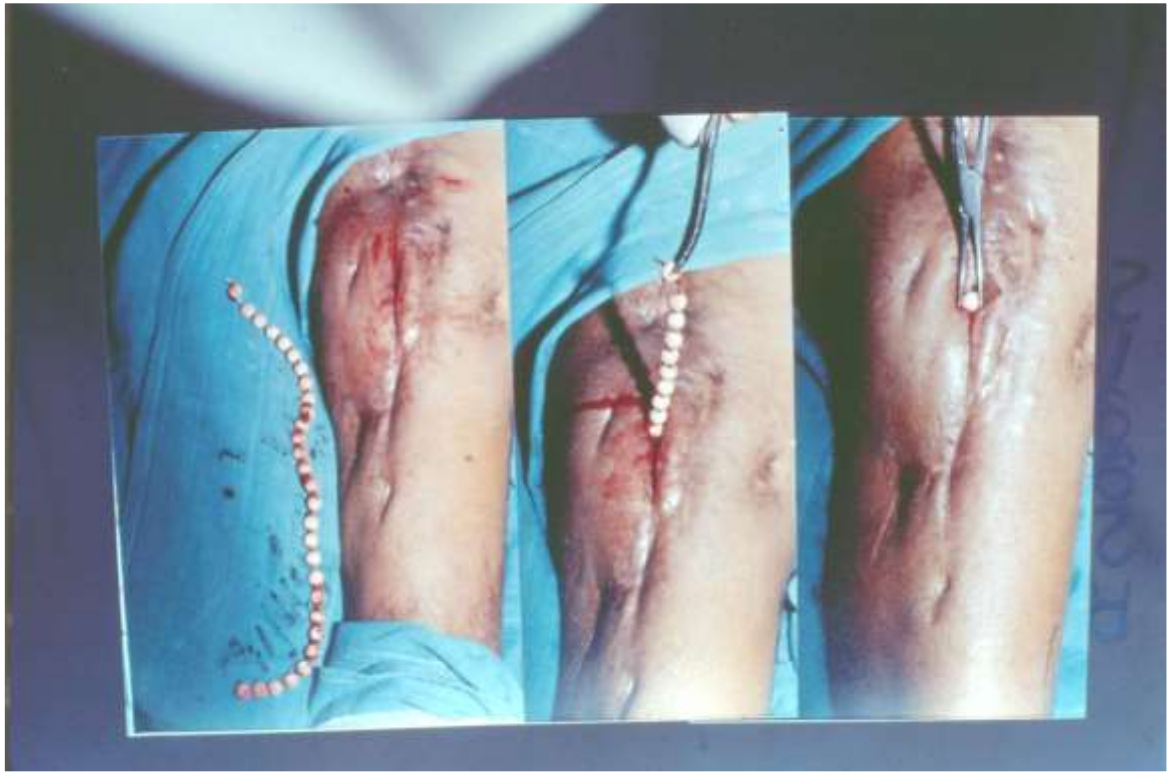
- D. Polymethyl-methacrylate Antibiotic bead chains.

Rationale - deliver antibiotics locally in high concentrations (X200 times).

Commercially available as string of 30 beads.

Each bead contains 7.5 mg of Gentamycin Sulphate + 4.5 mg of free Gentamycin.

PMMA Beads



TREATMENT-

- E. Closed Suction & Irrigation

High volume suction irrigation systems may be used over 3-21 days for resistant focal infections by topical instillation of a solution – Alevaire which contains –

Detergent (prevents penicillinase formation by bacteria)

Mucolytic (breaks up pus, mucus and necrotic tissue)

Bacteriostatic

Each 1 L irrigation solution contains 200ml Alevaire

800 ml saline

5 MU penicillin

2 gms antibiotic as per C/S report

Delivery rate is 80 ml/hr or 2L/24 hrs. This is continued till 3 consequent cultures from the outflow drain are sterile on culture.

