NSII
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Group B Streptococcus (galactiae)
Mycobacterium Leprae

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Group B *streptococcus (Streptococcus Agalactiae*)

Characteristics:

- Facultative anaerobic encapsulated gram-positive coccus
- Produces a narrow zone of β-hemolysis on blood agar
- Most strains are bacitracin resistant / hydrolyze hippurate
- Based on the specificity of its capsular polysaccharide; 10 Serotypes: type 3 is commonly associated with neonatal sepsis
- Normal flora of throat, colon, urethra and, and in 10-40% of women vagina.

Virulent factors

- S. agalactiae produces at least 12 virulence factors that include
  - Fbs A fibrinogen-binding proteins encoded by GBS; helps in colonization,
  - PI-1 pili that promotes the invasion of human endothelial cells,
  - a polysaccharide capsule that prevents the activation of the alternative complement pathway and inhibits phagocytosis,
  - and the toxin CAMP factor, which forms pores in host cell membranes and binds to IgG and IgM antibodies.

Group B *streptococcus (Streptococcus Agalactiae*)

Clinically:

1. Non pregnancy associated:

    Pneumonia, UTIs, meningitis, infective endocarditis and soft tissue infection (multisystem involvement)

Sepsis and septic shock

usually in unhealthy people e.g chronic illnesses,

immunocompromised and elderly

Group B *streptococcus (Streptococcus Agalactiae*)

Clinically:

2. Pregnancy associated:

MOT (mode of transmission):

1. Vertical (Ascending from vagina to placenta)
2. During delivery and birth canal passage of the baby (intrapartum)

Clinically:

1. Chorioamnionitis
2. Abortion
3. Neonatal sepsis: early and late sepsis

Group B *streptococcus (Streptococcus Agalactiae*)

Clinical picture / neonatal sepsis

Early sepsis:

- Risk factors
  1. Group B streptococcus genitally colonized mother
  2. prematurity
  3. Prolonged rupture of membrane (PROM)
  4. Prolonged labour
  5. Maternal Chorioamnionitis, leukocytosis and fever
  6. Previous delivery with GBS disease
- **Source of bacteria:** Ascending or during delivery
- Occurs in the first week of life, though most present within the first 48 hrs.
- Nonspecific signs (lethargy, cyanosis, apnoea and respiratory distress).
- Meningitis, pneumonia and septic shock are common.

Group B *streptococcus (Streptococcus Agalactiae*)

Clinical picture / neonatal sepsis

Late sepsis:

- Absent history of complicated delivery
- Usually hospital acquired (medical staff, visitors and mother)
- 1 week – 3months?

Clinically:

- Purulent meningitis is more common than in early
- Babies may have long-term problems, such as deafness and developmental disabilities, due to having GBS disease
- Other systems may also be involved leading to:
  1. Pneumonia, arthritis, endocarditis, osteomylitis, sinusitis and
  2. septic shock and multiorgans failure

Group B *streptococcus (Streptococcus Agalactiae*)

Neonatal sepsis / Diagnosis

Diagnosis:

1. Clinically
2. Septic work up:
   - Full Blood Count (**FBC**), liver function test (LFT), C-reactive protein and CXR
   - Blood, CSF, appropriate swabs: Culture and stain (CSF protein, cell count and glucose)

