

SKIN

Homeopathic Approach
to
DERMATOLOGY

Second Revised Edition



Includes
Colour Photographs

Dr. Farokh J. Master, M.D.

SKIN

HOMEOPATHIC APPROACH

to

DERMATOLOGY

DR. FAROKH J. MASTER

M.D. (Hom.)

Associate Editors

Dr. Priya Jani, B.H.M.S.

Dr. Pinky Bilimoria, B.H.M.S.

Dr. Farhad Adajania, B.H.M.S.

Dr. Jeenal Shah, B.H.M.S.

Dr. Jayesh Dhingreja, B.H.M.S.

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Preface to the Second Edition

The birth of this book was in the year 1993 (first edition). Subsequently, with support and encouragement from my colleagues in India and abroad, there was a reprint of my book in the year 1994. It has since then been acclaimed all over the world as a standard therapeutic book of dermatology.

I stress the fact, especially to younger generation, that recognition and treatment of skin disease is an important part of everybody's practice, since diseases of the skin covers 25% of the practice.

I also recommend to read more standard and reference books on dermatology like for example: Andrews' Diseases of Skin – Clinical dermatology.

This second edition has been completely re-written in light of the present day knowledge. Many chapters where homeopathic therapeutics was incomplete or not mentioned in the earlier editions have been completed or included. Many new conditions like Tuberculosis of skin, Hansen's disease, Disorders of pigmentation, Ichthyosis, Chicken pox, etc. have been included in the present edition. New cases have also been added. The peculiar symptoms of the homeopathic remedies has been described elaborately.

I sincerely acknowledge my thanks to all my extremely loving colleagues Dr. Vanmala Shroff, Dr. Jayesh Dhingreja and Dr. Trupti Pradhan who have so kindly and willingly worked for me. Their contribution has enhanced the prestige of this book.

I also thank Miss Sunita Shah for using digital camera and incorporating the photographs in Synthesis and Radar, helping me to search precise information from reference books and for her extra forbearance during my busy practice.

Finally my sincere thanks to Dr. Nandkishor Sonawane and Dr. Saudamini Suryawanshi who helped me in preparing therapeutics for this book.

Finally I thank Almighty for giving me good health, my wife Dilnavaz and my daughters Rukhshin and Mahaziver who have supported me all throughout.

Farokh J. Master, M.D. (Hom.)

24th May, 2006

“It is not only unbridled passion which damage the health of man. Living on ill-gotten money also causes ill health to some extent. Living on earnings got by unjust means causes many unknown diseases to take root in us.”

It is said,

“As is the Food, So is the Mind

As is the Mind, So are the Thoughts

As is the Thought, So is the Conduct

As is the Conduct, So is the Health.”

Bhagavan Sri Sathya Sai Baba

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Hansen's Disease - Leprosy

Synonym

Hansen's disease, Hanseniasis, Elephantiasis grecorum.

Definition

Hansen's disease or Leprosy is a highly infectious, chronic granulomatous disease that is caused by *Mycobacterium leprae*, where the peripheral nerves and the skin are primarily affected. The disease is so named since the bacilli known to cause it was discovered by Hansen.

Etiology & Epidemiology

All cases of leprosy, both in human being and the animal kingdom, are caused by the acid-fast *Mycobacterium leprae*, which is classified separately from the other mycobacteria due to the inability to culture it in vitro. They are pleomorphic, straight or curved, acid-fast, rod-like bacteria occurring in clumps like bunches of cigars inside the phagocytes termed lepra cells. Man is regarded as the only natural host of the disease though wild armadillos may also be infected.

This disease condition, though on a decrease now, is known to be endemic in *tropical* and *developing countries* like Bangladesh, Burma, Brazil, India, Indonesia, Central Africa, South and Central America, the Pacific Islands, the Philippines and the Hawaii.

- It is a disease that is usually acquired through genetic susceptibility, where the rate of incidence is especially high in cases of monozygotic twins.
- It is also seen to spread easily in areas where the general susceptibility of the population is low, with a poor nutritional status, inadequate housing, and unsuitable sanitation.
- Prolonged and close contact is ideal for its transmission. The disease occurs due to droplet infection, which is spread through an infectious patient either through coughing or sneezing; or it results from direct exposure to carrier organisms like armadillo, cockroaches or mice. Children are apparently more easily infected in these situations than adults. Indirect transmission may occur through wearing an infective patient's clothes and from articles of daily use. The point of entry could be either through the skin, mucous membranes of the respiratory tract or the GIT.
- Decorative tattooing, a trend that seems to be picking up in different countries, is another mode of transmission of the infection.
- It is seen especially in male adults rather than female adults. The peak ages of affection are from 10-15 years or between 30-50 years of age.
- Incubation period varies from 2-5 years, but can be much longer, especially in cases of lepromatous leprosy, where the incubation period can go upto 8-12 years.