TREATMENT-

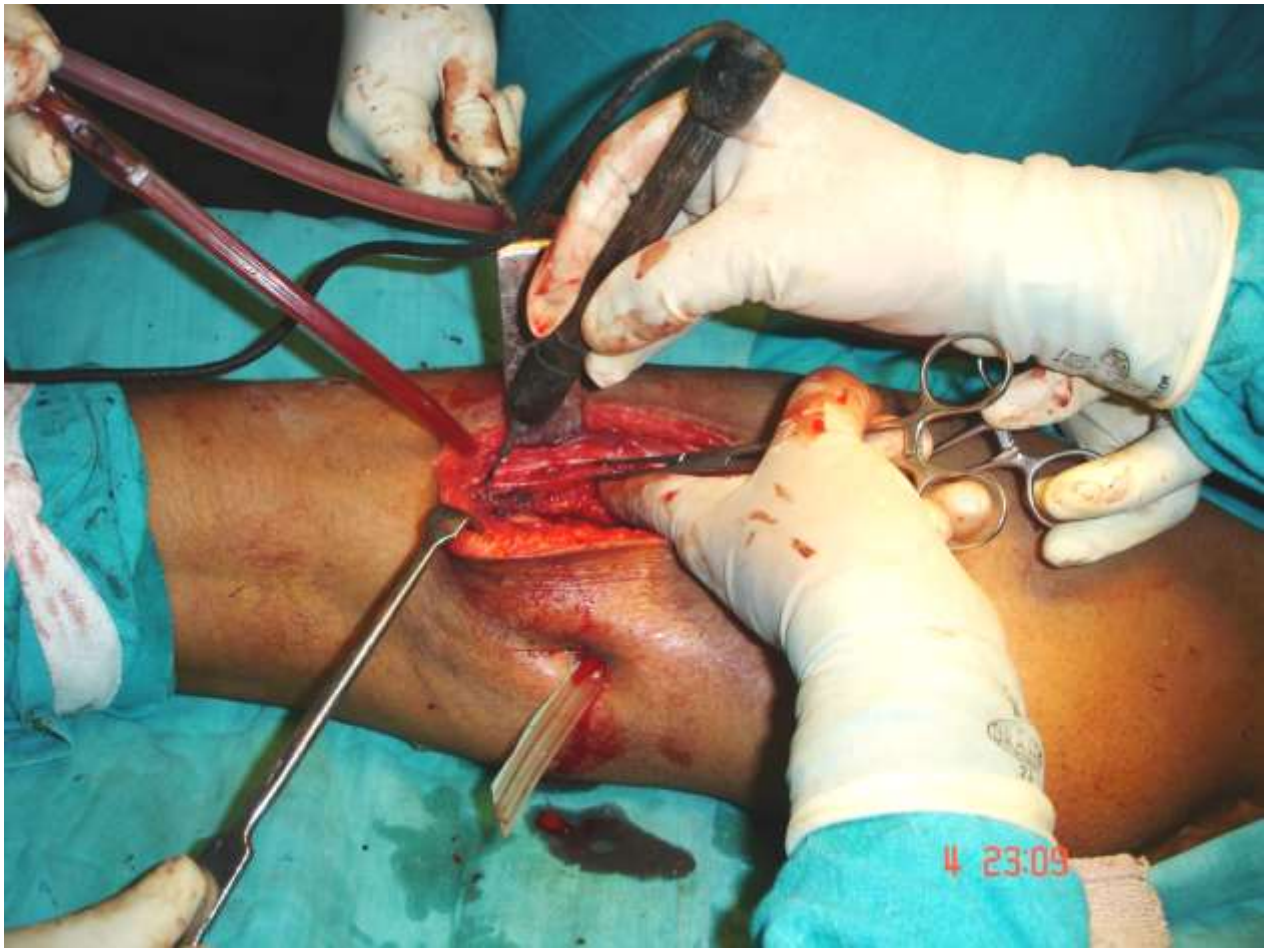
- F. Sequestrectomy & Curettage –

Goal – eradication of infection +

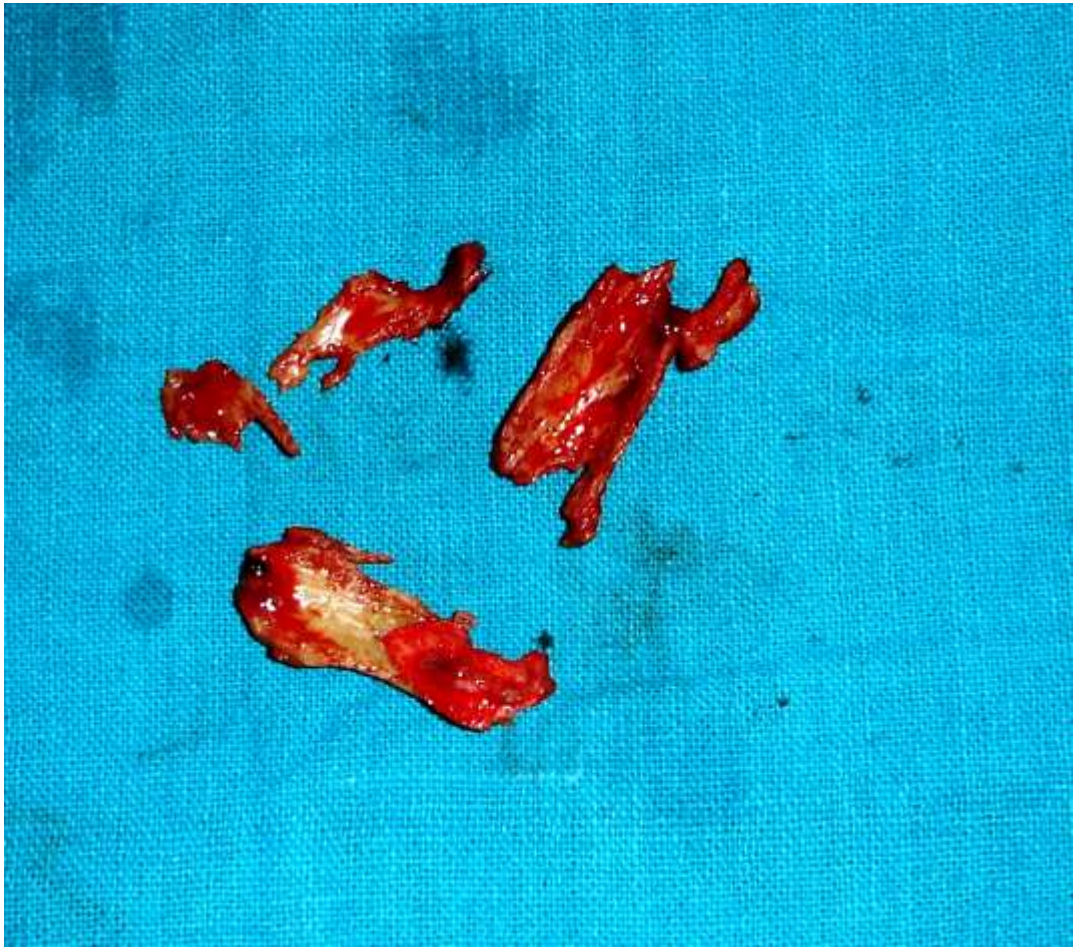
achievement of viable vascular environment.