Diagnosis

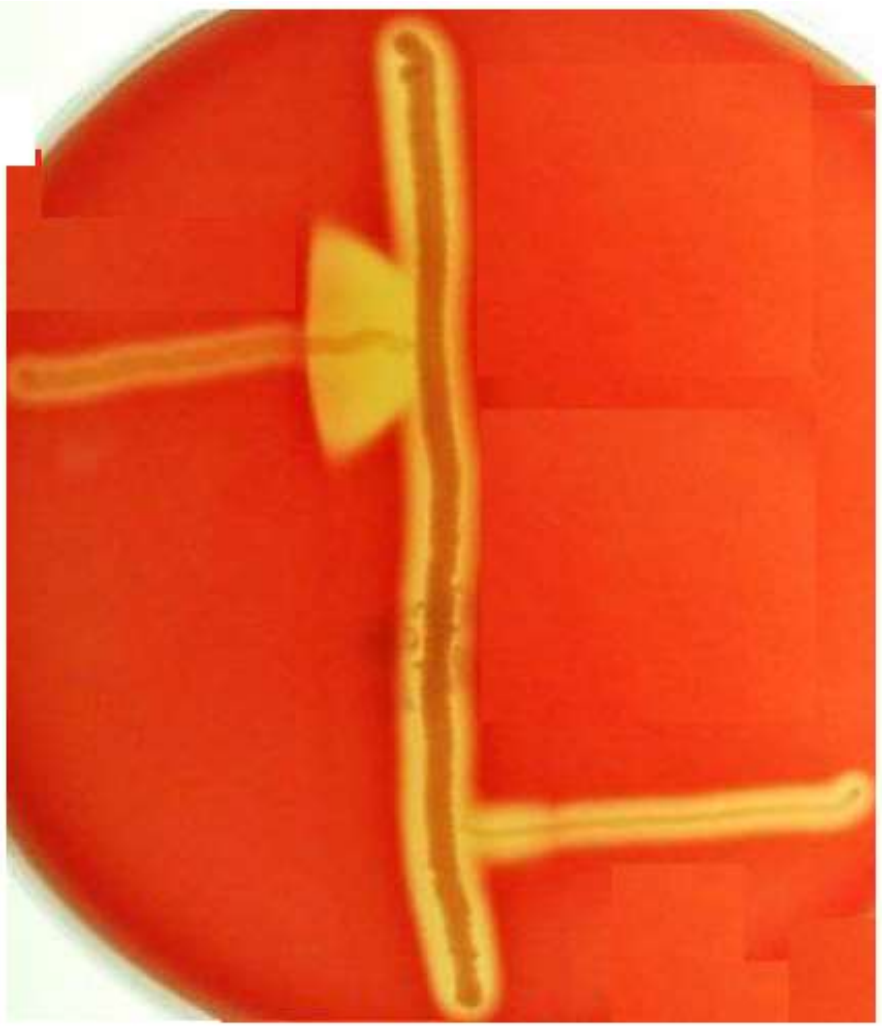
- is best confirmed by analysis of CSF obtained by a lumbar puncture. Abnormal levels of polymorphonuclear neutrophils (PMNs) (> 10 PMNs/mm3), glucose (< 45 mg/dL), and protein (> 45 mg/dL) in the CSF are suggestive of bacterial meningitis

CAMP (Christie Atkins Munch-Petersen) Test

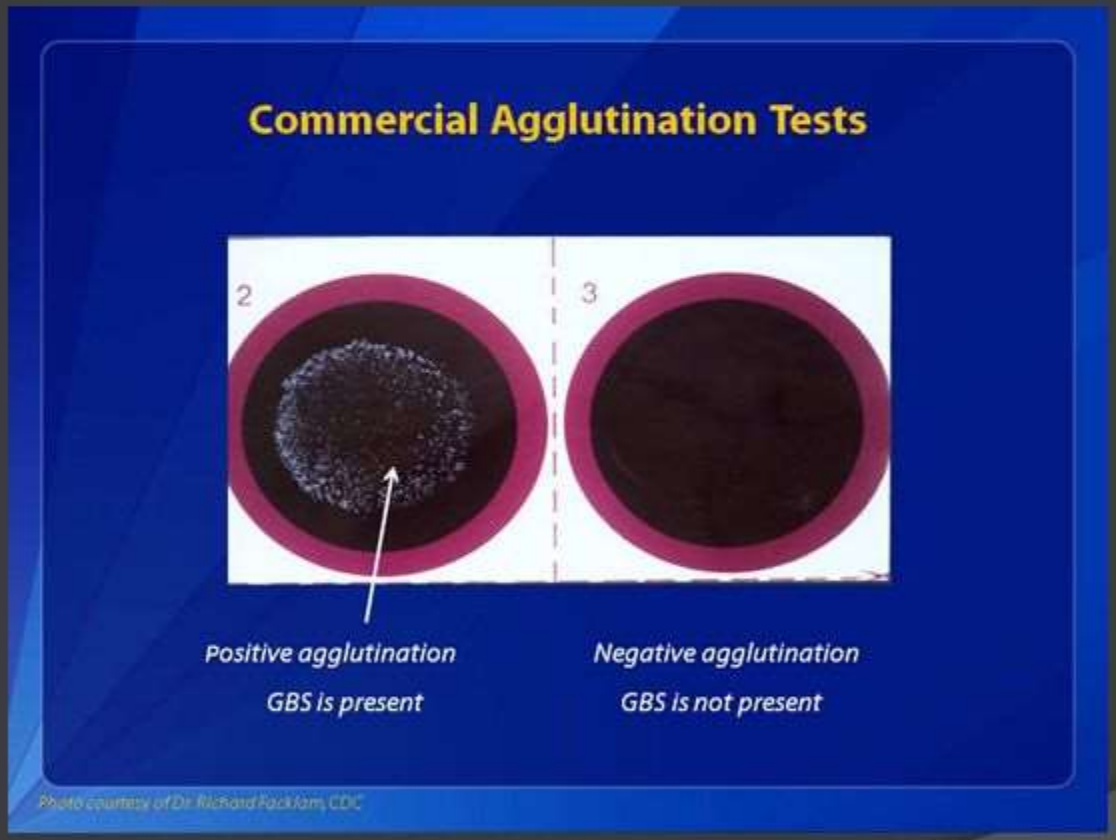
- to detect bacterial diffusible extracellular protein (CAMP factor)
- **Results**:

1- **Positive** : arrowhead (wedge)-shaped zone of enhanced hemolysis at junction of 2 organisms (***Strep agalactiae*** & staph).

2- **Negative** : NO enhancement of hemolysis (***Strep pyogenes***).



Detection–Latex agglutination



http://www.cdc.gov/groupbstrep/lab/lab-photos.html

Hippurate Hydrolysis Test

- **Hippurate hydrolysis test is used to detect the ability of bacteria to hydrolyse substrate hippurate into glycine and benzoic acid by action of hippuricase enzyme present in bacteria**
- **Results**:

1- **Positive**: deep purple colour.

2- **Negative**: NO colour change or slight yellow colour.

- **Important hippurate-positive bacteria**:

1- Streptococcus agalactiae.

Group B *streptococcus (Streptococcus Agalactiae*)

Neonatal sepsis / treatment

Treatment:

- If the mother had a risky delivery then give IV intrapartum antibiotics (Intrapartum antibiotic prophylaxis was defined as adequate when the initial dose was given at least four hours prior to birth).
- Continue antibiotics for 12-24 hrs with the baby and stop if asymptomatic and cultures negative
- If the baby is symptomatic / cultures positive, then 2-3 weeks of IV antibiotics and stop pending improvement
- Ampicillin + Gentamicin > IV
- Cefotaxime, Ceftriaxone or clindamycin are alternatives.

M. leprae

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What is leprosy(Hansen's disease)

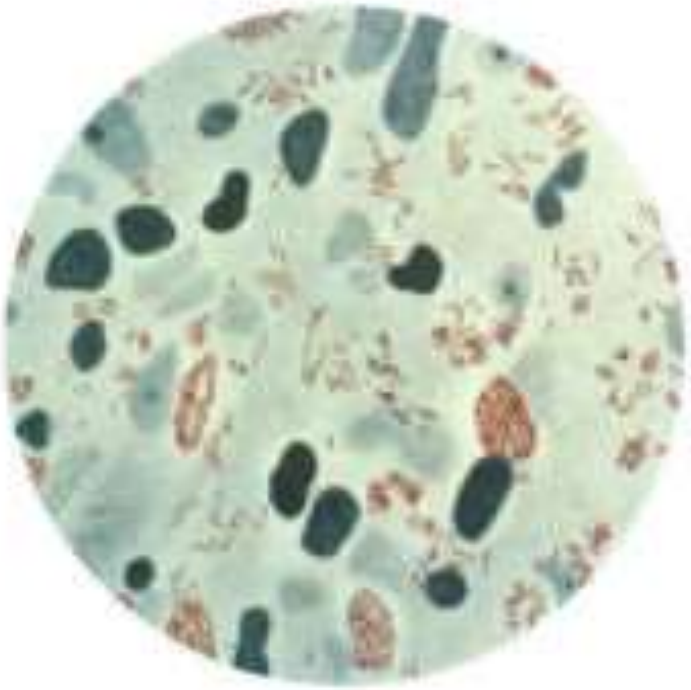
- Infectious bacterial disease of the skin, peripheral nerves and mucosa of the upper airway.
- Chronic, granulomatous.
- Only few from who exposed to infection develop the disease.

