Mycobacterium leprae

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Objectives

- Causative agent.
- Background.
- Epidemiology
- Clinical features & complications.
- Diagnosis.
- Reservoir, incubation period & transmission.
- Treatment .
- Control .
- Elimination.

What is leprosy

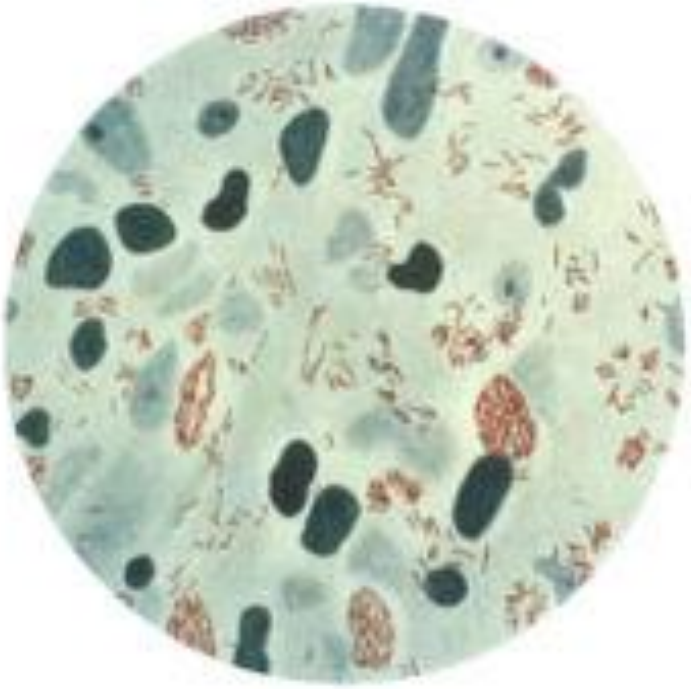
- Infectious bacterial disease of the skin, peripheral nerves and mucosa of the upper airway.
- Chronic and granulomatous.
- Only few from who exposed to infection develop the disease.

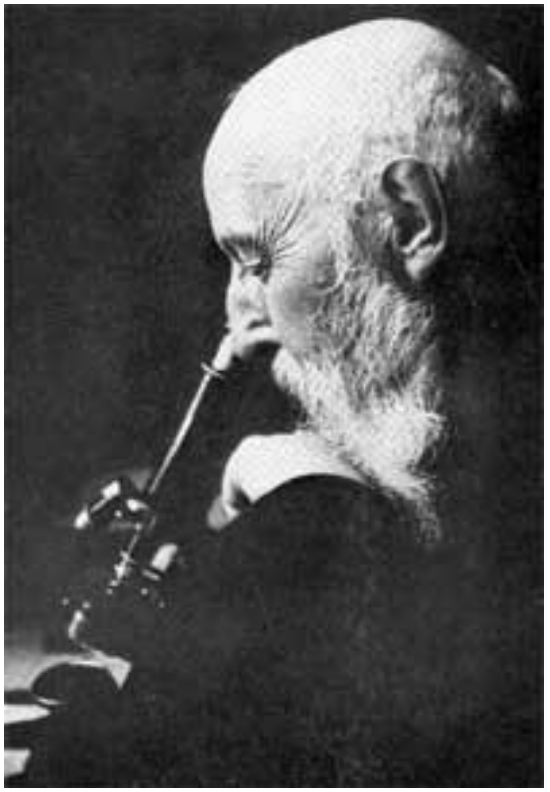
Causative agent

- Mycobacterium leprae.
- Stain positive with acid fast staining.

