

International Journal of Research and Reviews in Pharmacy and Applied science

[www.ijrrpas.com](http://www.ijrrpas.com)



Dr.Chandanwale.A.S<sup>1</sup>

Dr.Shivkumar.<sup>2</sup>

Dr. Pande G<sup>3</sup>

<sup>1</sup>Prof.Orthopedics Dept,

<sup>2</sup>Resident, Orthopedics Dept,

<sup>3</sup>.Asst. Prof. Dept.

Physiotherapy B.J.G.M.C., Pune

# LEPROSY AND ORTHOPAEDIC COMPLICATIONS – CURRENT STATUS IN INDIA

## INTRODUCTION

Today, the diagnosis and treatment of leprosy is easy. Still leprosy is more prevalent in endemic regions around the world. The number of new cases detected during 2010 was 228,474. <sup>1</sup> Peripheral neuropathy and orthopaedic complications form a major bulk of morbidity in leprosy patients.

Leprosy is an infectious disease caused by *Mycobacterium leprae*. It was Hansen 1 in 1873 who discovered *M. leprae*, the first bacterium to be identified as causing disease in humans. It is believed to be transmitted via droplets from the nose and mouth, through close contact with a person affected by the disease who has not received treatment. The bacillus multiplies slowly and it can take up to 20 years before symptoms appear. Leprosy primarily affects the skin and peripheral nerves, the upper respiratory tract and the eyes. Delayed treatment can result in physical and sensory disability, including damage to fingers and toes, contractures, inability to close the eyelids and blindness, which often lead to social exclusion. With early diagnosis and multidrug therapy treatment, monitoring and early treatment of orthopaedic complications can decrease long term morbidity, leprosy patient can practically lead normal life.

Approximately three million people are disabled by the disease worldwide. India achieved the elimination goal, (i.e. prevalence <1 case per 10,000 population) at the national level, as of December 2005. As of July 2006, there were 99,255 registered leprosy cases on treatment, giving a Prevalence Rate (PR) of 0.88 per 10,000 population<sup>2</sup>. The number of new cases detected during the year 2005 was 168,186.

About 25% of the leprosy patients who are not treated at the early stages of the disease develop deformities of the hands and feet<sup>3</sup>. About 80% of the joint lesions are in the metatarso-phalangeal joints of the foot or in the inter-phalangeal joints of the hands and feet<sup>4</sup>. The frequency of the bone changes which has been recorded in different studies has varied from 15% to 95%.<sup>5</sup>

*M. leprae* is an acid-fast rod and grows best in cooler tissues (the skin, peripheral nerves, anterior chamber of the eye, upper respiratory tract, and testes).

Patients with untreated lepromatous leprosy discharge bacilli from the nose. The principal portal of entry into the human body is the upper respiratory tract. *M. leprae* cannot traverse through intact skin in either direction, and the infection is not spread by touching. The incubation period is two to five years in tuberculoid disease and eight to 12 years in lepromatous disease. In contrast to tuberculosis, co-infection with HIV has no strong effect on the development of leprosy.<sup>6</sup>

## Pathogenesis

*M. leprae* has a predilection for Schwann cells and skin macrophages and is taken up by these cells early in infection<sup>7</sup>. In many cases (indeterminate leprosy), early lesions heal spontaneously with eradication of bacilli. If the bacilli persist and multiply in the skin and/or nerves, established leprosy develops. In tuberculoid leprosy there is involvement of skin and nerves. Lymphocytes breach the perineurium, and destruction of the Schwann cells and

axons may be evident, resulting in fibrosis of the epineurium, replacement of the endoneurium with epithelial granulomas, and occasionally caseous necrosis. Acid-fast bacilli are few or absent. Such invasion and destruction of nerves in the dermis by T lymphocytes is pathognomonic for leprosy<sup>8</sup>. Sometimes a caseous abscess may form inside the perineural sheath causing paralysis of the nerve. Lepromatous disease manifests with generalised involvement of skin, nerves and mucous membranes. The damage and hypertrophy of nerves resulting from bacillary invasion tends to be symmetrical and is more insidious and extensive than in tuberculoid disease. The granulomatous lesions contain macrophages and abundant bacilli. In skin lesions, the small dermal sensory and autonomic nerve fibres are damaged. This can lead to glove and stocking paraesthesiae and loss of sweating. Damage to peripheral nerves leads to sensory loss and paralysis.<sup>7</sup> Nerve damage occurs across the leprosy spectrum. Acute nerve pain with associated loss of function may occur as part of a leprosy reaction, an acute immunological reaction, which presents with inflamed skin lesions and neuritis.