	Indications	Contraindications
Sequestrum	**- partially extruded**	**large, not separated**
	separated, active	**quiescent**
	not getting incorporated	**getting incorporated**
Involucrum	**Sufficient**	**Insufficient**
Discharge	**Persistent**	**Absent**

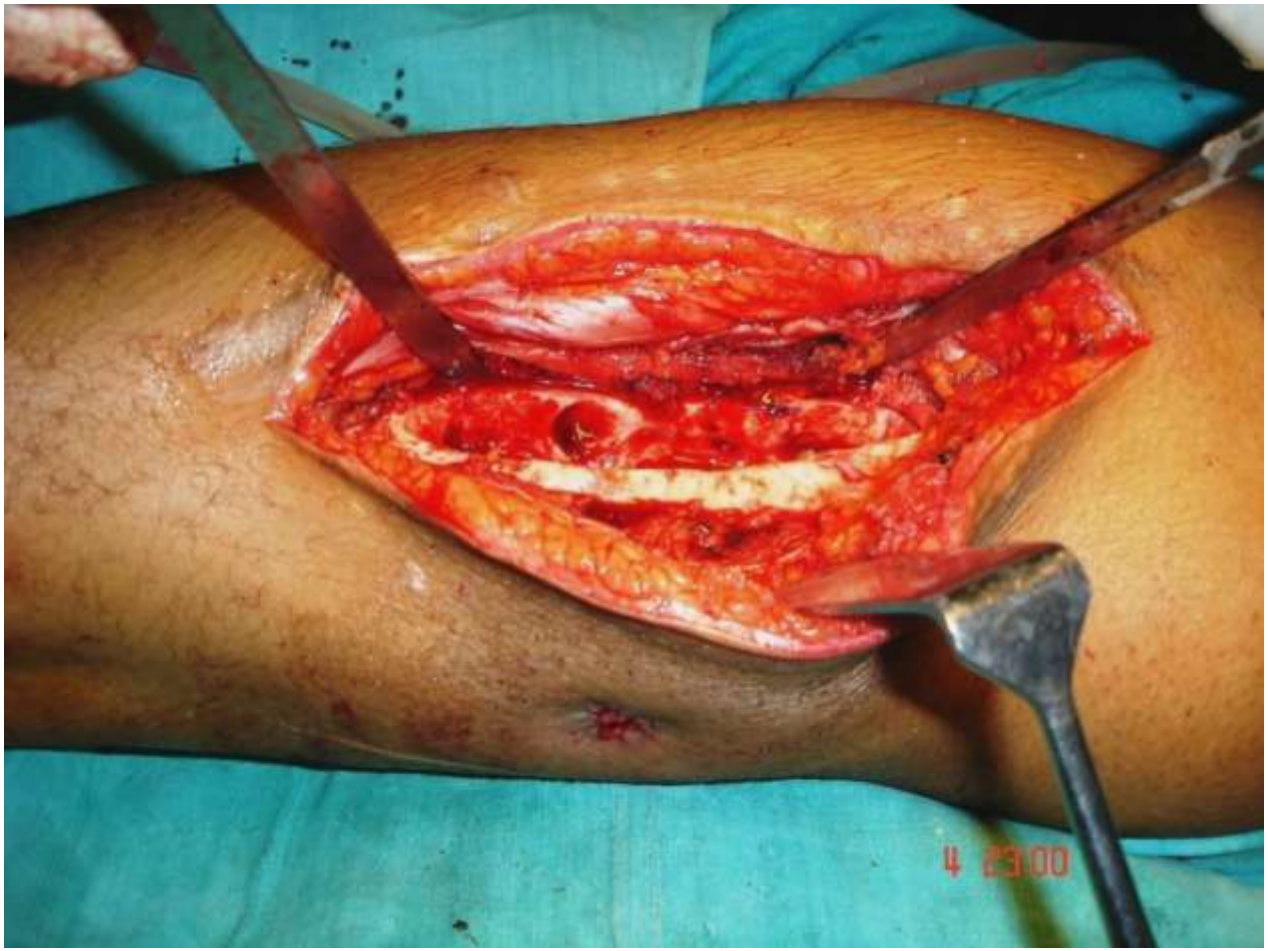
CURETTAGE !!