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…

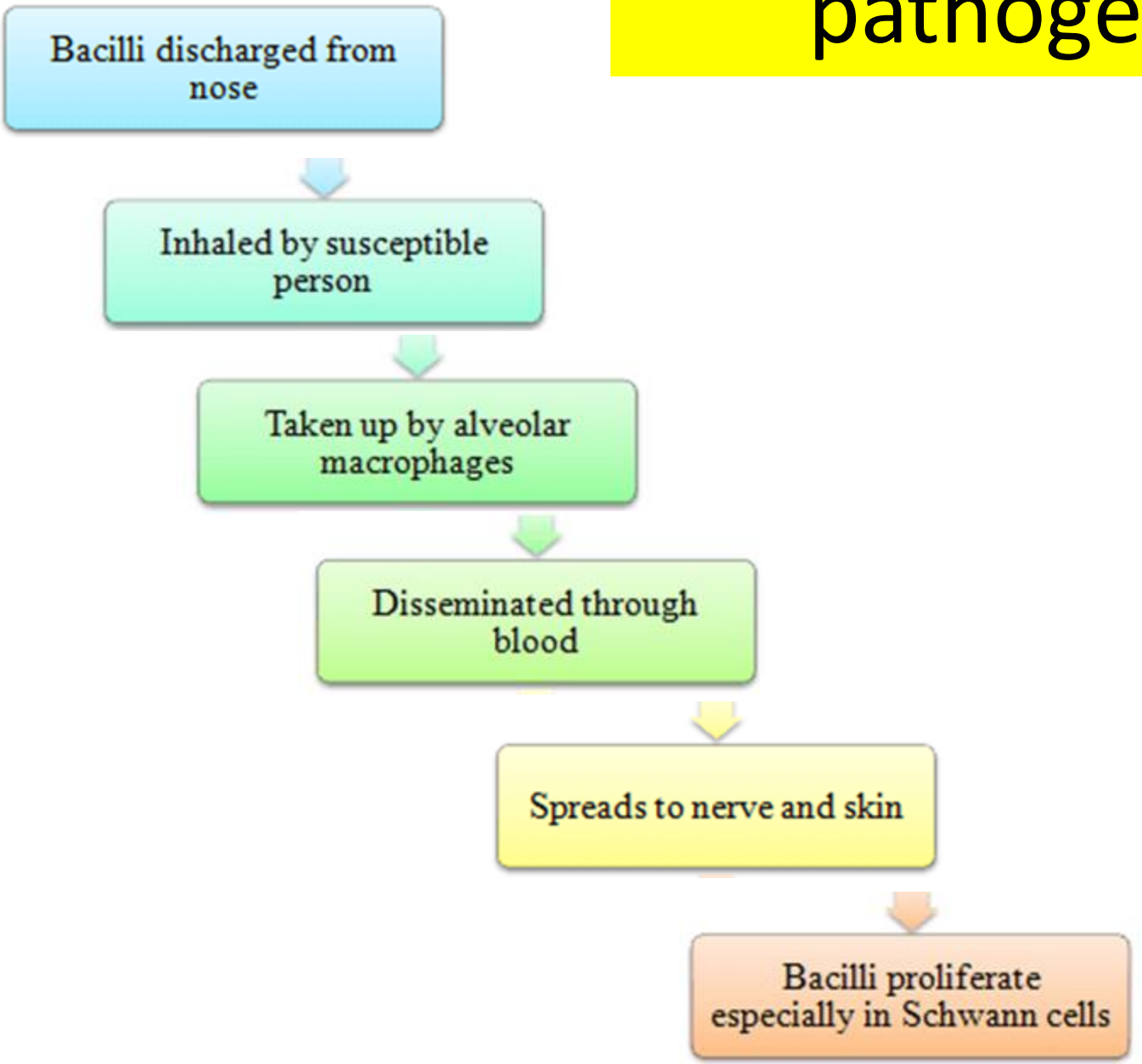
- **Airborn,** contact with infected soil, and insect vectors**.**
- Leprosy is not known to be either sexually transmitted or highly infectious.
- People are no longer infectious after **as little as two weeks of treatment**.
- Two exit routes are the:
  A. Skin
  B. Nasal mucosa
- The entry routes 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
- Sex: Males more than females (2:1), equal in Africa.
- Risk group:
  - children, people living in endemic areas (parts of India, China, Japan, Nepal, Egypt, and other areas
  - in poor conditions, with insufficient diet
  - have a disease that compromises their immunity (ie HIV).

pathogenesis

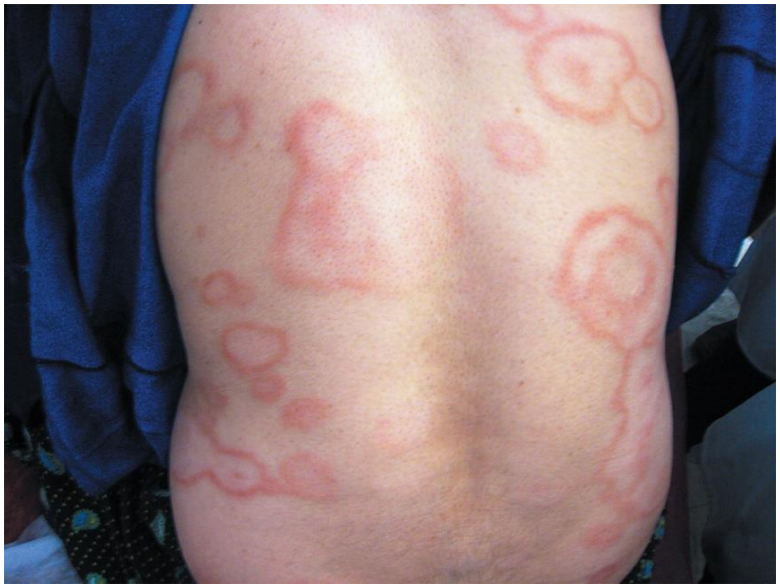


pathogenesis

- 2 to 40 years **incubation period (**average 5–7 years)
- *M. leprae* causes granulomatous lesions 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.

