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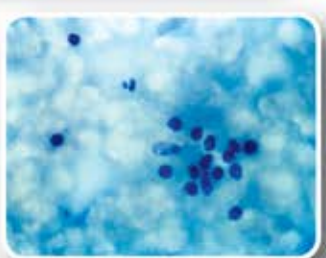
All About LEPROSY

2nd Edition







PVS Prasad

Foreword
Patrick Yesudian

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CHAPTER 3

Epidemiology

Prasad PVS

Chapter Outline

- Pathogen
- Incubation Period
- Portals of Entry and Exit
- Mode of Transmission
- Risk Factors

INTRODUCTION

The current strategy for eliminating leprosy is based on the presumption that once the prevalence is less than 1/10,000 at the global level (Box 3.1), transmission will dwindle and eventually stop.[1] This assumption is based on the fact that multidrug therapy (MDT) affects the pool of infectious patients, but the question is whether it affects this pool sufficiently is yet to be answered. Using registered prevalence as a proxy indicator for the epidemiology of leprosy (Box 3.2) has several caveats.[2] By definition, it only reflects registered cases and thus obscures true leprosy cases reported to private practitioners or practicing specialists. The prevalence taken for leprosy control is point prevalence and not a period prevalence. Therefore incidence of the disease is a better monitor, but so far no data are available on incidence rates.[3] Case detection rates (CDRs) are available for leprosy, but are far from good indicators of incidence, let alone transmission. The CDR may be inflated by backlog cases and by defaulting and relapse cases, and is heavily influenced by intensity of case finding effects. Trends in CDR

BOX 3.1: Global statistics

- Number of cases in 1985—12 million
- Global registered prevalence of leprosy at the beginning of 2011—192,246 cases
- New cases detected during 2012—232,857
- Number of cases in 2013—189,018
- New cases detected every year—500,000

BOX 3.2: Epidemiological features

- Age prevalence: Children are more affected
- Youngest age: 3 week
- Incubation period: 3–7 year
- Risk of infection: Intrafamilial
- Other factors: Poor hygiene, overcrowding
- Top 6 countries: India, Brazil, Nepal, Mozambique, Angola and Myanmar
- India accounts for 78% of cases detected
- Portal of entry: Skin and upper respiratory tract
- Portals of exit: Oral, nasal mucosa, skin and breast milk

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may thus be distorted as a result of bias and confounding. CDR trends at a global level may be relatively stable, while at a country level CDRs may show a declining, stable or even a rising trend. *Mycobacterium leprae* (*M. leprae)* cannot be cultured in vitro and hence it is impossible to assess the exposure and the onset of infection and disease. As a consequence, the chain of infection, considered as the relationship between *M. leprae* transmission and the human host is poorly understood.

PATHOGEN

Mycobacterium leprae cannot be cultured in the laboratory and thus cannot be used to meet Koch's postulates. It is an obligate intracellular bacterium in the order actinomycetales. It is nonmotile, non-spore-forming, microaerophilic, acid-fast-staining bacterium that usually appears as slightly curved or straight rods (refer Box 3.1). It measures 1.0–8.0 × 0.2–0.5 μm and its optimal growth temperature has been determined to be 30°C. In mice it divides only once in 12–14 days and the bacteria may remain viable for up to 2 days outside the body. *M. leprae* is a poor pathogen. The presence of *M. leprae* is not associated with disease per se. The presence of antibodies to PGL-1 is probably specific, but good quality testing often indicates only a relatively low percentage of positives (up to 5%) among the very high-risk groups such as household contacts of multibacillary patients.[4] Transient nasal carriage of *M. leprae* in the healthy population, as evidenced by polymerase chain reaction (PCR) is not likely to reflect infection, but rather environmental contaminants.[5] On the basis of these studies, it is often concluded that *M. leprae* infection is far more common than believed, thus implicating causal contact as a major factor in transmission, as in tuberculosis (TB).