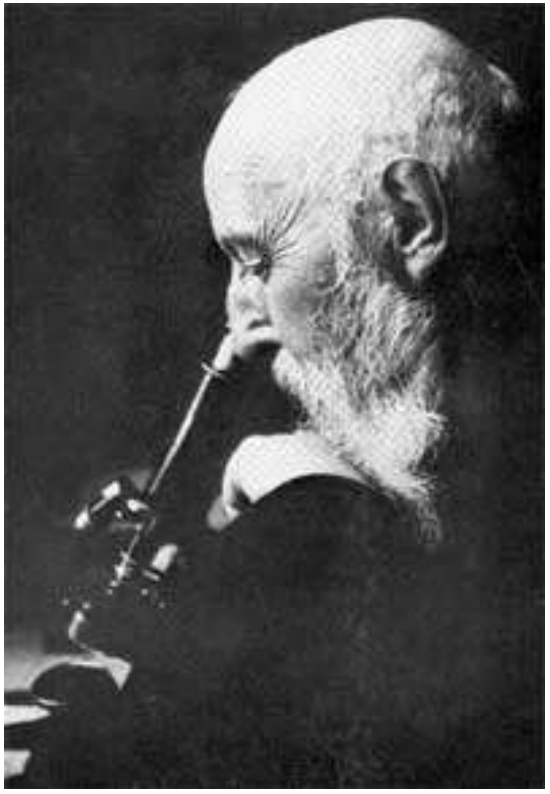
Causative agent

- Mycobacterium leprae.
- Acid fast, rod shaped bacillus.
- Stain with Ziehl Neelsen carbol fuchsin.
- acid-fast intracellular Gram-positive bacillus, which shows tropism for macrophages and Schwann cells

Background

Gerhard Henrik Hansen was a physician who first identified *Mycobacterium leprae* as the cause of leprosy in 1873