pathogenesis

Leprosy has two forms…….(Why)



Tuberculoid form

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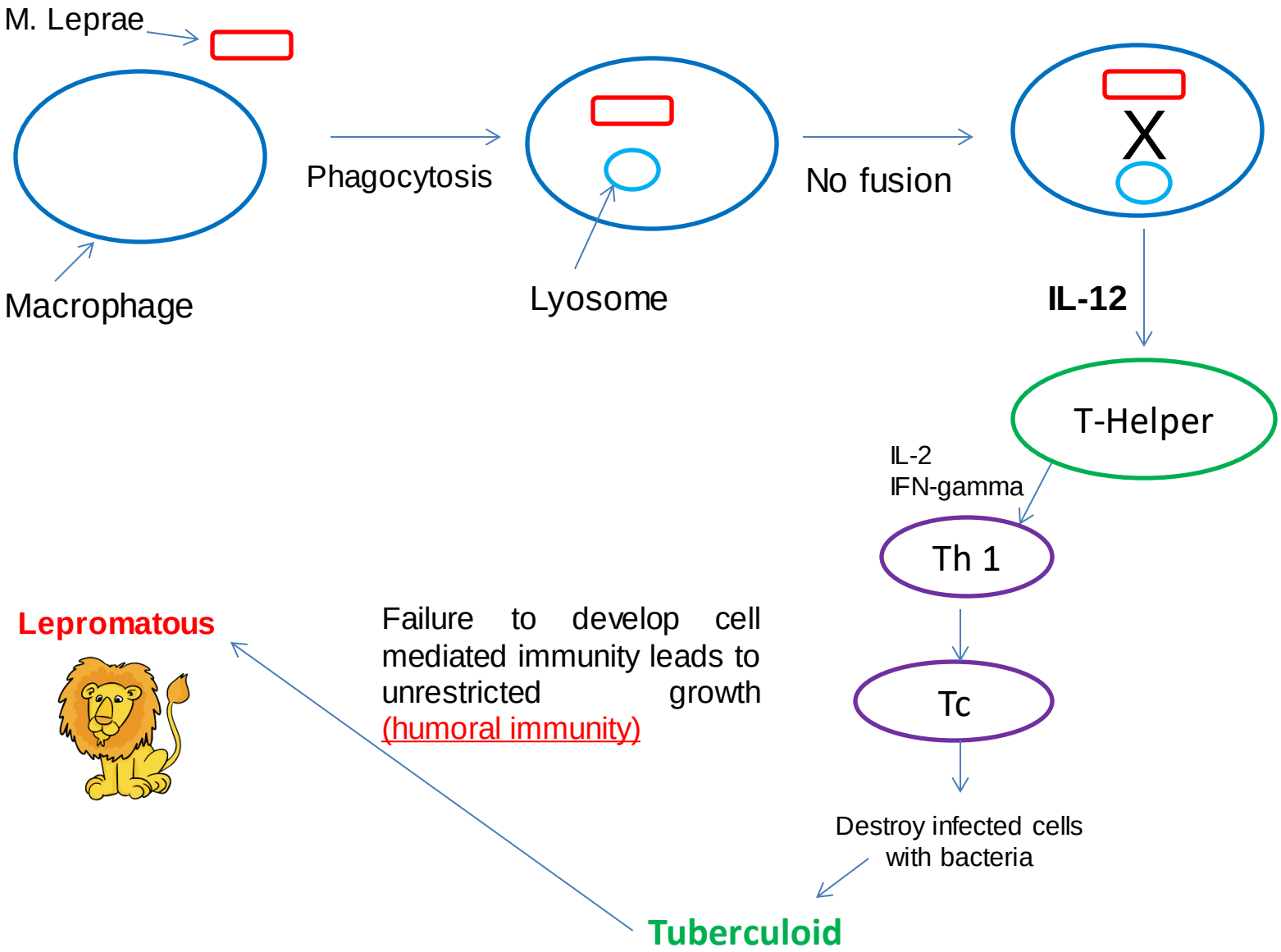
Lepromatous form (Leonine face)

Pathophysiology



pathogenesis

Tuberculoid leprosy

- Skin lesions typically develop in areas of nerve damage.
- **The skin ulcers occur by**
  - direct action of bacteria on the peripheral nerves
  - 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