INCUBATION PERIOD

It is difficult to measure the incubation period of leprosy as it is difficult to calculate the time of onset of infection and the time of onset of the disease as its early indeterminate stage may be asymptomatic. Studies showed a wide variation in the incubation period from 3 months to 3 years and in an average it lasts from 3 to 7 years.

PORTALS OF ENTRY AND EXIT

Multibacillary (MB) leprosy patients are thought to be a main source of infection, being able to shed large numbers of bacteria from the nose, on an average 10^7 viable *M. leprae* per day.[6] If indeed, nose is the portal of exit of *M. leprae*, transmission is likely to be mediated by droplet spread. Droplet spread requires close proximity and do not spread more than 1 meter. Apart from portal of exit, nose is also the portal of entry of bacteria. This entry may be facilitated by small injuries to nasal mucosa.[7] Impairment of nasal function may enhance the development of disease, as seen in other infectious diseases.

The skin is also considered as a portal of exit and entry of *M. leprae*. This is due to prominent role played by the skin in the disease manifestation. Untreated lepromatous patients are likely to be able to shed large numbers of bacilli from their ulcers or from injured skin. There is no evidence that the bacteria can penetrate intact skin. Only accidental inoculation with *M. leprae* would favor the skin as possible portal of entry. Evidence for a possible role of arthropods in transmission of leprosy is circumstantial at best.[8]

MODE OF TRANSMISSION

Unlike most other infectious diseases, the mode of transmission for leprosy is still under debate. Most leprologists suggest the most likely route of infection is airborne by way of upper respiratory tract. This seems

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logical because of abundance of *M. leprae* in the nasal discharge from lepromatous leprosy (LL) patients and the evidence that *M. leprae* is discharged in the environment through sneezing and coughing.[9] Some authors believe that the most important port of exit of *M. leprae* is the respiratory system, particularly in the nose; its dissemination through the skin seems to be less important.[10]

Other authors say that *M. leprae* enters the body via the nose and then spreads to the skin and nerves via circulation.[11] Alternative source of *M. leprae* infection could be subclinically infected individuals, a vector or environmental contamination.[12] Naffs pointed out that the skin might be an important portal of entry of *M. leprae* especially, if skin is abraded or ulcerated.[13]

Household Contact

Most cases of leprosy arise from non-contact population, similar to TB, where casual contact plays a significant role in transmission. The long incubation time of leprosy makes it difficult to obtain contact histories.

Close contact is more important than often believed. The risk of developing leprosy is greatest among close household contacts, neighbors and social contacts. The attack rate among persons living in the household of a multibacillary patient is five to ten times as high as among noncontacts. Household contacts of paucibacillary (PB) patients show a twofold risk of developing the disease when compared to noncontacts. Thus, the likelihood of the disease varies with the intensity of the infectious source and degree of contact.[14]

Studies on risk factors for developing leprosy showed contacts of MB cases have an eight-fold increased risk, compared with noncontacts, while contacts of PB cases have only slightly increased risk. A recent study from Indonesia showed that neighbors and social contacts also have an increased risk, so that up to 78% of new cases could be linked to a previously diagnosed leprosy patient. The relative risk for household contact was 9.4, for next door neighbors 4.0 and for the next house 1.6. Hereditary factors also play a role. Age is also a risk factor with two peaks of high incidence, firstly in children under 14 years and secondly in older adults.[15]

Environmental Sources of *Mycobacterium leprae*[16]

Though *M. leprae* is considered to be primarily a parasite of humans, there are evidences to indicate a possible non-human source of the agent. Thus different authors have suggested that *M. leprae* may be harbored in the soil, water, on plants, insects, fish, primates and armadillos. Extra human source of *M. leprae* is important for leprosy epidemiology and control. If non-human sources exist, their recognition may help to explain patterns of infection and disease in human population.

Leprosy cases can shed large numbers of bacilli into their environment through bodily secretions, or while sneezing, coughing or talking. Some leprosy bacilli can remain viable in certain cell-free environments for periods of hours, days or even weeks. They may even replicate. There are only two non-human environments for the *M. leprae*. They are the foot pad of mouse (*Mus musculus*) and the nine-banded armadillo (*Dasypus novemcinctus*). *M. leprae* is harbored in wild armadillos over a wide area of North America.