7/29/1841-2/12/1912

Transmission…

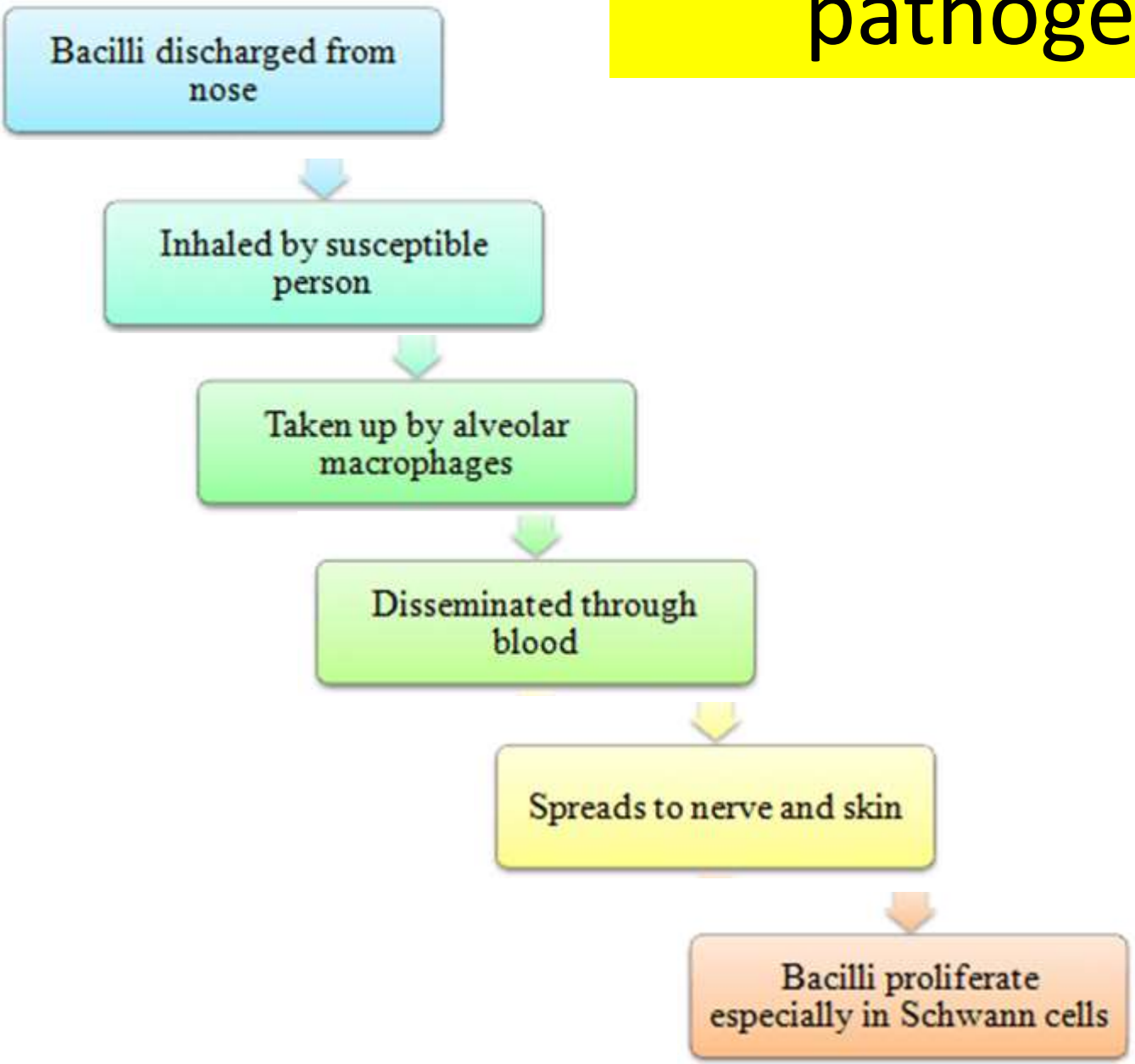
- **Airborn,** contact with infected soil, and insect vectors**.**
- Leprosy is not known to be either sexually transmitted or highly infectious.
- People are no longer infectious after **as little as two weeks of treatment**.
- Two exit routes are the:
  A. Skin
  B. Nasal mucosa
- The entry routes are the:
  A. Skin
  B. The upper respiratory tract are most likely.

- **Reservoir:** Human being, only known. Similar organisms detected in wild armadillo. History of handling armadillos reported.

Epidemiology

- Age: All ages, from early infancy to very old age.
- Youngest age reported is 1 and a half months.
- Sex: Both. Males more than females, 2:1 (equal in Africa).
- Risk group: children, people living in endemic areas, in poor conditions, with insufficient diet, or have a disease that compromises their immunity (ie HIV).
- People who live in the areas where leprosy is endemic (parts of India, China, Japan, Nepal, Egypt, and other areas)