Pathogenesis

In a significant number of individuals, especially children, there is presence of *Mycobacterium leprae* in the skin, without giving rise to any clinical symptoms; and this stage is called the 'silent phase' of the infection. The host's immune reaction to the leprosy

bacillus is an important factor in determining the outcome of the infection. The classification of this spectral disease is thus based on differences in the cell-mediated immunity of the individuals.

At one pole, individuals with a good immunity are known to develop the *tuberculoid* type of leprosy; and at the other pole there are individuals with a poor immune status who tend to develop the *lepromatous* type. Between these two poles, there are individuals with an intermediate level of immunity tend who to develop the *borderline* type (BB) of leprosy. In this group individuals showing features closer to lepromatous leprosy are labeled as 'borderline lepromatous' (BL), and those with features closer to tuberculoid leprosy are labeled as 'borderline tuberculoid' (BT) types. One should remember that although the cell-mediated immune response of lepromatous patients to *M. leprae* is reduced, these patients are not immune suppressed for other infectious agents.

Restricted spread and self-healing are characteristic features of the tuberculoid type, whereas borderline and lepromatous types are more progressive, with a slow and extensive bacillary spread, with antigen overload, occurring at the sites of the lesions in cases of lepromatous leprosy.

Lepra reactions that are immune-mediated reactions are commonly seen in borderline cases of leprosy. Type 1 reactions are reversible ones caused by a delayed hypersensitivity after recognition of *M. leprae* antigens in the skin and various nerve sites. Type 2 reactions, with the development of erythema nodosum, occur due to immune-complex deposition and occur in borderline lepromatous and lepromatous leprosy cases.

Tuberculoid patients have positive lepromin skin tests and lepromatous patients negative ones.

Histopathology

The organism *M. leprae* is known to affect primarily the peripheral nervous system, entering the nerve and affecting especially the Schwann cell via endoneural blood vessels. Abnormalities in nerve-conduction studies and a histological picture

of small fibre loss are seen with segmental demyelination and remyelination.

Punch biopsies performed from the active border of typical lesions will be adequate for diagnosis.

TUBERCULOID LEPROSY

In this type of leprosy, histiocytes are turned into epithelioid cells. There is the presence of dermal tuberculoid granulomas forming foci around the nerves and skin appendages. The granulomas, along with an infiltrate of macrophages, lymphocytes and giant cells, tend to extend right up to the epidermis. No acid-fast bacilli are seen, but diagnosis is established by checking for selective destruction of nerve trunks or swollen cutaneous nerves.

LEPROMATOUS LEPROSY

Here there is thinning of the epidermis with the typical diffuse leproma in the deeper layer of the dermis, consisting of foamy macrophages (foamy lepra cells) with the addition of lymphocytes and plasma cells. Acid-fast bacilli are typically abundant. In cases of neuropathic affections in this type of leprosy, one finds asymptomatic bacillation, followed by foamy degeneration of the Schwann cells. There is a resultant demyelination, damage and destruction of the neural axis, followed by Wallerian degeneration, nerve fibrosis and hyalinization in later stages.

BORDERLINE LEPROSY

In *borderline tuberculoid (BT)* leprosy, the changes are similar to that seen in the tuberculoid type, except that the epithelioid cell granulomas is more diffuse and shows some vacuolation, and the acid-fast bacilli may be present. There is an uninvolved narrow, papillary zone that separates the inflammatory infiltrate from the overlying epidermis. Also, the dermal nerves are moderately swollen by cellular infiltrate or may show only Schwann-cell proliferation.

In *mid-borderline (BB)* leprosy, diffuse epithelioid granulomas are seen with very scanty lymphocytes with foamy cytoplasm, and absence of giant cells. The nerves are slightly swollen by cellular infiltrate, and acid-fast bacilli are present in moderate numbers.

In *borderline lepromatous (BL)* leprosy, foamy changes are seen with increased lymphocytes, which are dispersed diffusely within the small granulomas. The Schwann cells are full of bacilli and the resultant widespread nerve damage occurs due to acute neuritis. Oedema of the granuloma compresses on the remaining Schwann cells causing rapid functional loss in an already compromised nerve.

Clinical Features

The range of symptoms seen in Hansen's disease can be said to have two stable poles – the tuberculoid and the lepromatous types. The early or initial signs seen in this insidious condition are pretty indeterminate and can usually go unnoticed. They consist of an area of numbness or presence of solitary, mildly hypopigmented spots or rarely a red erythematous macular formation on the skin, with poorly defined margins. The areas that are usually affected are the cheeks, upper arms and thighs, buttocks or the trunk.

The earliest sensory changes are loss of sensation of cold and light touch, especially in the feet and hands. Peripheral nerves are not enlarged and there is no formation of plaques or nodules.