Classification of leprosy: leprosy is classified into six types based on the clinical features (Ridley & Jopling classification):<sup>9</sup> The type of the disease is a reflection of the immune status of the host. The first sign of the disease is the feeling of numbness or loss of sensation for temperature (heat) followed by touch and pain which usually begins at the extremities. The skin lesions appear later during the course of the disease.

Disease	Clinical features
Indeterminate leprosy	They are the first type of skin lesions characterized by hypo-pigmented spots. The lesions undergo healing spontaneously
Paucibacillary (tuberculoid leprosy)	A large red patch with well-defined raised borders or a large hypo pigmented asymmetrical lesion. Lesion is dry and hairless. Infectivity is minimal at this stage. Loss of sensation is seen. Nerves become thick followed by loss of function. It either progresses to the borderline stage or spontaneously get cured
Borderline tuberculoid leprosy	Characterized by small and numerous skin lesions. The disease goes back to the tuberculoid stage or progresses to the next stage

Borderline borderline leprosy	Several small, irregular red lesions are seenModerate sensory loss is seenIt either goes back to the previous stage or progresses to the next
Borderline lepromatous leprosy	Several lesions such as plaques, macules, papules, and nodules are seen.Lesions have a characteristic inverted saucer like appearance.
Multibacillary Hansen’s disease (lepromatous leprosy)	Early symptoms:Several lesions such as plaques, macules, papules, and nodules are seen.Nasal congestion, discharge and bleeding is seenInflammation of the leg and anklesProgressive symptoms:Thickening of the dermis (skin) in the forehead and ear lobesLoss of eyebrows and eyelashesEye defects such as glaucoma and blindness are seenNodules in the legs break and form ulcersEnlargement of the breast and sterility occurs in the malesInternal infection results in the enlargement of the liver and lymph nodesLoss of sensation in the peripheral nerves. Deformation of the fingers and toes results due to painless repeated trauma.

**Bone changes in leprosy**

These can be divided into two groups, specific changes, secondary changes. primary lesions are due to the direct involvement of bone by the organism, whereas secondary lesions are the result of trauma and infection (Fig. 4) imposed upon denervated tissues.

**Specific bony changes**

Specific bony lesions in leprosy are rare with an incidence of between 3% and 5% among hospitalisedpatients,<sup>10</sup> and are mainly confined to the small bones of the face, hands and feet. These lesions are characterisedby granulomatous tissue reactions, which are destructive and manifest radiographically as focal areas ofincreased rarefaction. The margins are thin, but may be sclerotic. Obliteration of the cavity may result from its collapse

with flattening of the articular surface. In the hands and feet, the disease mainly involves the proximal and/or the middle phalanges. It may present as thinning of the endosteum with corresponding widening of the medullary canal localised to the area of the metaphysis. Fusiform swelling of the soft tissues overlying the corresponding part of the affected digit is more common.<sup>11</sup> This is occasionally associated with enlarged nutrient foramina.

As the disease progresses and the trabeculae are destroyed, the radiographs reveal a 'honeycomb and cystic' appearance. With healing, radiographic changes become sharply defined as cysts with sclerotic margins. The articular surface can also be involved and the intrinsic forces in the hand may result in fracture, subluxations and rigid clawing of the fingers.

Leprous osteitis in the hands commonly involves the distal ends of the proximal and middle phalanges, whereas it usually affects the metatarsal heads in the feet. Thinning and irregularity of cortex, concentric cortical erosion, areas of bone destruction, subarticular erosion are more common in feet. Because of weight-bearing forces, there may be comminuted pathological fractures of the metatarsal heads.

### **Secondary changes**

These are common in leprosy. Localised osteoporotic changes result from immobilisation, most frequently because of disuse associated with fixed contractures of the fingers. Motor denervation is sometimes associated with absorption of the cancellous bone and the development of a concentric type of bone atrophy. It affects the length, the width or both. The most common changes in leprosy however are those due to combined absorption of length and width of bone. The result is a tapered appearance at the end of the bone, termed 'licked candy stick'. These are the most common bony changes, and are due to distal absorption affecting the ends of insensitive fingers and toes. When this process is complicated by infection, it may be followed by progressive absorption with loss of the digits and the development of the so-called 'mitten hand'.<sup>11</sup>

Osteoporosis mainly causes vertebral fractures, intertrochanteric fractures and Colles fractures.