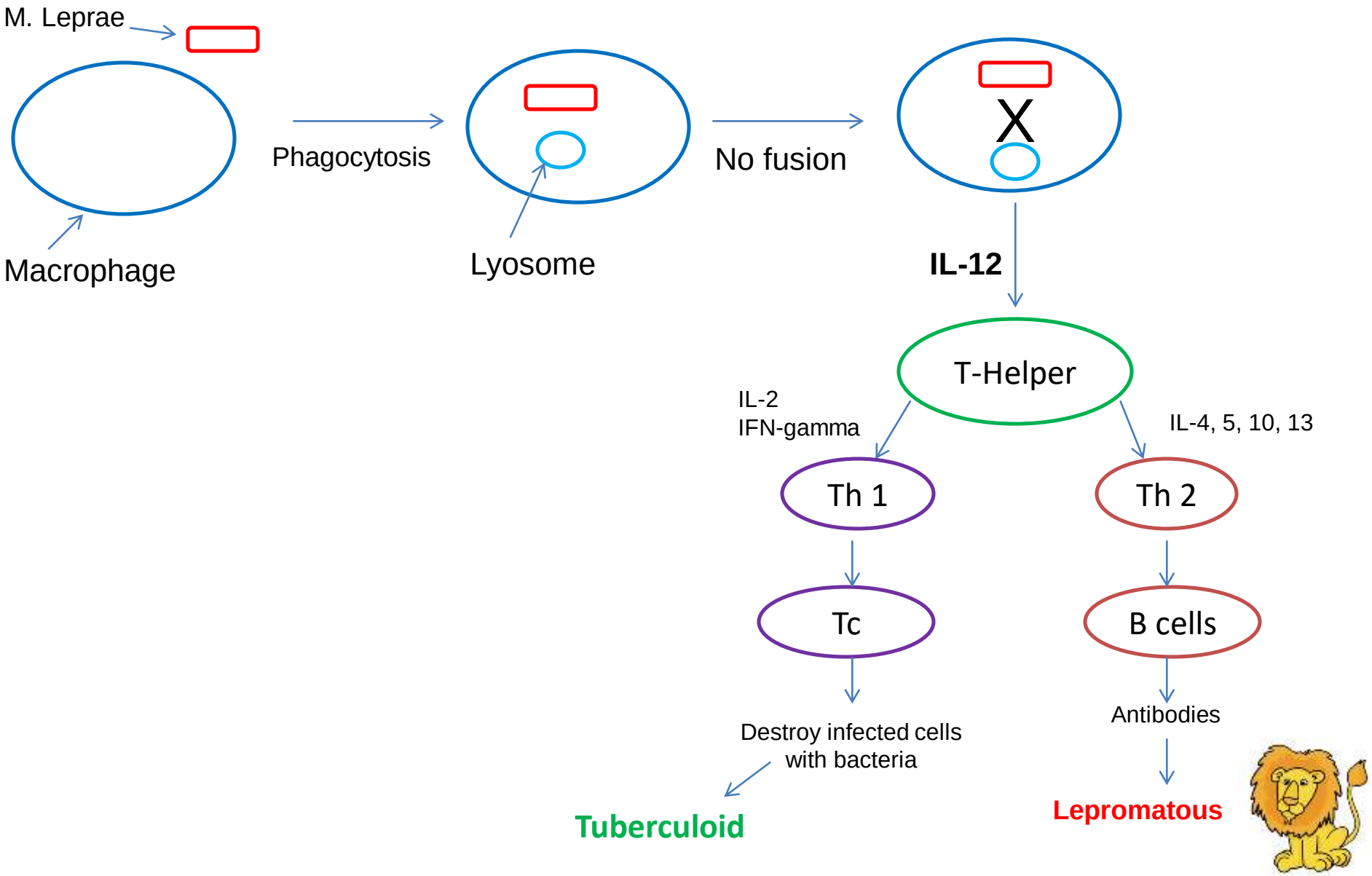
pathogenesis



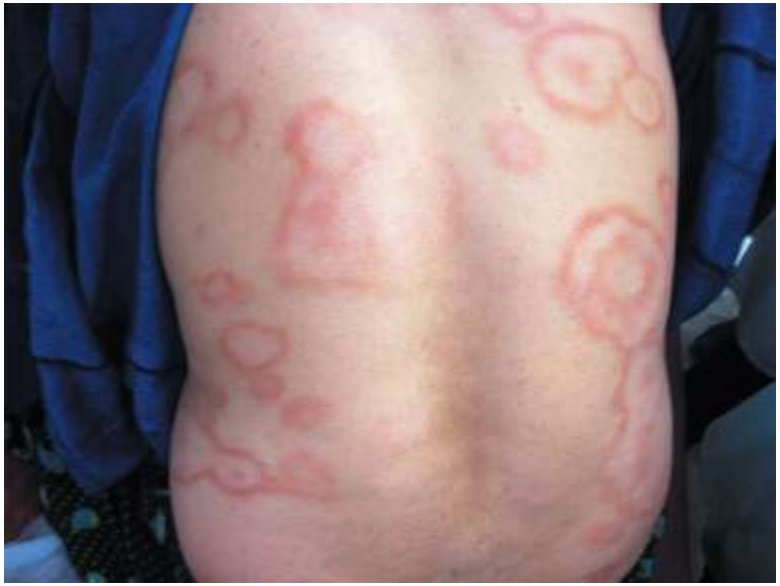
pathogenesis

- **The incubation period:**
  - Can vary between 2 and 40 years, although it is generally 5–7 years in duration
- *M. leprae* causes granulomatous lesions resembling those of tuberculosis, with epitheloid and giant cells
- The organisms are predominantly intracellular and can proliferate within macrophages, like tubercle bacilli.
- Leprosy is distinguished by its chronic slow process and by its damaging lesions.
- The organism has a preference for skin and nerves particular affinity for Schwann cells of the peripheral nervous sys.

