Tuberculosis

- *Mycobacterium tuberculosis* is most common
- *Mycobacterium bovis*, rare, spread to humans by milk
- Dormant tuberculosis of the skin can also be reactivated by systemic corticosteroids, immunosuppressants and new anti-TNF biological agents.
Inoculation tuberculosis

- Inoculation into skin causes a wart-like lesion at the site
lupus vulgaris

- Systemic spread to the skin (lupus vulgaris) from an underlying infected lymph node, or from a pulmonary lesion.
- reddish-brown scaly plaque slowly enlarges, and can damage deeper tissues such as cartilage, leading to ugly mutilation which may cause scarring and contractures.
- Lesions occur most often around the head and neck.
- Diascopy shows up the characteristic brownish ‘apple jelly’ nodules.

The clinical diagnosis should be confirmed by biopsy.
**Scrofuloderma**

- mixture of lesions (irregular puckered scars, fistulae and abscesses) of skin overlying a tuberculous lymph node or joint, most commonly seen in the neck.

**Tuberculides**

- Papulonecrotic tuberculides – By finding mycobacterial DNA by polymerase chain reaction (PCR)- are recurring crops of firm dusky papules, which may ulcerate, favouring the points of the knees and elbows.

**Erythema induratum (Bazin’s disease)**

- deep purplish ulcerating nodules occur on the backs of the lower legs, usually in women with a poor ‘chilblain’ type of circulation.
- Erythema nodosum may also be the result of tuberculosis elsewhere.
Fig. 16.8 A plaque with the brownish tinge characteristic of lupus vulgaris. Diascopy was positive.
Investigations

Biopsy for:
- microscopy (tuberculoid granulomas)
- bacteriological culture
- detection of mycobacterial DNA by PCR

Mantoux test
Chest X-ray
Treatment

• The treatment of all types of cutaneous tuberculosis should be with a full course of a standard multidrug antituberculosis regimen.

Prevention

• Bacillus Calmette–Guérin (BCG) vaccination of schoolchildren, immunization of cattle and pasteurization of milk.
Leprosy

Cause
- Caused by *Mycobacterium leprae*
- main route of infection is through nasal droplets from cases of lepromatous leprosy, rarely from eating infected armadillos.
Presentation

depends upon the immune response of the patient

• Those with a high resistance develop a paucibacillary tuberculoid type
• Those with low resistance a multibacillary lepromatous type
• between the extremes lies a spectrum of reactions classified as ‘borderline’
• Nerve thickening is earlier and more marked in the tuberculoid than lepromatous type.
The spectrum of leprosy: tuberculoid to lepromatous

- **Tuberculoid**: High resistance (+ve lepromin test), Organisms hard to find, Non-infectious, Localized lesions
- **Borderline (BB)**: Intermediate resistance, Some organisms, Slightly infectious, Scattered lesions
- **Borderline tuberculoid**:
- **Borderline lepromatous**: Low resistance (−ve lepromin test), Many organisms, Infectious, Generalized lesions
- **Lepromatous**:
**Tuberculoid**

- Involvement of Skin and nerves only

**Lepromatous**

- Many tissues

**Lesions**

- One or two only Commonly on face
  - Sharply marginated hypopigmented macules
  - Slightly raised purplish rim
  - Hairless and anhidrotic patches

- Innumerable Widespread
  - Macules, papules, nodules, plaques
  - Thickened on face leonine facies

**Involvement of nerves**

- Thickened in vicinity of lesion
  - Hypoesthesia of lesion
  - Absent sweating of lesion

- Most peripheral nerves thickened
  - Lesions not hypoesthetic but glove and stocking anaesthesia, trophic ulcers of periphery, muscle paralysis
Fig. 16.10 Tuberculoid leprosy: subtle depigmentation with a palpable erythematous rim at the upper edge.
Fig. 16.11 The ‘leonine’ facies of lepromatous leprosy.
Differential diagnosis

- Leprosy is a great imitator
  
  **Tuberculoid leprosy**
  - vitiligo
  - pityriasis versicolor
  - pityriasis alba
  - post-inflammatory depigmentation of any cause

  **Borderline leprosy**
  - Sarcoidosis
  - Granuloma annulare
  - necrobiosis lipoidica.

  **Lepromatous leprosy**
  - Widespread leishmaniasis
  - neurofibromatosis
  - mycosis fungoides
  - multiple sebaceous cysts
Investigations

- **Biopsy** of skin or sensory nerve.
- **Skin or nasal smears**, with Ziehl–Neelsen or Fité stains, will show up the large number of organisms seen in the lepromatous type.
- **Lepromin test**, of no use in the diagnosis of leprosy but, once the diagnosis has been made, it will help to decide which type of disease is present (positive in tuberculoid type).
Treatment

• The emergence of resistant strains of *M. leprae* means that it is no longer wise to treat leprosy with dapsone alone.
• Dapsone in combination with rifampicin, and also with clofazimine for lepromatous leprosy.
• Rifampin is rapidly bactericidal, making patients non-infectious and able to return to the community.
• Tuberculoid forms are usually treated for 6 months; multibacillary leprosy needs treatment for at least 1 year.
• Special care is needed with the two types of lepra reaction that can occur during treatment.

**Type 1 (reversal) reactions**
• seen mainly in BT disease
• Lesions become red and angry, and pain and paralysis follow neural inflammation.
• Treatment is with salicylates, chloroquine, nonsteroidal and steroidal anti-inflammatory drugs.
• Nerve palsies need prompt treatment with corticosteroids to preserve function.

**Type 2 reactions**
• common in lepromatous leprosy
• include erythema nodosum, nerve palsies, lymphadenopathy, arthritis, iridocyclitis, epididymo-orchitis and proteinuria.
• They are treated with the drugs used for type 1 reactions, and also with thalidomide.
• The household contacts of lepromatous patients are at risk of developing leprosy and should be followed up.
• Child contacts may benefit from prophylactic therapy and BCG inoculation.
The End