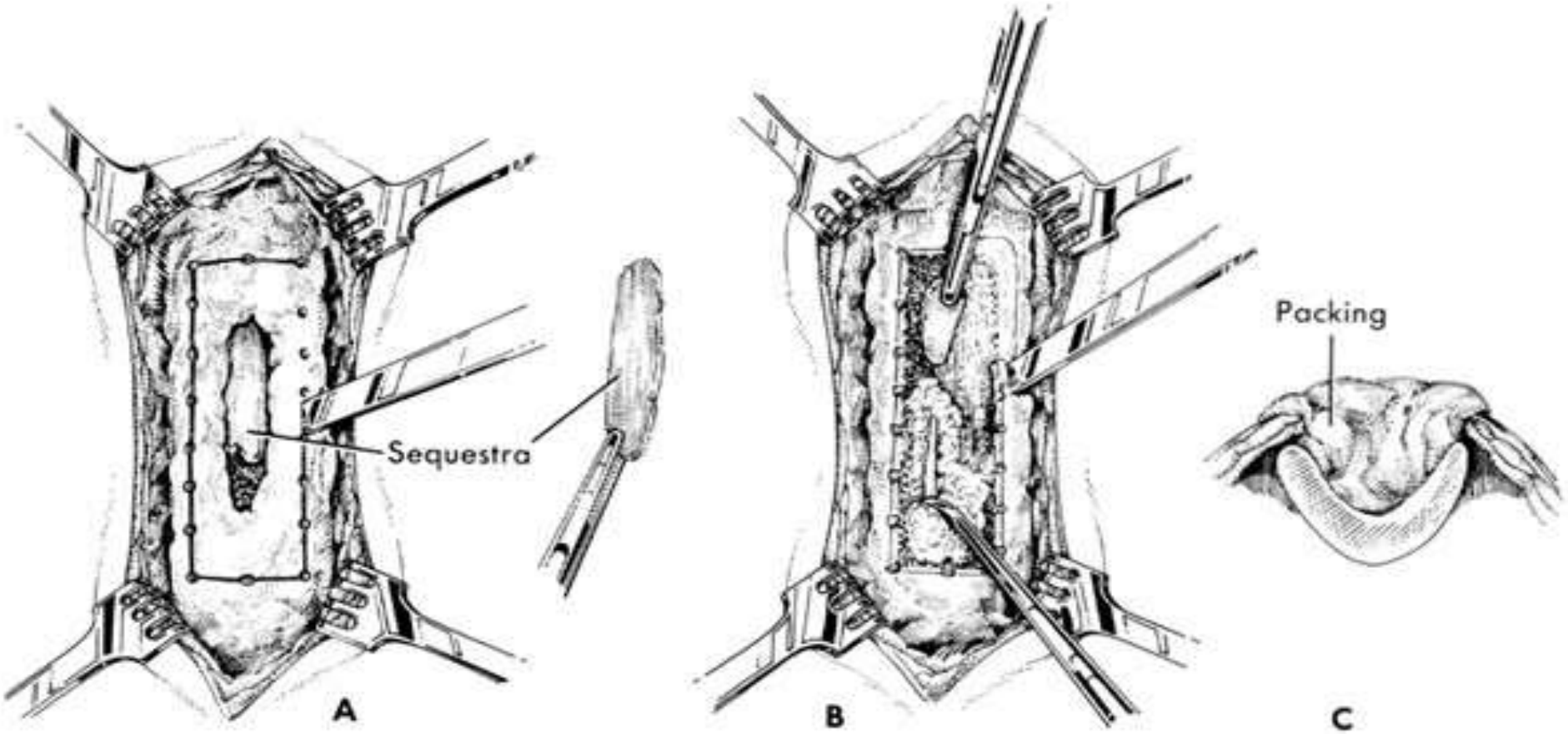
SEQUESTRECTOMY !!



SAUCERIZATION!!



Sequestrectomy & Curettage ctd.



A. Bone exposed, sequestrum removed.
B. All infected matter removed.
C. Wound is either packed open or closed loosely over drains.