Pathophysiology



pathogenesis



Tuberculoid form

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Lepromatous form (Leonine face)

pathogenesis

Tuberculoid leprosy

- Skin lesions typically develop in areas of nerve damage.
- **The skin ulcers occur by**
  - direct action of Mycobacterium leprae on the peripheral nerves, with changes in the sensory, autonomic and motor fibers (neuropathic ulcers).
  - direct invasion of bacilli in the vascular endothelium, causing vasculitis, cutaneous necrosis and ulcer.
- These lesions may have raised and erythematous border with a dry scaly appearance in the center with complete anesthesia.
- The skin lesions are commonly found on the face, limbs, buttocks, or elsewhere but are not found in the axilla, perineum, or scalp.
- Neuritis leads to patches of anesthesia in the skin.

pathogenesis

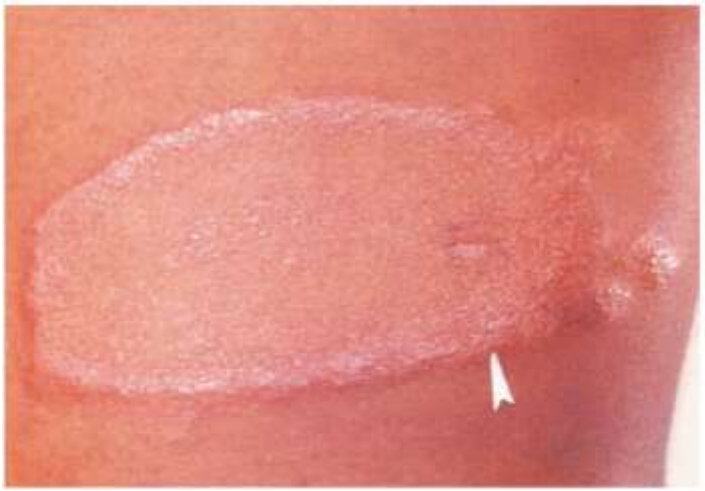
Tuberculoid leprosy

- The organisms grow and cause thickening in nerve sheaths.
- These thickened nerves can be felt through the skin, a characteristic of leprosy.
- Damage of the nerve can result in wrist drop or foot drop.
- There are few bacteria in the lesions also called as paucibacillary.
- The patient mounts a strong cell-mediated immune response and develops delayed hypersensitivity, which can be shown by a skin test with lepromin, a tuberculin-like extract of lepromatous tissue.

pathogenesis

Tuberculoid leprosy

- The infected individuals to exhibit large flattened patches with raised and elevated red edges on their skin. These patches have dry, pale, hairless centers, accompanied by a loss of sensation on the skin. The loss of sensation may develop as a result of invasion of the peripheral sensory nerves.



A well-defined, hypopigmented, anesthetic macule with anhidrosis and a raised granular margin (arrowhead).

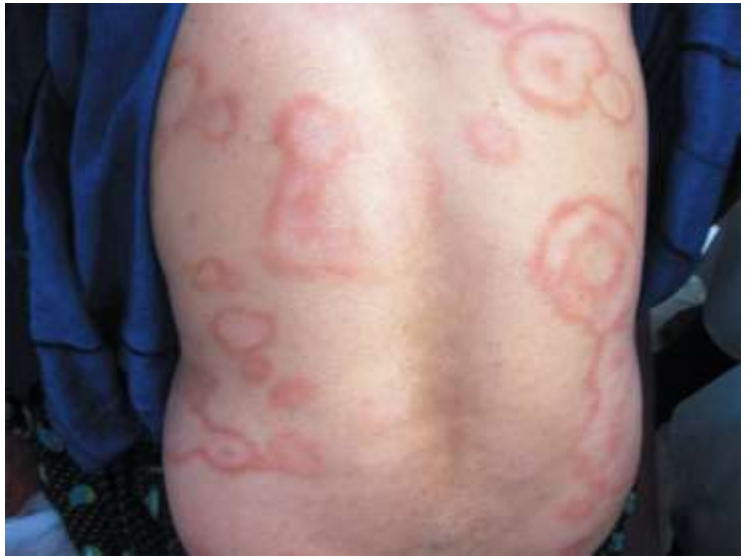


exhibit large flattened patches with raised and elevated red edges on their skin. These patches have dry, pale, hairless centers, accompanied by a loss of sensation on the skin.