This includes the typical pathology, skin test reactivity, genomic sequence analysis, specific serology and PCR isolation and passage of the bacilli in other animals and epidemiological associations of armadillo contact with human cases.

Other Sources of Transmission

Studies from JAMA Institute of Leprosy had shown that most LL patients harbor *M. leprae* in the oral mucosa, nasal mucosa and in

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genital mucosa. Acid-fast bacilli (AFB) are secreted from breast milk and may be responsible to infect 32% of breastfed infants. The mean count of bacilli ranged from 4.3×10^4 to 1.53×10^5/10 mL of breast milk. Interestingly, significant amount of antileprosy drugs are also secreted in breast milk after initiation of chemotherapy and thereby reducing the risk of infection. Leprosy bacilli do not cross the placenta, but bacillary antigens may be able to do so.

RISK FACTORS

The immune status of the host has been implicated as an important factor in the process leading from infection to disease and to a lesser extent to transmission itself. The environment can both undermine and boost the immune system, while innate resistance is determined by genetic determinants. Genetic predisposition is the only factor in the complex process leading to leprosy. Similarly, socioeconomic status involves a complex of associated variables and it is difficult to assess the relevance of each variable. However, as is true for most infectious diseases, improvement of socioeconomic circumstances is an efficacious means for control, if only because it goes along with improved quality and coverage of health services. Bacille Calmette-Guérin (BCG) vaccination provides a wide range of protection according to various studies from various countries. The protection ranges from 20 to 80%.[17] The BCG vaccine also leads to a shift in immune response from multibacillary to PB leprosy. Human immunodeficiency virus infection suppresses cellular immunity and the disastrous effects on TB are known. There is no evidence that HIV epidemic leads to a significant increase in number of leprosy patients. The impact of HIV may be minimal as it takes too much time for symptoms to develop from an infection with *M. leprae.*[13]

Age Prevalence

Children are more susceptible than adults. The youngest age reported was 3 weeks in Martinique. The disease peaks at 10–14 years and again between 30 and 50 years. Male to female ratio is 1.5:1. Males have greater risk of infection as a result of their lifestyle. Similarly children show lesions on the exposed areas. The incidence in children is 3% with no infected parents and an incidence of 5% if one or both parents are affected. The rate of conjugal transmission is around 5%.

CONCLUSION

There is a definite decline in the epidemiology of leprosy. The pathogen is fully understood and its characteristics have been studied well. As the incubation period is very much prolonged from 3 to 7 years, which makes difficult to eliminate the disease. Even though nasal mucosa is the important source of entry of the organism, other modes of spread have also been proved. The chances of getting infected through household contacts are found to be very high. Some animals including armadillo can harbor *M. leprae,* which may be one of the reasons for continuous survival of the organism. Children are more susceptible to get the infection than adults. The incidence of leprosy in children is a good indicator of the leprosy control in the world.

Key Points

- *Mycobacterium leprae* is nonmotile, non-spore-forming, microaerophilic, acid-fast-staining bacterium that usually forms slightly curved or straight bacteria.
- Antibodies to phenolic glycolipid-1 (PGL-1) is probably specific.

Contd...

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Contd...

- Causal contact as a major factor in as in tuberculosis.
- The most likely route of infection is airborne by way of upper respiratory tract.
- The immune status of the host has been implicated as an important factor in the process leading from infection to disease.
- There are only two non-human environments for the *M. leprae*. They are the foot pad of mouse and the nine-banded armadillo.
- Acid-fast bacilli (AFB) is secreted from breast milk and may be responsible to infect infants.
- The youngest age reported was 3 weeks.

VIVA VOCE QUESTIONS AND ANSWERS

Q1. What was the prevalence and detection rate of leprosy in the beginning of 2001?