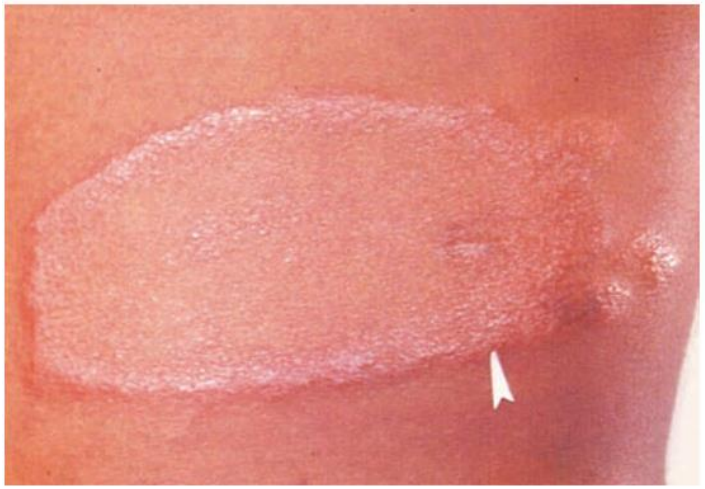
Tuberculoid leprosy

- 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 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.



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

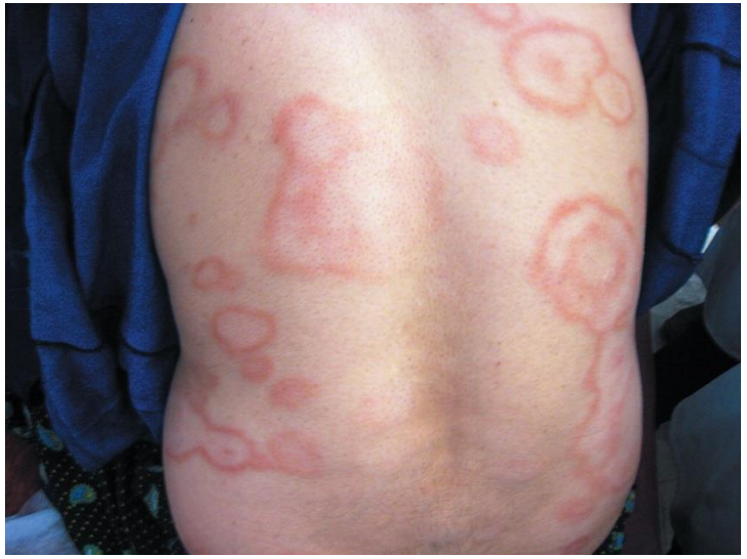


exhibit large flattened patches with raised and elevated red edges on their skin. These patches have dry, pale, hairless centers, accompanied by a loss of sensation on the skin.

pathogenesis

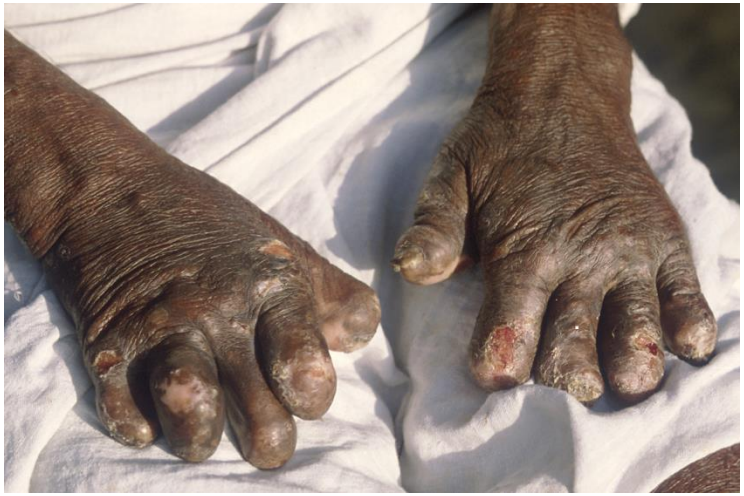
Lepromatous form

- 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

- Diagnosis of leprosy is most commonly based on the clinical sign and symptoms.
- In an endemic country or area, an individual should be regarded as having leprosy if shows ONE of the following basic signs :
  - skin lesion consistent with leprosy and with definite sensory loss, with or without thickened nerves
  - Positive skin smears.
  - Lepromin positive test.

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 45-year-old man comes to the opd due to a nonpruritic, nonpainful skin lesion on the right upper arm that began 3 months ago. He has also had tingling and numbness of the right fingers. The patient has no medical history and does not take any medications. He emigrated from Southeast Asia a year ago. Temperature is 97.8 F, blood pressure is 126/82 mm Hg, and pulse is 74/min. Skin examination shows a 4-cm, well-circumscribed, hypopigmented patch on the right upper arm with no sensation to pinprick. The ulnar nerve is thickened and tender at the right elbow. Touch and pain sensation is absent in the right ulnar nerve distribution. Which of the following is most likely to confirm the diagnosis in this patient?

A. *Anti-Borrelia burgdorferi* antibody assay

B. KOH preparation of skin scrapings

C. Nerve conduction studies

D. Skin biopsy from the edge of the lesion

E. Treponemal serologic testing

F. Tuberculin skin testing

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.