This state may persist for a few days, but the picture gradually evolves into either of the two poles or if the immunity of the individual is good, the condition may spontaneously be resolved with no signs or symptoms of leprosy.

Further discriminating or characteristic features of each type are as follows:

TUBERCULOID LEPROSY

In cases of this high-resistance form of leprosy, only a few solitary lesions tend to develop, which are well-defined, raised, erythematous, copper coloured or purple with a flattened and hypopigmented center, distributed asymmetrically. The lesions are seen most commonly on the face, limbs or the trunk. Neural involvement is early and prominent in tuberculoid leprosy and in quite a few cases, there may be no skin lesions, but only symptoms like pain and swelling of the affected nerve is seen followed by anesthesia, muscle weakness, muscle wasting and paralysis of the facial muscles and foot drop.

The damage to the nerves in the skin makes the lesion anesthetic while damage to the neighboring structures such as the hair, the sebaceous glands and the sweat glands causes loss of hair, dry scaly skin and anhidrosis, respectively.

Diagnosis

On examination, usually just beyond the outer edge of the lesion, there is a thickened or indurated sensory nerve or nerve trunk palpable, which may be tender to touch. A positive Lepromin skin test is diagnostic.

Prognosis

The prognosis is good with spontaneous remission occurring in quite a few cases, usually within three years of time, with or without treatment.

LEPROMATOUS LEPROSY

This highly bacillated form of leprosy occurs in those with a low immunity and is always slowly progressive.

Initially the patient may only present with coryza, noseblock, epistaxis or oedematous swelling of the hands and feet. Gradually one can see small and multiple lesions on the skin, which are

bilaterally symmetrical, diffuse, and hypopigmented occurring in the form of macules, papules or nodules. In cases of macules, the lesions are small, ill-defined, multiple, shiny, slightly erythematous or hypopigmented. Papules and nodules (lepromas) are usually of skin-color or at times can be erythematous. The lesions can occur anywhere on the body, except for the scalp, axillae, groins and perineum (since these parts have a bit of a higher temperature). In a few cases diffuse plaques are seen with shiny, wrinkled and atrophic skin.

In the initial stages, there is not much of alopecia or loss of sensation occurring over the lesions, but gradually numbness and anesthesia starts to occur, especially over the hands and feet (called as bilateral 'glove and stocking' anesthesia). Gradually progressive fibrosis and thickening of the peripheral nerves occurs.

In untreated and chronic cases, the skin over the face (especially the forehead) becomes thick, and is thrown into wrinkles or convoluted folds, which gives it a lion-like appearance (leonine facies). There is loss of hair over the eyebrows and eyelashes, thickened earlobes, distorted or collapsed nose (due to septal perforation and a depressed nasal bridge), waxy shiny face, hoarseness of voice, and loss of upper incisors. Involvement of the bones and digits gives rise to their resorption and the very slow nerve involvement leads to the appearance of claw hands, wrist drop and foot drop.

Diagnosis

The Lepromin skin reaction is negative in this type.

Associated Features

- Affection of the nerves of the eye can cause lagophthalmos and anesthesia of the cornea and conjunctiva, resulting in dryness of the cornea and repetitive injury. This further results in avascular keratitis, corneal lepromas, pannus formation, corneal opacity, corneal ulceration and finally blindness. There is also a tendency to develop a painful red eye condition due to iridocyclitis.

- The neural involvement predisposes anesthesia of the part (with resultant complications like repetitive trauma, bruises, burns or cuts, trophic ulcers, cellulitis, etc.), muscular weakness and contracture, and autonomic dysfunction
- Deformities (crooked or short) of the hands and feet are also seen with thin and brittle nails. X-ray shows osteoporotic changes especially of the phalanges, small osteolytic cysts and often hairline or compression fractures.
- Except for the gastrointestinal tract, lungs, and brain most of the other organs can contain leprae bacilli. The most frequent complications seen are lymphadenopathy, bone marrow affection, hepatosplenomegaly, testicular atrophy, gynaecomastia and sterility or impotency can result.

Prognosis

It is a slowly progressive disease with a poor prognosis if left untreated, resulting usually in death due to recurrent infections, ulcerations, amyloidosis or renal failure. Also, the occurrence of 'lepra reactions' in this type of leprosy makes the prognosis grave.

BORDERLINE LEPROSY

In this group one can find a variable mixture of features of both the tuberculoid and the lepromatous types, depending on the position of the patient within the borderline spectrum of the two poles (tuberculoid or lepromatous). Borderline leprosy is the commonest type of disease encountered, with BT usually predominating in Africa and BL in Asia.

At the '*tuberculoid end*' of the spectrum (BT type), one finds very few, asymmetrical and dry lesions with a more pronounced alopecia, anhidrosis, anesthesia and fewer bacilli in smears. The lesions are similar to TT lesions, except that they are smaller and more numerous (usually three to ten) and satellite lesions around large macules or plaques are characteristic.