TREATMENT-

- G. Filling of Bony & Soft tissue defects

Done to ↓ chance of continued infection & loss of function.

Techniques

1. Bone grafting with 1° / 2° closure.

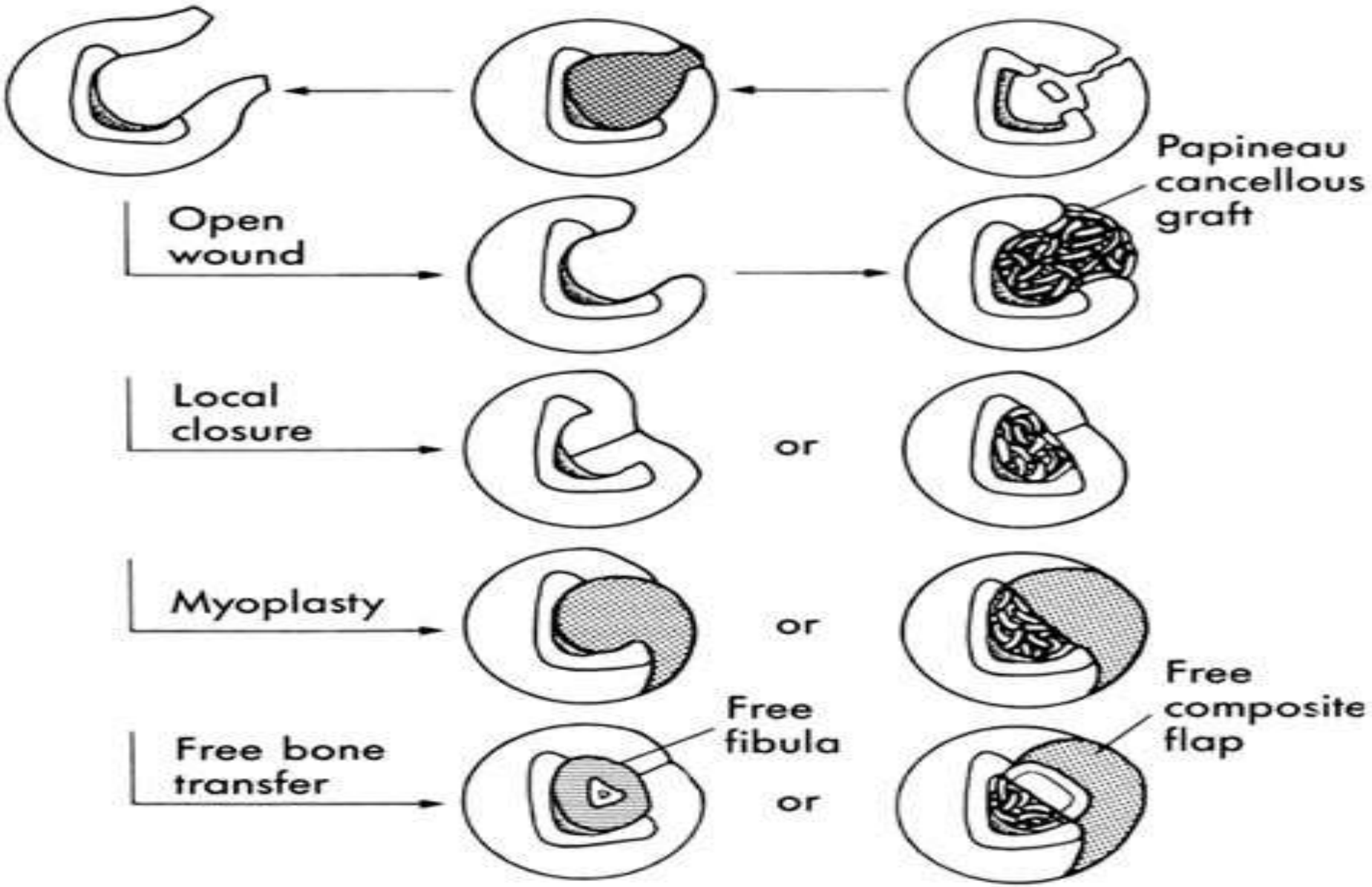
2. PMMA beads as temporary / permanent filler

3. Skin grafting & local muscle flaps with / without bone grafting ; microvascular muscle transfer.

4. Myocutaneous, osseous & osteo-cutaneous flaps.

5. Bone transport (Ilizarov technique)

Immediate, Biological management of dead space with living tissue / cancellous bone grafts.