Honey combing in the heads of metacarpals, primary periosteitis with licked candy appearance and contracted fingers of the right hand.

Bone cysts in the middle phalanges of both thumbs and absorption of terminal phalanx with tuft erosion of the fingers



Leprotic hand showing distal phalanx absorption of little and ring finger and clawing of other fingers.

Absorption secondary to trauma and infection has three common sites of predilection in the insensitive foot. The first is the distal type affecting the tips of the toes. In the second type the metatarsophalangeal joint is at risk of damage and the third involves the tarsus.

The common paralytic deformities of the foot, which are potentially damaging to the metatarsophalangeal joints, are clawed toes and drop foot. Static deformities such as hallux valgus, metatarsus primus varus and pesplanus are also associated with plantar ulceration, which may lead to bone absorption.<sup>11</sup> Tarsal disintegration is not infrequent and may involve one or more tarsal bones. The most common tarsal disintegration affects the medial arch. The lateral arch is less commonly involved and occurs as a late complication of a rigid deformed foot.<sup>11</sup>

Leprotic deformities are more common in males. Deformities are more common after 40 years of age. It may be due to added osteoporosis due to old age or due to longer period of disease. Secondary changes are more common in Lepromatous leprosy.

Other secondary changes involve Arthritis, Osteomyelitis due to insensitive skin and chronic nonhealing ulcer, these are more common in feet because of weight bearing and trauma tendency.

Contracted fingers / claw hands / claw toes, Subluxation / dislocation, Cupping of joints, Fractures, Secondary periosteitis, Disintegration of tarsal bones are other documented bony changes mainly involving tarsal bones except claw hands.

## CONCLUSION

Though leprosy is an ancient disease, it is not rare in India and it still continues to be the most feared due to social stigmata and deformities. The bone changes with deformities are preventable, since all these are not due to the disease itself. The study of the bony changes may help Early detection and management of the permanent loss of function and the occurrence of deformities and disabilities.

## BIBLIOGRAPHY

1. <http://www.who.int/lep/situation/prevalence/en/index.html>
2. [http://www.searo.who.int/en/Section10/Section20\\_9888.htm](http://www.searo.who.int/en/Section10/Section20_9888.htm)
3. Iyere BB. Leprosy deformities: experience in Molai leprosy hospital, Maiduguri, Nigeria. *Lepr Rev* 1990; 61: 171-179.
4. Carayon A, Dharmendra. Bone and joint changes in leprosy. In: Dharmendra, editor, *Leprosy*, Vol 2. Bombay: Samant and company; 1985;546-550.
5. Thappa DM, Sharma VK, Kaur S, Suri S. Radiological changes in the hands and feet in disabled leprosy patients: a clinico-radiological correlation. *Indian J Lepr* 1992; 64(1):58-66.

6. HIV-M. Leprae Interaction: Can HAART Modify the Course of Leprosy?EuzenirNunesSarno, MD, PhD, Ximenallarramendi, MD, PhD, José A. Costa Nery, MD, PhD, Anna M. Sales, MD, MSc, Maria C. Gutierrez-Galhardo, MD, PhD, Maria L. FernandesPenna, MD, PhD, Elizabeth Pereira Sampaio, MD, PhD, and Gilla Kaplan, PhD.Public Health Rep. 2008 Mar-Apr; 123(2): 206–212.
7. Britton WJ, Lockwood NJD. Leprosy. Lancet 2004;363:1209-19.
8. 5. Gelber RH. Leprosy. In: Braunwald E, Harrison TR, eds. Harrison's principles of internal medicine. Fifteenth ed. London: McGraw-Hill, 2001:1035-40.
9. [http://www.medindia.net/patients/patientinfo/Leprosy\\_Classification.htm](http://www.medindia.net/patients/patientinfo/Leprosy_Classification.htm)
10. Paterson DE, Rad M. Bone changes in leprosy, their incidence, progress, prevention and arrest. Int J Leprosy 1961;29:393-422.
11. Enna CD, Jacobson RR, Rausch RO. Bone changes in leprosy: a correlation of clinical and radiographic features. Radiology 1971;100:295-306.