Prevalence	India	World
Number of cases	439,782	635,404
Prevalence rate per 10,000	4.3	1
Cases detected in 2001 (data as per January 2002)	635,404	763,315

Q2. What is the youngest age of occurrence reported in leprosy?
The youngest age reported for the occurrence of leprosy was 3 weeks in Martinique.

Q3. What is the peak age of onset of leprosy?
The peak ages range from 10 to 14 years and 30 to 60 years.

Q4. What is the sex distribution in leprosy?
Males are affected more frequently than females in the ratio of 2:1.

Q5. What is the reason for male preponderance?
Males in general expose themselves to greater risks of infection as a result of their lifestyle and hence there is a male preponderance.

Q6. What are the predisposing factors of the disease?
They are hot and humid climate, nutritional deficiencies, low literacy rate and socioeconomic status.

Q7. Why do we consider leprosy as an epidemic disease?
The number of cases reported in Nauru Island between 1912 and 1924 increased from 12 to 24%. This is a classic example to consider that leprosy is an epidemic disease.

Q8. What is the natural course of TT leprosy?
An earlier study conducted at Saidapet, Madras state in India before dapsone era had shown that 90% of tuberculoid cases healed on its own.

Q9. What is the maximum load of organisms per gram of tissue?
The maximum load is 7 billion organisms per gram of tissue.

Q10. What is the total number of organism, in the nasal secretion per day?
Nasal secretions from lepromatous patient can yield 10 million viable organisms per day.

Q11. What is the number of organism in breast milk?
Single feed from a lepromatous mother can yield 2 million organisms.

Q12. What are the portals of entry and exit for leprosy?
- The portals of entry are nasal mucosa and skin
- The portals of exit are the oral, nasal mucosa, skin and breast milk.

Q13. What are the modes of transmission of leprosy?
The modes of transmission are:
- By direct contact

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- By respiratory route
- By ingestion of the bacilli through breast milk and through insects
- By transplacental route.

Q14. What are the important factors in the transmission of the disease?

They are closeness of contact and severity of infection of index case.

Q15. What is the period of viability of *M. leprae* outside the body?

Mycobacterium leprae can survive up to 36 hours outside the human host.

Q16. What is the incubation period of leprosy?

- The incubation period of *M. leprae* ranges from a few weeks to as many as 30 years. It is 2–5 years on an average
- The incubation period varied between 2.9 and 5.3 years for tuberculoid leprosy, and 9.3 and 11.6 years for lepromatous leprosy.

Q17. What are the tests for subclinical infection of leprosy?

They are lymphocyte transformation test (LTT), which is an in vitro test for CMI, phenolic glycolipid-1 based enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR), ligase chain reaction (LCR) and FLA-Abs test, which are serological tests for detecting humoral antibodies.

Q18. What is the incidence of conjugal infection reported from various countries?

The rate of conjugal infection is 0.3 % from Japan, 5.5% from South India and 7.8% from Brazil.

Q19. What is the attack rate of leprosy from parents to children?

If only mother is affected the attack rate is 6.8 and 3.0%, if father is affected.

Q20. What are the evidences for carrier state in leprosy?

- Nasal mucosal biopsy of healthy household contacts has revealed local proliferation of nerves and chronic inflammation in a significant proportion suggesting a carrier state at this site
- About 10% of the general population has antibodies to PGL-1 epitope of *M. leprae* with over 80% showing low antibody levels.

Q21. What are the factors that determine the clinical expression of leprosy?

They are:

- Genetic factors.
- Route of infection.
- Reinfection.
- Host's immune status.

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Salient Features

- Book has been updated almost with the latest features
- 100% more information compared to first edition
- More photographs for better understanding
- Viva Voce Questions at the end of chapters
- Updated appendices with important university examination questions and glossary for understanding terms
- Recent concepts such as immune reconstitution inflammatory syndrome, rehabilitation, etc. have been added
- Separate chapter on recent advances in leprosy
- Unique chapter on panel discussion has been added incorporating various questions and answers discussed in various conferences on leprosy
- Enables readers to be updated with latest scenario and also with positive/negative aspects of various issues in leprosy.



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