pathogenesis

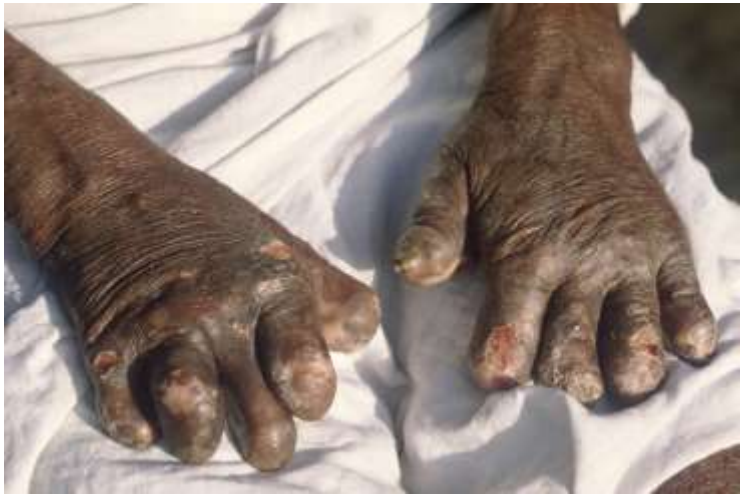
Lepromatous form

- This form of the microbe proliferates within the macrophages at the site of entry.
- Bacilli are numerous in the skin (as many as 109/g), where they are often found in large clumps , and in peripheral nerves, where they initially invade Schwann cells, resulting in foamy degenerative myelination and axonal degeneration
- patients present with symmetrically distributed skin nodules ,raised plaques, or diffuse dermal infiltration, which results in lion face appearance.
- Extensive penetration of this microbe may lead to severe body damage; for example the loss of bones, fingers, and toes.

pathogenesis

deformity

Lepromatous form



Loss of fingers

Case definition

(WHO operational definition):

- Is a person having one or more of the following
- Hypopigmented or reddish skin lesion(s) with definite loss of sensation
- Involvement of the peripheral nerves (definite thickening with loss of sensation)
- Skin smear positive for acid-fast bacilli.

Diagnosis

- In an endemic country or area, an individual should be regarded as having leprosy if shows :
  - skin lesion consistent with leprosy and with definite sensory loss, with or without thickened nerves
  - Detection of Mycobacterium leprae in slit skin smear is a gold standard technique for the leprosy diagnosis.
  - Lepromin positive test. People with a particular type of leprosy, called lepromatous leprosy, will also have no skin reaction to the antigen

Diagnosis

Lepromin test:

Method:

- Injection of a standardized extract of the inactivated bacilli intradermally in the forearm.
- **Positive reaction:** 10 mm or more induration after 48 hrs/ or 5 mm or more nodule after 21 days.
- Negative In lepromatous leprosy because of humoral immunity not cell mediated.

Treatment

- Infection caused by *M. leprae* is characterized by persistence of the microorganism in the tissues for years, necessitates very prolonged treatment to prevent relapse.

- For many years **dapsone**, a sulphone derivative has been used. This drug has the advantage that it is given orally and it is cheap and effective.

- However, widespread use as monotherapy has resulted in the emergence of **resistance** and multidrug regimens are therefore preferable. **Rifampicin** can be combined with dapsone. Alternatively clofazime is active against dapsone-resistant *M. leprae*, but it is expensive.

Case presentation

- A 20-year-old man reported a large single, hypopigmented, well defined anaesthetic lesion on his left thigh extending to his knee which had been present for 2 years.
- There was no other nerve involvement.
- Clinical diagnosis was tuberculoid leprosy
- Six months of multidrug treatment was advised immediately.