TREATMENT-

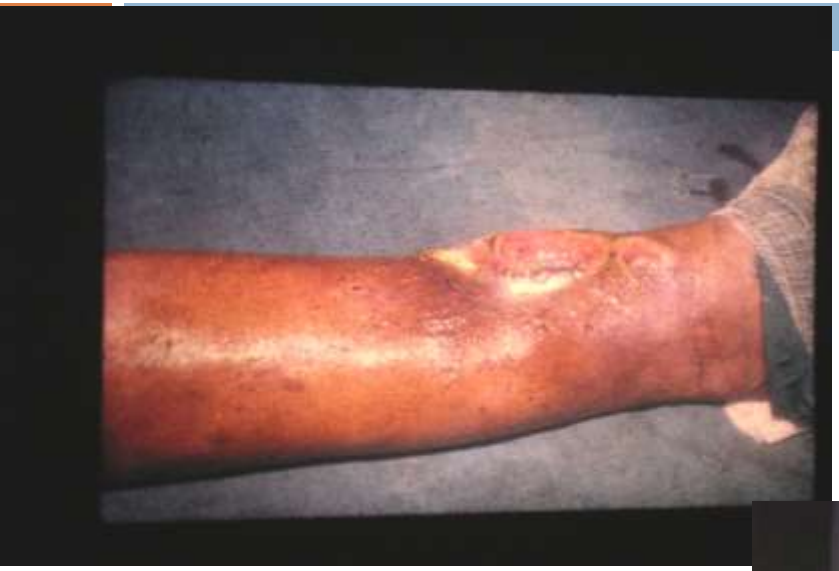
H. Open bone grafting.

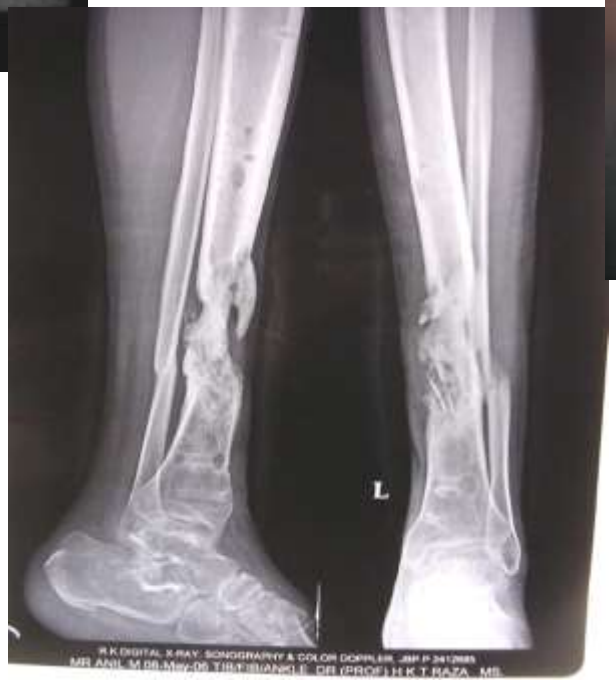
- Based on Papineau's principles and technique.

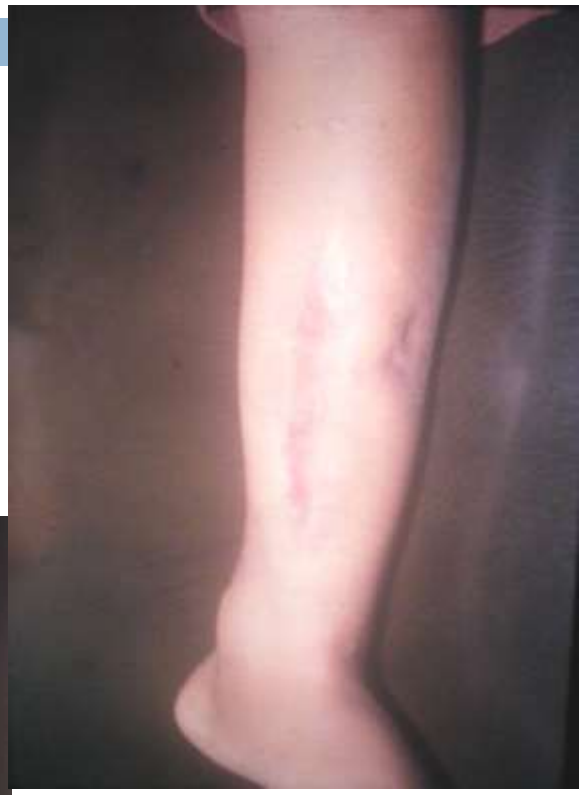
Principles

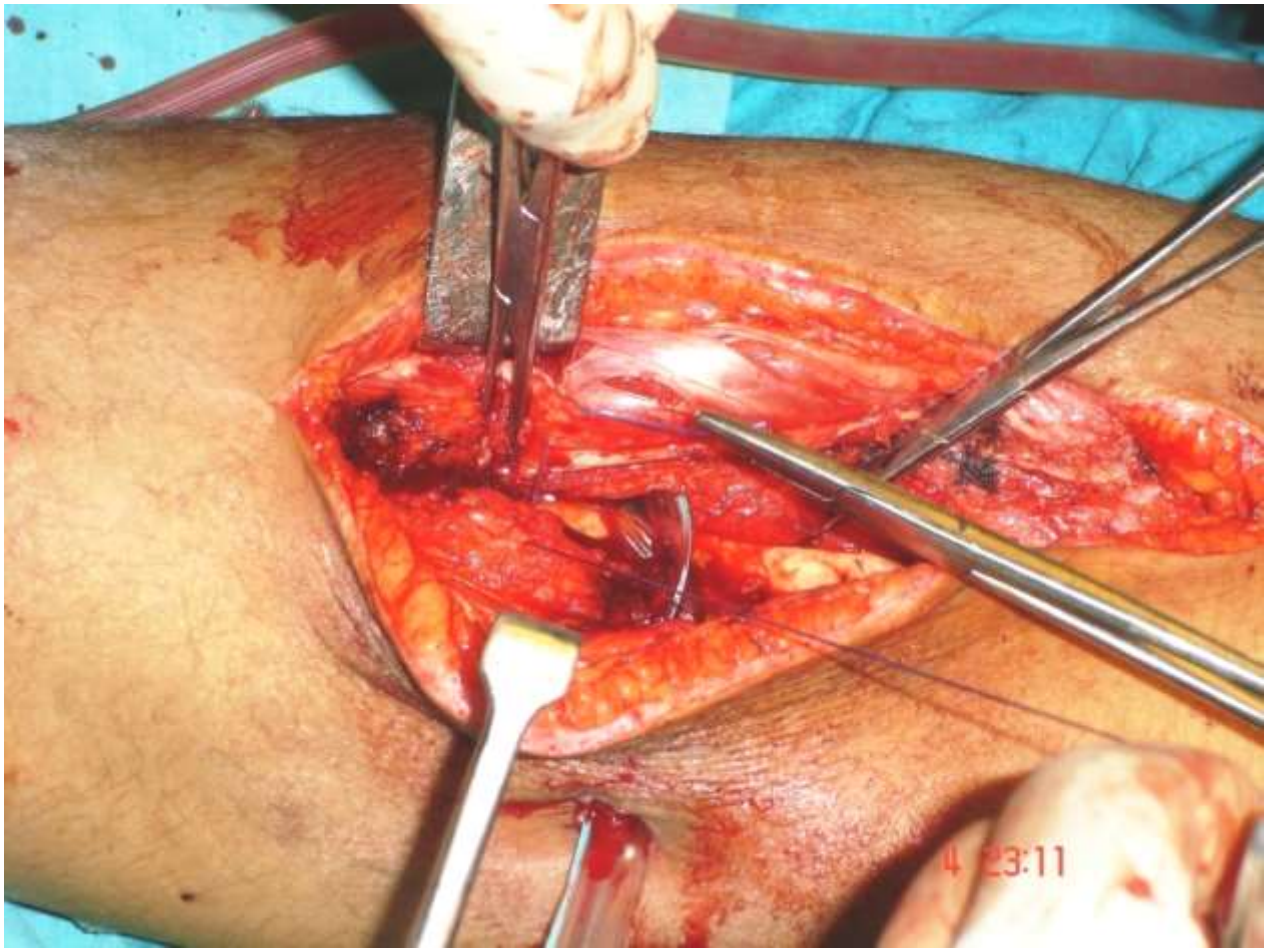
1. **Granulation tissue markedly resists infection.**
2. **Autogenous grafts are rapidly revascularized & resistant to infection.**
3. **Infected area is completely excised.**
4. **Adequate drainage is provided.**
5. **Adequate immobilization is given.**
6. **Appropriate antibiotics for long duration.**

Papineau Technique









TREATMENT-

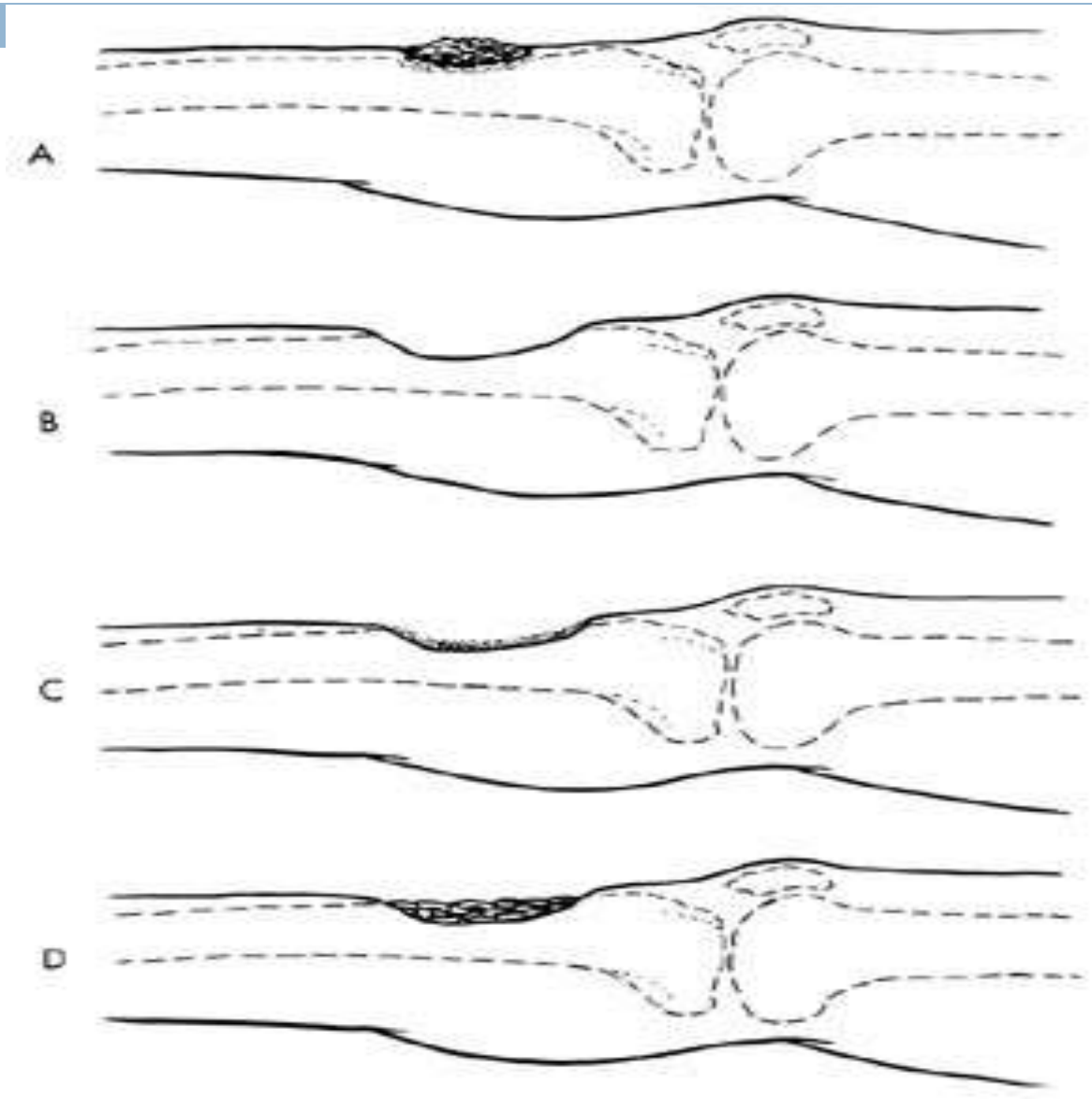
H. Open bone grafting Papineau's technique :

Chronic OM →

Debridement & development of granulation tissue →

Open Bone graft →

Blood clot in place →



TREATMENT-

- I. Open bone grafting.

To fill dead space after debridement.

Ranges from local muscle flap on vascular pedicle to microvascular free tissue transfer.

Rationale

Improves host defence mechanisms ,

Improves antibiotic delivery &

Bone and soft tissue healing.

TREATMENT-

- J. Ilizarov technique.

Useful in chronic OM & infected non-unions.

Allows radical resection or en-bloc resection of infected bone following which bone transport is done until union is achieved.

Disadvantages

Time needed to achieve solid union.

High incidence of associated complications.

TREATMENT-

- K. Segmental bone transport.

Done where large amount of bone needs to be resected

TYPES-

External –(bone loss <5cm)

Internal –(>5-10 cm)

Combind –(>10cm)

TREATMENT-

- L. Resection / Excision of bones

Not advisable, however with new techniques of

bone and soft tissue transport it may be undertaken.

Resection of expendable bones may be done.

TREATMENT-

- M. Amputation

Infrequently performed for OM.

Indications

Associated with malignant changes.

Arterial insufficiency.

Major nerve paralysis.

Severe contractures.

VARIANTS OF CHRONIC OM

A. Brodie's abscess

Localised form of chronic OM

Usually caused by organisms of low virulence

Often seen in long bones of lower limbs in young adults.

Varied appearance on X-rays.

Biopsy gives confirmatory diagnosis.

Treatment : Curettage + Antibiotics

- **B. Sclerosing OM of Garre'(Idiopathic cortical sclerosis)**

  Cause unknown, possibly anaerobic bacteria.

  Affects children and young adults.

  Symptomatology over long duration.

  X-rays :- Thickening of bone with sclerosis.

  Biopsy :- Chronic low grade non-specific OM.

  Cultures negative.

Treament

No treatment has been predictively helpful but fenestration of sclerotic bone and antibiotics are advisable.

D/ds

Osteoid ostema

Paget's disease

COMPLICATIONS OF CHRONIC OM

- Acute exacerbation
- Septicaemia
- Septic arthritis
- Joint stiffness
- Pathological fracture
- Deformity
- Growth disturbance
- Squamous cell Ca.
- Amyloidosis
- Amputation

THANK YOU